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Managing Associate Editor
Phone: (714) 456-2326
Shahram.Lotfipour@uci.edu

Project BETA: Best practices in Evaluation and Treatment of Agitation

A multidisciplinary guidelines collaboration from the
American Association for Emergency Psychiatry (AAEP)
www.emergencypsychiatry.org

Project Chair/Editor-in-Chief
Garland H. Holloman Jr, MD, PhD
University of Mississippi Medical Center

Project Founder/Director
Scott L. Zeller, MD
Alameda County Medical Center

Project Editor
Toni Nouri, RPh

Project Administration, AAEP
Jacquelyn Coleman, CAE
Jacquilyn Davis

Project Contributors:

Michael H. Allen, MD
University of Colorado School of Medicine

Eric L. Anderson, MD
Spectrum Behavioral Health

Jon S. Berlin, MD
Medical College of Wisconsin

Benjamin Bregman, MD
George Washington University

Suzanne Bruch, MD
Alameda County Medical Center

Glenn W. Currier, MD
University of Rochester Medical Center

David Feifel, MD, PhD
University of California, San Diego

Avrim Fishkind, MD
JSA Health Telepsychiatry

Peter Florence, MD
Dalhousie University

Rachel L. Glick, MD
University of Michigan

Herbert J. Harman, MD
Carolinas Medical System

Doug Hughes, MD
Boston University School of Medicine

Pria Joglekar, MD
Kaiser Permanente, Pleasanton, CA

Daryl K. Knox, MD
Mental Health and Mental Retardation Authority of Harris County

Judy S. Linn, MS, RN
Alameda County Medical Center

Anthony T. Ng, MD
Acadia Hospital

Kimberly Nordstrom, MD, JD
Denver Health Medical Center

Jagoda Pasic, MD, PhD
University of Washington Harborview Medical Center

David Pepper, MD
Hartford Hospital/Institute of Living

Guy C. Qvistgaard, MS, MFT
Alameda County Medical Center

J. J. Rasimas, MD, PhD
Penn State College of Medicine

Janet S. Richmond, MSW
Tufts University School of Medicine

Muhamad Aly Rifai, MD
Blue Mountain Health System

John S. Rozel, MD, MSL
re:solve Crisis Network

Susan Stefan, Esq

Victor Stiebel, MD
University of Pittsburg

Keith R. Stowell, MD, MSPH
University of Pittsburgh

Michael P. Wilson, MD, PhD
University of California, San Diego

B. K. P. Woo, MD
University of California, Los Angeles Olive View Medical Center

Leslie S. Zun, MD
Mount Sinai Hospital

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Overview of Project BETA: Best practices in Evaluation and Treatment of Agitation

Garland H. Holloman Jr, MD, PhD* * University of Mississippi Medical Center, Department of Psychiatry, Jackson, Mississippi
Scott L. Zeller, MD† † Alameda County Medical Center, Department of Psychiatry, Oakland, California

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Agitation in emergency settings is a major concern, with a staggering 1.7 million episodes annually in the United States alone.[1] Agitated individuals are at risk of becoming aggressive and violent, and of causing harm to themselves, others, and property. Agitation is a leading cause of hospital staff injuries and can cause untold physical and psychological suffering for patients and all those nearby.[2–4]

Yet, despite the pervasiveness of agitation, there is surprising inconsistency in treatment approaches, which can vary widely by region and institution. Many facilities now use techniques such as intervention teams, which are paged instantly when there is an agitated patient, or "management of assaultive behavior" protocols that seek to engage patients into voluntarily accepting treatment. However, far too many agencies still treat all episodes of agitation in a fashion that might best be described as "restrain and sedate."

Although regulatory agencies and advocacy groups have called for a reduction in the use of restraint and less coercion in psychiatric treatment, there has been inadequate discussion regarding effective, alternative management of the agitated patient. Clearly, a void has existed in quality guidelines for the treatment of agitation.

To help address this need, the American Association for Emergency Psychiatry (AAEP), in October 2010, embarked on Project BETA (Best practices in Evaluation and Treatment of Agitation). Recruiting dozens of emergency psychiatrists, emergency medicine physicians, and others associated with acute care of the mentally ill, Project BETA has intended to provide guidelines that are not only effective and safety minded but also in the best interests of the patient.

Creating quality guidelines for agitation is no easy task. Unlike most disease states, the research database on agitation is quite limited. Much of this can be ascribed to the difficulty in obtaining the informed consent necessary for most clinical studies. How does one get informed consent from a combative, threatening individual? Further, in those studies that do involve informed consent, questions might arise as to the severity of subjects' levels of agitation, if indeed they were even able to comply with the consent process.

Given these obstacles, the Project BETA team determined that the best guidelines would be ascertained through a synthesis of the best available research with the expert consensus of seasoned clinicians.

Until now, existent guidelines for agitation have focused solely on medication strategies. Yet, agitation can result from myriad origins, and its treatment is multifaceted, with pharmacology only playing 1 part. The Project BETA members recognized that to truly address the agitation spectrum, for the first time, guidelines should be developed that would direct clinicians in all interventional aspects, including triage, diagnosis, and verbal de-escalation, as well as medicine choices.

Thus, 5 study workgroups were developed by using the basic approaches of emergency psychiatry as a foundation. The treatment goals of emergency psychiatry are as follows: (1) exclude medical etiologies for symptoms; (2) rapid stabilization of the acute crisis; (3) avoid coercion; (4) treat in the least restrictive setting; (5) form a therapeutic alliance; and (6) appropriate disposition and after-care plan.[5] The 5 workgroups, projected in the order of following a patient through an intervention, were established to address the following topics:

- Medical evaluation and triage of the agitated patient
- Psychiatric evaluation of the agitated patient
- Verbal de-escalation of the agitated patient
- Psychopharmacologic approaches to agitation
- Use and avoidance of seclusion and restraint

Each group then created a written article and guidelines derived from evidence-based research and consensus outcome, which follow in this issue of *Western Journal of Emergency Medicine*. Although each article is able to stand on its own, the

entire group is intended to be read and used collectively, as the articles are intertwined, referring to and leading into each other.

Working with an agitated patient can be challenging, and, as in managing other medical emergencies, it requires both knowledge and skills. As in advanced cardiovascular life support training, the former can be learned in the classroom, but the latter requires practice.

An important first step is learning to balance how to evaluate and manage the patient simultaneously. Medical assessment is essential to rule out life-threatening causes of agitation; yet, the patient who is agitated may not be cooperative with the evaluation. Thus, one's observation of the patient and medical judgment must drive decisions while engaging the patient in verbal de-escalation to obtain cooperation.

Some patients with agitation can be de-escalated to calmness by verbal de-escalation alone. However, others will require medication, and the preferred medication should be one that targets the underlying etiology.[6] Therefore, there is a need to establish a working diagnosis before instituting appropriate pharmacologic intervention.

Mastering verbal de-escalation will result in many positive rewards for the clinician. Although some might believe that in their busy clinic there is no time to attempt de-escalation and restraining a patient is the speediest solution, it can indeed be just the opposite. Verbal de-escalation can typically be quite effective in a relatively brief period, while placing a patient in restraints can require significant staff involvement—from the time needed to "take down" and restrain the patient to the obligation for one-to-one observation. Throughput can be even more affected from a disposition standpoint, as many receiving facilities will not consider accepting a patient who has been recently restrained or a patient who is oversedated from injudicious use of medication.

Avoiding the restraint process altogether can have safety and long-term implications. Perhaps as many as two thirds of staff injuries involving psychiatric patients occur during "containment" procedures for restraint.[7] Furthermore, patients who have not been restrained and forcibly medicated during an emergency department visit will be less likely to mistrust and fear medical personnel and, thus, may feel more comfortable seeking assistance in the future, hopefully before reaching a highly agitated state.

The authors of Project BETA understand that not all of the guidelines can be followed in every situation and have endeavored to make accommodations for that. The algorithms included in the articles provide guidance for noncoercive evaluation and management of the agitated patient, but allow for direct implementation of more restrictive interventions for those unfortunate patients who are so combative or delirious that other options would not be practical. Still, it is hoped that these guidelines will assist clinicians in recognizing that agitated individuals need not necessarily go straight into restraints but instead can be treated in a more benign, collaborative fashion, which will lead to less injuries, better therapeutic alliance, improved throughput and superior long-term outcomes.

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Address for Correspondence: Garland H. Holloman Jr, MD, PhD, University of Mississippi Medical Center, Department of Psychiatry, L-740, 2500 N State St, Jackson, MI 39216. E-mail: gholloman@umc.edu.

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Medical Evaluation and Triage of the Agitated Patient: Consensus Statement of the American Association for Emergency Psychiatry Project BETA Medical Evaluation Workgroup

Kimberly Nordstrom, MD, JD*
Leslie S. Zun, MD†
Michael P. Wilson, MD, PhD‡
Victor Stiebel MD§
Anthony T. Ng, MD||
Benjamin Bregman, MD¶
Eric L. Anderson, MD#

* Denver Health Medical Center, University of Colorado Denver, Department of Psychiatry, Denver, Colorado
† Mount Sinai Hospital, Chicago Medical School, Department of Emergency Medicine, Chicago, Illinois
‡ UC San Diego Health System, Department of Emergency Medicine, San Diego, California
§ University of Pittsburgh, Department of Psychiatry, Pittsburgh, Pennsylvania
|| Acadia Hospital, Bangor, Maine
¶ George Washington University, Department of Psychiatry, Washington, DC
# Johns Hopkins, Department of Psychiatry and Behavioral Sciences, Baltimore, Maryland

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Numerous medical and psychiatric conditions can cause agitation; some of these causes are life threatening. It is important to be able to differentiate between medical and nonmedical causes of agitation so that patients can receive appropriate and timely treatment. This article aims to educate all clinicians in nonmedical settings, such as mental health clinics, and medical settings on the differing levels of severity in agitation, basic triage, use of de-escalation, and factors, symptoms, and signs in determining whether a medical etiology is likely. Lastly, this article focuses on the medical workup of agitation when a medical etiology is suspected or when etiology is unclear. [West J Emerg Med. 2012;13(1):3–10.]

INTRODUCTION

Agitation is an extreme form of arousal that is associated with increased verbal and motor activity. These symptoms are caused by a variety of etiologies, both medical and psychiatric. Patients with agitation may present not only to an emergency department (ED) or a psychiatric emergency service (PES), but also to a clinic or acute care center that does not have the onsite medical resources of a physician or midlevel practitioner, such as a community mental health facility (termed *nonmedical facility*).

This article is one of a set of companion articles to address Best practices in the Evaluation and Treatment of Agitation in the emergency setting, Project BETA.[1]

LEVEL OF AGITATION

Various assessments of agitation are available to determine the patient's level of agitation, such as the Overt Agitation Severity Scale[2] or Overt Aggression Scale.[3] Another scale is the Behavioural Activity Rating Scale or BARS (Table 1).[4] The American Association for Emergency Psychiatry does not consider one agitation rating scale to be better than another, although we find the BARS is easy to use reliably, even for one not trained in psychiatry or emergency medicine. The BARS was created to help assess agitation in pharmaceutical study trials. It is simple to use and does not require the participant/patient to answer questions.[4] This scale is especially useful in the nonmedical setting, where decisions are made by those who are not medically trained.

TRIAGE OF THE AGITATED PATIENT

Nonmedical Setting

When an agitated patient presents to a clinic without a medical team, the immediate task is to determine if the patient

Table 1. Behavioural Activity Rating Scale.[4]

1 = Difficult or unable to rouse
2 = Asleep but responds normally to verbal or physical contact
3 = Drowsy, appears sedated
4 = Quiet and awake (normal level of activity)
5 = Signs of overt (physical or verbal) activity, calms down with instructions
6 = Extremely or continuously active, not requiring restraint
7 = Violent, requires restraint

Table 2. Findings that require immediate evaluation by a clinician.

Symptoms
Loss of memory, disorientation
Severe headache
Extreme muscle stiffness or weakness
Heat intolerance
Unintentional weight loss
Psychosis (new onset)
Difficulty breathing
Signs
Abnormal vital signs: pulse, blood pressure, or temperature
Overt trauma
One pupil larger than the other
Slurred speech
Incoordination
Seizures
Hemiparesis

needs transfer to a higher level of care. This determination is based on whether a medical etiology is suspected by history or obvious signs and symptoms, as listed in Table 2, and on the patient's level of agitation. Algorithm 1 illustrates this process (Figure 1).

For any patient with signs or symptoms listed in Table 2 or with BARS score of 1, immediate transfer to a medical ED is indicated. Patients with a BARS score of 2, 3, or 7 should be transferred immediately to either a medical emergency department or a PES with medical capability. In the case of the highly agitated patient, transfer to an emergency service should happen quickly. For mild to moderate agitation (BARS = 5 or 6), the patient may be calmed with verbal de-escalation techniques, and environmental modification. If this is successful, further evaluation at the clinic is acceptable. For patients who do not respond, transport to an acute care facility, such as an ED or PES, is indicated. To protect the patient and staff, emergency medical services or ambulance should be contacted through 911 or a similar emergency system. The ED or PES should be notified before patient arrival to ensure that the staff is ready to receive the patient. The receiving facility needs to have a room, crash cart, and adequate number of staff ready to meet the ambulance on arrival.

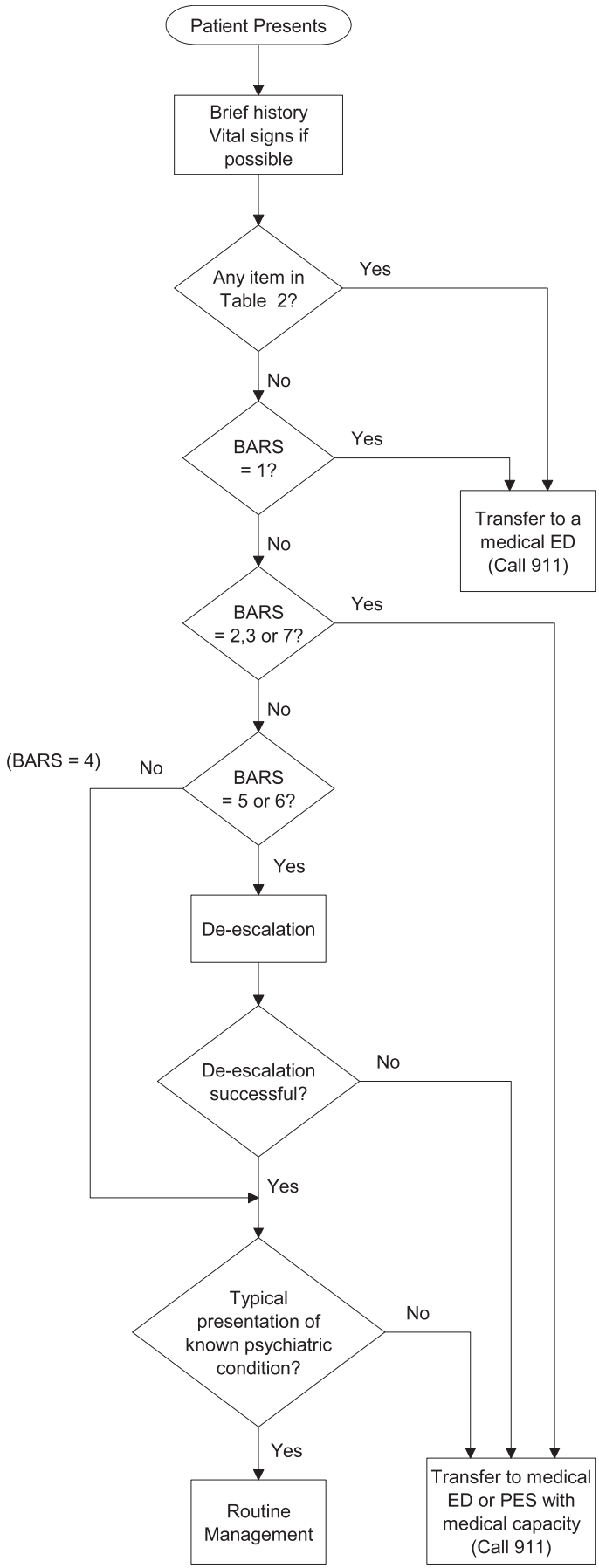
Medical Setting

Management of the agitated patient in the medical setting is illustrated in algorithm 2 (Figure 2). When triaging an agitated patient in the medical setting, a brief history and vital signs should be obtained if possible. If any item in Table 2 is found or suspected at initial intake triage, immediate evaluation by a clinician is indicated. Oxygenation level and blood sugar level should also be obtained if possible. The initial examination should be directed at identifying factors (Table 3) that could indicate serious, possibly life-threatening, conditions such as listed in Table 4. These, as well as other conditions that may be discovered after a more complete examination, are discussed in detail below. Unless immediate intervention is indicated, de-escalation should be attempted in an effort to gain the patient's cooperation. Likewise, if initial intake assessment does not point to items in Table 2, de-escalation should be attempted so that the patient can better participate in evaluation.

As visualized in algorithm 2 (Figure 2), the agitated patient may, at any point during the medical evaluation, require medication, restraint, and increased behavioral support. For patients with high-risk indicators for a medical etiology of agitation, listed in Table 3, de-escalation may have to be interrupted and physical restraint may be needed to save the patient's life. Likewise, severely agitated patients who may not be cooperative are at risk for violent behavior and, thus, require restraint if de-escalation is impractical or ineffective. If a patient must be restrained, oxygenation level and finger stick blood glucose should be done immediately if previously it could not be obtained.

Although not fully illustrated in the algorithm, the complete history and physical examination of the patient may have to be interrupted to address the treatment of the patient's agitation, and will need to be resumed after the patient is less agitated. The patient may need treatment to reduce the level of agitation without having a definitive diagnosis. The goal of medication is to calm the patient enough to allow for a thorough evaluation.

Once de-escalation is accomplished, an important triage decision is to determine if the patient has a known psychiatric illness and if the presentation is typical of that illness. If so, the patient should have a directed medical evaluation to rule out an acute medical problem. If none is present, psychiatric evaluation and management are indicated. All other patients should have a complete history, physical examination, and mental status examination followed by directed laboratory and radiologic assessment to determine the definitive diagnosis.



OVERVIEW OF MEDICAL EVALUATION

During the time the patient is being behaviorally stabilized, the ambulance crew, police, family, friends, relatives, and nursing home personnel should be interviewed to obtain a history or collaborative information. According to Olshaker and colleagues,[5] the patient history has a sensitivity of 94% and the physical examination has a sensitivity of 51% for detecting medical problems during the evaluation of psychiatric patients.

The history and physical examination can lead to clues that a nonpsychiatric etiology of agitation may be responsible for the presentation. If a patient presents with known psychiatric disease that is inconsistent with previous presentations, a medical etiology should be suspected. New onset of agitation in a person older than 45 years with no psychiatric history would suggest a medical etiology, since most psychiatric disorders have an earlier onset.

Abnormal vital signs, abnormal physical examination results, overt signs of alcohol or drug intoxication or withdrawal,[6] evidence of exposure to toxins,[7,8] and decreased awareness with attentional problems[9] are all indicative of a medical etiology. Neurologic problems must also be considered. Head injury[10] or a history of disease that can cause neurologic changes,[7] such as history of stroke,[11] cancer, parkinsonism, and multiple sclerosis, would also indicate that the agitation has a medical etiology.

As previously mentioned, decreased awareness and attentional problems are very important because these are signs and symptoms of delirium, which can be subtle and frequently overlooked. Delirium can be defined as a disturbance in level of awareness and reduced ability to direct, focus, sustain, or shift attention and a change in cognition, such as deficits in orientation, executive ability, language, visuoperception, learning, and memory.[12] Careful assessment is most important because delirium indicates an underlying medical etiology.

Agitation can be due to a general medical condition,[9,13] intoxication and withdrawal,[6] and decompensated psychiatric disease. Medical conditions include head trauma;[10] infection[7] leading to meningitis, encephalitis,[14] or sepsis; encephalopathy, usually from renal or liver failure; hypoxia; metabolic derangement;[7] thyroid disease;[15] toxic levels of psychiatric or antiseizure medications;[8] and exposure to environmental toxins.[7] Alcohol or other drug intoxication or withdrawal[6] is a common source for agitation. Conditions that must be considered when evaluating the agitated patient are listed in Table 4.

For those in the nonmedical setting, the only evidence of these at triage may be agitation, disorientation, abnormal vital signs, signs of trauma, odor of alcohol, and history of substance

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Figure 1. Evaluation in a nonmedical setting. *BARS*, Behavioural Activity Rating Scale; *ED*, emergency department; *PES*, psychiatric emergency service.

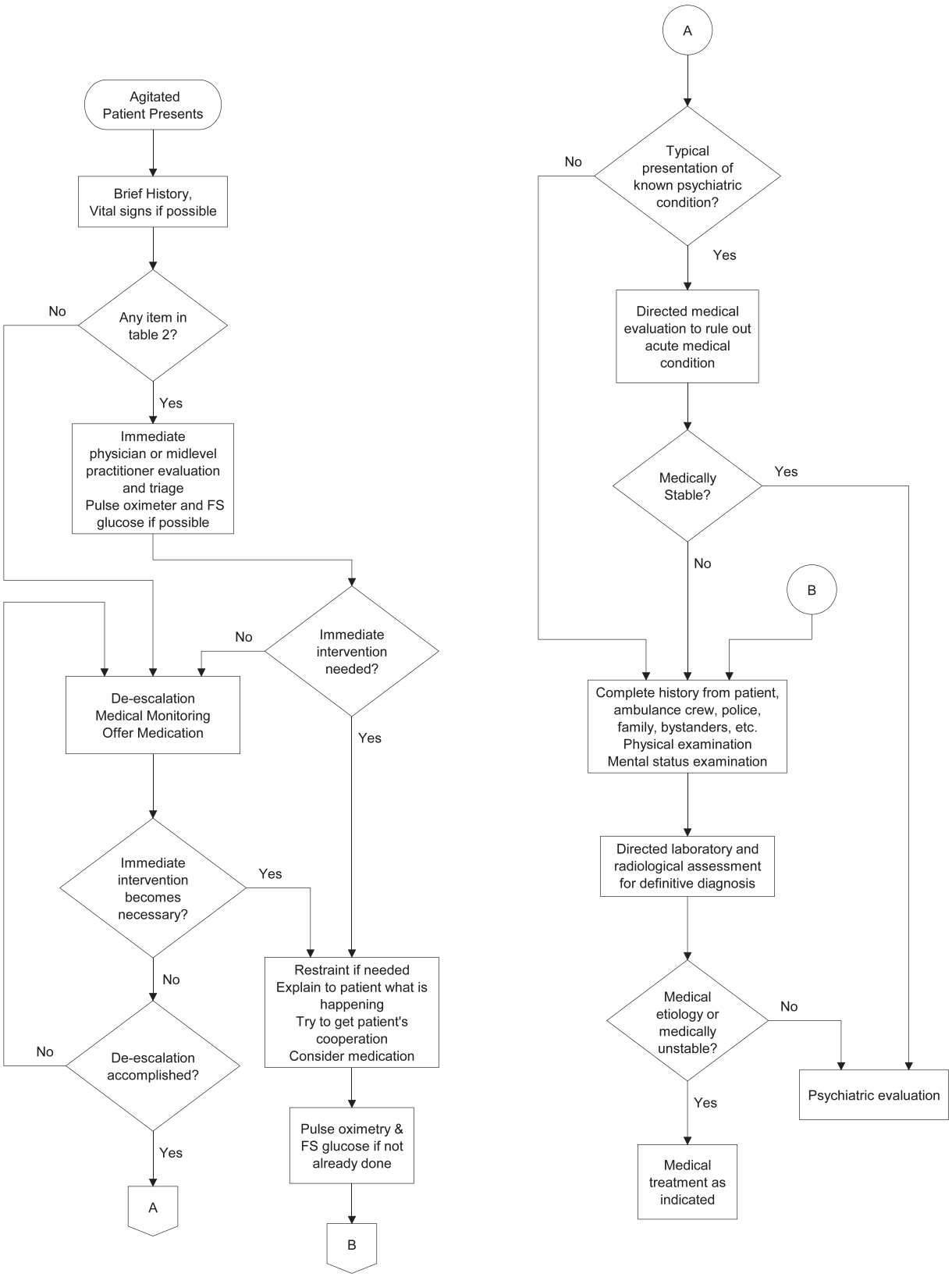

Figure 2. Medical evaluation in the emergency department or psychiatric emergency service. *FS glucose*, finger-stick glucose.

Table 3. Factors that could indicate serious, possibly life-threatening, conditions.

1. New onset at age >45 years
2. Abnormal vital signs
3. Focal neurologic findings
4. Evidence of head injury
5. Substance intoxication
6. Substance withdrawal
7. Exposure to toxins or drugs
8. Decreased awareness with attentional problems

use. Presence of these signs and symptoms usually indicate that a medical evaluation is indicated to determine if a reversible etiology of the agitation needs to be addressed. Conditions can be life-threatening if not treated quickly. Many of the specific symptoms or signs related to medical conditions that cause agitation can be elicited after obtaining a directed history and observing the patient (Table 2). If any of these conditions are suspected, the patient will need a complete physical examination by a physician or midlevel practitioner and must be transferred to an emergency department for medical evaluation.

MAJOR CATEGORIES OF ETIOLOGY OF AGITATION

Agitation from a General Medical Condition

When a person has head trauma, there will often be history given of the incident. Bleeding or contusions may be visible. The person might complain of headache, memory loss, or loss of consciousness. There may be physical signs such as abnormal vital signs, one pupil being significantly larger than the other, slurred speech, or other motor problems. With encephalitis[14] or encephalopathy, the person may have an altered mental state in the form of confusion or agitation, may exhibit inattentiveness and impaired judgment, and may exhibit new physical symptoms, such as motor incoordination, seizures, or hemiparesis. Fever, headache, or stiffening of the neck are concerning symptoms.[14] A generalized, systemic infection will usually cause a fever (this might not be present in an immunocompromised patient). With sepsis, there may be a high fever, possible seizures, disorientation, and agitation.[7] Hallucinations can also occur, especially visual. In fact, visual hallucinations are a common symptom in delirium, especially in elderly patients.[16,17] Exposure to environmental toxins can cause a wide variety of symptoms, depending on the toxin. Clues often come from the history. Symptoms are often nonspecific and related to a toxic metabolic disturbance; these symptoms include disorientation, somnolence, agitation,[8] and seizures. If this is suspected, poison control should be contacted immediately. Metabolic derangements can cause encephalopathy, cardiac arrhythmias, mental status changes,[7,16] hemiparesis, seizures, and abnormal neurologic findings on examination. Hypoglycemia and hyperglycemia,[7] for instance, can lead to these symptoms, if left untreated, and are easily reversible. The same is true with hypoxia. Key signs that the patient is not receiving enough oxygen include changes in breathing pattern, difficult or rapid breathing, and an abnormal oxygen saturation level. Thyrotoxicosis may happen suddenly. Common symptoms include heat intolerance, recent unintentional weight loss, proximal muscle weakness, palpitations, and anxiety. This condition can progress to cardiac arrhythmias, agitation, psychosis, and even death if left untreated.[15] Physical signs of thyrotoxicosis may include skin

Table 4. Conditions that may cause agitation.

Agitation from general medical condition	Head trauma
	Encephalitis, meningitis, or other infection
	Encephalopathy (particularly from liver or renal failure)
	Exposure to environmental toxins
	Metabolic derangement (eg, hyponatremia, hypocalcemia, hypoglycemia)
	Hypoxia
	Thyroid disease
	Seizure (postictal)
	Toxic levels of medications (eg, psychiatric or antiseizure)
Agitation from intoxication/withdrawal	Alcohol
	Club or recreational drugs (cocaine, ecstasy, ketamine, bath salts, inhalants, methamphetamines)
Agitation from psychiatric disease	Psychotic disorders
	Mania
	Agitated depression
	Anxiety disorders

that is warm and moist to the touch, infrequent blinking or lid lag, and a failure to wrinkle the brow with an upward gaze. Vital signs are often abnormal with a marked tachycardia.[18,19] If a person has recently had a seizure, the "postictal" state can leave the patient confused and agitated.[20,21] When a person ingests toxic levels of psychiatric or antiseizure medications, disorientation, somnolence, or agitation may be the first symptoms. Certain psychiatric medications can lead to life-threatening syndromes such as neuroleptic malignant syndrome (NMS)[22] and serotonin syndrome.[23] In both of these cases, abnormal vital signs such as tachycardia, labile blood pressures, and fever are typically noted. In NMS, the patient has "lead pipe" rigidity whereas the patient with serotonin syndrome has myoclonus and hyperreflexia.

Agitation from Alcohol and/or Recreational Substance Intoxication or Withdrawal

A common reason for change in mental status is intoxication with drugs or alcohol or a patient in substance withdrawal.[6] Getting a drug and alcohol history can add insight and help direct immediate care. In a young person presenting with agitation, without history of psychiatric illness, drug intoxication needs to be considered. It may be difficult to get a reliable history from the agitated, intoxicated patient. The only clues might be abnormal vital signs, odor of alcohol, and possibly drug paraphernalia on the person. If intravenous drug use is suspected, there may be evidence of drug injection, such as track marks on the arms or legs. For withdrawal syndromes, alcohol and benzodiazepine withdrawal are the most medically concerning. They can cause disorientation, agitation, hallucinations, as well as seizures.[6] A person in severe withdrawal may also experience autonomic instability.[6]

Agitation from Psychiatric Disease

If no medical etiology is found for the agitation, the patient can be seen by mental health specialists. Causes of agitation may be related to psychosocial stressors or a primary psychiatric disorder. During an evaluation, history will be helpful in determining likely causes and aid in the intervention or focus of treatment. Any of the major psychiatric illnesses can manifest as agitation, including schizophrenia, agitated depression, or bipolar illness. Patients with a new psychiatric complaint or a presentation that is inconsistent with their prior psychiatric illness may require more intensive testing, as a medical cause may be more likely.

MEDICAL WORKUP

The medical workup of an agitated patient, as with any patient who presents to an ED or PES, begins with completion of the history, vital signs, physical examination, and diagnostic tests, if they are deemed necessary for the evaluation process.[24] The physical examination should be a focused, unclothed, but gowned, examination of the patient that includes an assessment of the heart, lungs, abdomen, skin, and neurologic system. The patient presenting with abnormal behavior needs a full neurologic examination as well as adequate mental status examination. During this assessment, healthcare providers should attempt a provisional diagnosis of the most likely etiology of the agitation. The workup will then largely be guided by this provisional diagnosis.

Agitation from General Medical Condition

Agitation, in which a medical condition is strongly suspected, in which the patient has abnormal vital signs, or in which the patient has medical comorbidities, such as immunosuppressive disease or neurologic disease, requires a medical workup. The workup should be targeted to the underlying condition and may include directed laboratory analysis, neuroimaging, or lumbar puncture, as appropriate.

Agitation from Substance Intoxication or Withdrawal

The medical workup for these individuals may be variable. Young healthy adults with a good history of ingestion may be observed for withdrawal, especially from alcohol or benzodiazepines. A workup should be considered if the amount of ingestion is unknown or if there are symptoms or signs that suggest something more serious, such as alcohol poisoning. A more thorough workup, however, should be strongly considered for older adults with medical comorbidities or who are chronic substance abusers.

Undifferentiated Agitation

Agitation in this category (for which no provisional diagnosis can be assigned, or for which information is not immediately available) should be presumed to be from a general medical condition until proven otherwise. Workup should be targeted to any underlying conditions and may include laboratory analysis, neuroimaging, or lumbar puncture, as appropriate.

Agitation from Psychiatric Disease

If psychiatric disease is strongly suspected, but the patient has no previous history of psychiatric disease (or is younger or older than a typical patient with psychiatric disease), then the agitation should be presumed to be from a general medical condition until proven otherwise, as noted above. In a patient with preexisting psychiatric disease who presents with symptoms similar to his or her previous psychiatric disease, and with normal vital signs and normal mentation, little to no testing may be considered.

The routine examination should include a complete set of vital signs, blood glucose measurement (finger stick), and determination of oxygenation level, if not obtained during triage. The laboratory tests completed should be directed by likelihood of a medical illness causing or exacerbating the psychiatric presentation. Universal laboratory studies done *without indication* tend to be low yield[25,26] and can increase healthcare costs.[27] Evidence also shows that routine tests for a

large population may find abnormalities that were not originally suspected but that have no clinical significance.[28] On the other hand, patients without prior psychiatric history need a complete evaluation that may include detailed laboratory evaluation and radiographic imaging.[29]

CONCLUSION

The consensus of Project BETA members is as follows:

1. Routine laboratory testing is not indicated; rather, directed testing should be based on the most likely diagnosis. Workup of agitation from a general medical condition should be directed toward identifying most likely causes.
2. New-onset agitation should be presumed to be agitation from a general medical condition.
3. There should be a high level of suspicion of agitation from a medical condition in a patient with concerning past medical history, such as immunosuppression, or when onset is outside the normal age ranges of psychiatric disease.

Address for Correspondence: Kimberly Nordstrom, MD, JD, PO Box 776396, Steamboat Springs, CO 80477. E-mail: nordstrom_kimberly@yahoo.com.

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Psychiatric Evaluation of the Agitated Patient: Consensus Statement of the American Association for Emergency Psychiatry Project BETA Psychiatric Evaluation Workgroup

Keith R. Stowell, MD, MSPH* * University of Pittsburgh School of Medicine, Department of Psychiatry, Pittsburgh, Pennsylvania
Peter Florence, MD† † Dalhousie University, Department of Psychiatry, Halifax, Nova Scotia
Herbert J. Harman, MD‡ ‡ Carolinas Medical System, Department of Psychiatry, Charlotte, North Carolina
Rachel L. Glick, MD§ § University of Michigan School of Medicine, Department of Psychiatry, Ann Arbor, Michigan

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It is difficult to fully assess an agitated patient, and the complete psychiatric evaluation usually cannot be completed until the patient is calm enough to participate in a psychiatric interview. Nonetheless, emergency clinicians must perform an initial mental status screening to begin this process as soon as the agitated patient presents to an emergency setting. For this reason, the psychiatric evaluation of the agitated patient can be thought of as a 2-step process. First, a brief evaluation must be aimed at determining the most likely cause of agitation, so as to guide preliminary interventions to calm the patient. Once the patient is calmed, more extensive psychiatric assessment can be completed. The goal of the emergency assessment of the psychiatric patient is not necessarily to obtain a definitive diagnosis. Rather, ascertaining a differential diagnosis, determining safety, and developing an appropriate treatment and disposition plan are the goals of the assessment. This article will summarize what components of the psychiatric assessment can and should be done at the time the agitated patient presents to the emergency setting. The complete psychiatric evaluation of the patient whose agitation has been treated successfully is beyond the scope of this article and Project BETA (Best practices in Evaluation and Treatment of Agitation), but will be outlined briefly to give the reader an understanding of what a full psychiatric assessment would entail. Other issues related to the assessment of the agitated patient in the emergency setting will also be discussed. [West J Emerg Med. 2012;13(1):11–16.]

INTRODUCTION

Often, agitated patients are uncooperative or unable to give a relevant history, leaving clinicians to make decisions based on limited information. Fortunately, definitive diagnosis is not considered a primary goal of the initial emergency assessment of the agitated patient. However, a major decision to be made early in the assessment is whether or not the patient has an underlying medical problem that should be addressed in the medical setting. This is discussed in detail in a Project BETA (Best practices in Evaluation and Treatment of Agitation) companion article.[1,2] Project BETA represents recommendations for best practices in the evaluation and treatment of agitated patients by workgroups of the American Association for Emergency Psychiatry. In this article, we discuss the initial assessment of the agitated patient, including developing a working differential diagnosis based on the patient's mental status examination, to guide the appropriate course of care, whether it be a full psychiatric evaluation or ongoing medical investigation or both.

When a patient arrives in a state of agitation, triage, initial assessment, and de-escalation must occur at the same time the initial assessment is done. When evaluating the patient for a

psychiatric illness, being able to determine a broad category that defines the patient's presenting problem is very important. Knowing the patient's problem in these terms is useful when choosing a medication to help calm the patient. De-escalation, pharmacologic management, and issues related to seclusion and restraint are discussed in detail in Project BETA companion articles.[3–5] The discussion below will focus on broad identification of the agitated patient's problem during the initial interaction.

PSYCHIATRIC EVALUATION AND MANAGEMENT OF THE AGITATED PATIENT

Psychiatric evaluation of the agitated patient includes visual observation of the patient before direct patient interview and paying careful attention to the patient's verbal and nonverbal interaction with the examiner during de-escalation. Collateral information can be very helpful. While de-escalation is in process, another team member can obtain verbal reports from family, paramedics, or police officers or review written material that may accompany the patient. Medical records are also an important source of information, and electronic records, if available, can be readily accessed to determine previous diagnoses and medications. These sources of information can be invaluable in determining the cause of agitation. Once it is determined that the patient does not have an acute medical problem, there are several important questions, the answers to which will guide the next step in management of the patient. These are illustrated by the algorithm shown in the Figure.

The first question is whether the patient has a delirium. It is not uncommon for a patient to go through initial screening and have a diagnosis of delirium overlooked. The patient may be mistakenly diagnosed as being psychotic, or the signs and symptoms of delirium may be subtle and easily overlooked. In delirium, the patient has an altered level of awareness and problems directing, focusing, sustaining, or shifting attention.[6] The examiner must pay close attention to how the patient interacts during the encounter to even recognize these often subtle signs. Does the patient seem confused and unable to focus? Are there perseverative behaviors? Does the patient appear to be responding to visual hallucinations? Are there signs of language impairment, problems naming, or other cognitive deficits? If agitation is associated with any of these findings, especially in the setting of drug or medication use or medical illness, the presumptive diagnosis is delirium.

Next, the examiner must consider whether the patient has chronic cognitive impairment that is contributing to the current state of agitation. The patient with a history of brain injury, developmental disability, or dementia can be easily upset in unfamiliar settings, and might respond to the hospital visit with agitation. Although the examiner may notice cognitive deficits in these patients at presentation, history from family members, friends, or other caregivers may be all that is available, since the agitated patient may not be able to participate in a formal examination. Brief cognitive screening, using tools such as the Folstein Mini Mental State Examination[7] or the Brief Mental Status Examination, based on the Orientation-Memory-Concentration Test[8] and described by Kaufman and Zun,[9] can be attempted. However, these instruments may have to wait until the patient is calmer and able to participate. If defects in cognition are found, collateral history is needed to determine if these are old or new.

The next question is whether the patient is intoxicated or in withdrawal. History of recent drug use is important, as is consultation of the *Diagnostic and Statistical Manual of Mental Disorders*,[10] which describes specific criteria for intoxication and withdrawal syndromes caused by common drugs. The emergency clinician should be familiar with these diagnostic criteria, many of which can be picked up by observation. This is illustrated by the following examples: (1) cocaine intoxication criteria include pupillary dilation, perspiration, vomiting, confusion, dyskinesias, dystonias, and seizures; (2) the patient intoxicated with opiates has pupillary constriction and may have slurred speech; (3) alcohol withdrawal is associated with sweating; hand tremor; vomiting; transient visual, tactile, or auditory hallucinations; and anxiety. All of these signs are readily observable.

The next question is whether the patient is agitated owing to psychosis caused by a known psychiatric disorder. Family or friends who have brought the patient to the emergency department may know of an existing psychotic disorder. If the patient is alone, someone may try to call to get collateral information from family, friends, outpatient care providers, or any other individuals who might know about the patient's history. While there may be confidentiality concerns, a patient's state of agitation must be considered a medical emergency, and obtaining information from others is necessary to provide appropriate care in this setting.

Finally, there are those patients who do not fall into the above categories. If the patient is not psychotic but exhibiting signs and symptoms of mania, the treatment is the same as for the patient with psychosis.[11] For agitation due to nonpsychotic depression or an anxiety disorder, treating the underlying anxiety is appropriate.[12] If the patient is simply angry or out of control (often in the setting of a personality disorder), verbal de-escalation techniques may work, even with the aggressive patient.[3]

When the patient is calm enough to undergo an interview, formal psychiatric assessment can be completed. There is no established standard assessment; however, the evaluation of an agitated patient should be as in depth and as complete as possible. Assessment should include not only discussion with the patient, but also collection of collateral history and review of available records, both of which are invaluable if the patient is unable to engage in an interview. Chief complaint, history of present illness, past psychiatric history, past medical history, substance use history, social history, family history, and the

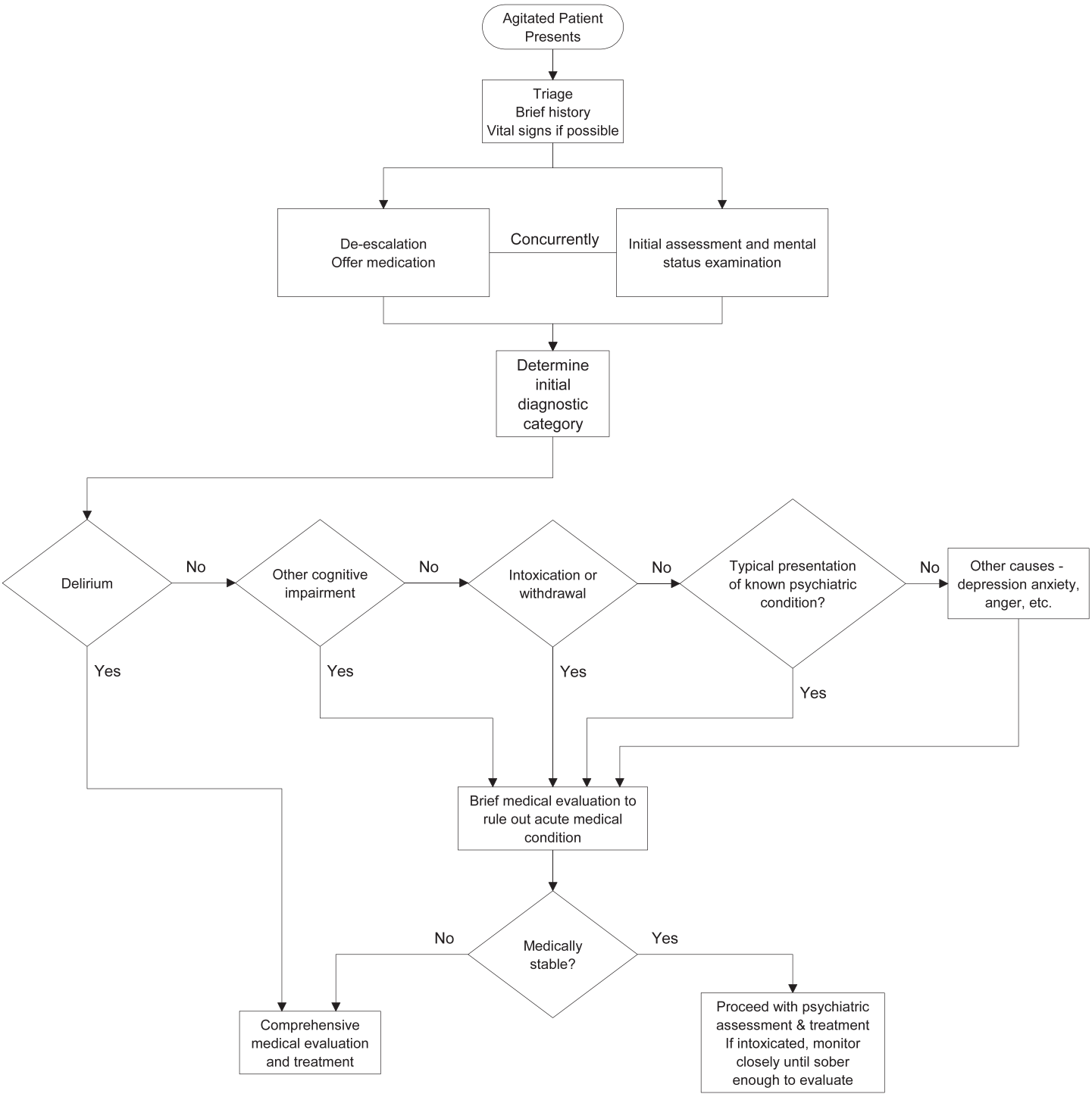

Figure. Algorithm for psychiatric assessment of the agitated patient.

mental status examination should be covered. These are summarized briefly in the upcoming text.

Chief Complaint

The patient may give a different reason for being brought to an emergency setting than that given by family members, police officers, or others who may accompany the patient. Both reasons should be noted and considered. A skilled interviewer can use this part of the assessment to tease out the stated chief complaint from what is really the issue that has brought on the crisis. For example, the patient may give a chief complaint of "feeling down," but a family member may report that he has been obsessed with his ex-girlfriend since a recent breakup and has been going to her house. The ex-girlfriend has had to call the police on 2 occasions. In this

case, the family member provided additional detail to the patient's more general complaint.

History of Present Illness

The patient's story should be heard. Invaluable information can be obtained just by listening to the patient. The patient's history should guide an exploration of diagnostic criteria to help arrive at a definitive diagnosis. The time frame during which symptoms developed should be determined. Stresses identified by the patient should be explored and the patient's support system or lack thereof should be reviewed. It is also important to identify issues related to safety of the patient and others. Suicide risk and risk of violence toward others should be discussed with the patient.

Past Psychiatric History

Psychiatric history should include past contacts with psychiatric care, past diagnoses, medication trials, hospitalizations, suicide attempts, history of violence, and the patient's current care providers. When possible, this history should be corroborated with current providers.

Past Medical History

All current and past medical illnesses and previous surgeries should be documented. Special attention should be paid to head injuries. Also, deceleration injuries that do not involve direct head trauma can result in brain injury.[13] Thus, a history of a motor vehicle collision in which the patient did not have a direct blow to the head but broke both femurs is significant. Determine all medications currently taken and why, including a review of any over-the-counter or herbal/alternative remedies that are being taken or recently have been taken. Allergies to medications should also be noted.

Substance Use History

A review of alcohol and street drug use, including the effect these have had on the patient's life, and any past treatment, should be obtained. This should be supplemented with questions about nicotine, caffeine, and other psychoactive substance use.

Social History

The social history provides a better understanding of who the patient is. Were there developmental problems? What is the patient's level of education? Has the patient had previous arrests? If the patient was in the military, does he have an honorable discharge? Does the patient have a consistent work history? Has the patient had a stable marriage or has he been married multiple times? Does he pay child support? Does the patient have spiritual concerns? While knowledge of past physical or sexual abuse can be important and can explain why the patient has responded in certain ways to behavioral management (such as restraint or seclusion), delving into abuse history is rarely appropriate in the emergency setting.

Family History

A complete family history should be obtained to include medical illness, mental illness, and substance use. Be sure to ask about family suicides or suicide attempts, as both are known risk factors for suicide.

Mental Status Examination

All components of the mental status examination should be included. Particular attention should be paid to the patient's appearance and behavior; affective state and stability; thought process; suicidal and homicidal ideation; the presence of psychotic symptoms; level of awareness; attention and concentration; judgment/insight; executive functions and reasoning; and reliability. If not already done, a screening cognitive examination, such as the Folstein Mini Mental State Examination[7] or the Brief Mental Status Examination[9] can be a helpful tool for assessing basic cognitive abilities and deficits.

OTHER ASPECTS OF EMERGENCY EVALUATION AND MANAGEMENT

Assessment for Risk of Suicide and Other Violence

An important part of the assessment of the agitated patient in the emergency setting is addressing the potential of harm to self or others. This will be a key focus in developing an appropriate disposition plan, but an exhaustive review of the evidence to use in suicide/violence risk assessment is beyond the scope of Project BETA. Therefore, in this article we will summarize the important points all clinicians should keep in mind.

Patients often arrive at an emergency department indicating they have thoughts, intent, or plan to harm themselves or others, or behaving in a way that suggests they may be dangerous. The emergency provider must quickly establish a treatment plan that will mitigate the risk of self-harm or violence toward others. Unfortunately, there is no specific tool that can be used to assess all such suicidal or potentially violent patients. While several scales are available, their utility in a busy emergency department setting is often rather limited. Further, while many such scales often have some utility in research settings, they do not have demonstrated predictive validity for clinical practice.[14] As such, a thorough understanding of the many static and dynamic risk factors for suicidal or violent behavior is needed. Relying solely on the patient's report that he or she is not suicidal or homicidal has been found to be inadequate.[15,16] Instead, a thorough mental status examination, a reasonable effort at obtaining collateral information, and a review of the patient's past behaviors, with a focus on suicidal or violent behaviors, are indicated.

In early stages of evaluation, careful attention should be given to collateral informants such as police or family members who may have vital information regarding recent acts of self-harm, aggression, threats made, and possible drug and alcohol intoxication. Often, the licensed provider responsible for treatment planning is not part of the triage process, and efforts

should be made to educate and train other clinical staff to gather pertinent clinical data, while it is easily obtainable, at the time when the patient presents to the emergency setting with others, whether family, emergency medical technicians, or police. In the evaluation of suicidality and homicidality, it is important to determine the nature of suicidal or violent thoughts in detail, including how often they occur, how long they last, and how the patient copes with such thoughts. Clinicians should ask specific questions to ascertain the urgency of these thoughts, with the understanding that they occur on a continuum. The assessment should include a risk factor review, including those that are modifiable. One especially important factor to assess in the emergency setting is access to guns, since this is a potentially modifiable risk factor with major impact. Other important areas of risk to assess include history of prior suicide attempts or acts of violence, substance use, limited support, and poor engagement or nonadherence with treatment. Protective factors should also be reviewed. These include strong spiritual beliefs, feeling that suicide or violence is immoral, custodial children or other family members under the patient's care, ability to identify reasons for living, and engagement in school or work. This will ultimately allow for a broader classification of risk and help in the determination of disposition. To be sure, this process does not allow for the prediction of suicide or violence, but rather, is a clinical judgment based on the available information to help estimate the likelihood of suicide or violence.[14,17]

Collateral History, Confidentiality, and Family Involvement

As discussed, collateral information should ideally be obtained from multiple sources including police and emergency personnel, physicians, nursing and other clinical staff in the emergency setting, and from family and friends who accompany the patient. Relevant historical information can be shared among those with a duty of care to the patient. However, ethical and legal issues of patient confidentiality arise with third parties. It is generally considered ethical and legally defensible practice to reveal what is medically necessary to third parties in an emergency, without the patient's consent. In addition, the duty to maintain confidentiality does not prevent the clinician from receiving information from third parties.[18] This is an important consideration when such information is necessary for thorough emergency assessment and management of the agitated patient.

Family and friends are often a good source of historical information and important collaborators in disposition planning in the emergency setting.[19] Additionally, recovery-based models consider family and peers to be an important part of the recovery process of mental illness. Clinical experience suggests that the presence of family or friends with an agitated patient can be both beneficial and detrimental, often during the same visit.[20] Often, the presence of family members can have a calming effect on patients initially, but may exacerbate agitation when there are apparent differences of opinion about management among the patient, family members, and clinical staff. One such situation is when a decision is made to restrain or involuntarily hospitalize the patient. Family members may need to be removed from view during procedures such as restraint or administration of parenteral medication to avoid escalation of the patient's agitation. Input from patients, their families, and peers about the emergency management of agitation should be an important part of practice when evaluating patients in the emergency setting.

Other Legal Issues

Medical-legal issues are often at the forefront of the assessment of the agitated patient. These include involuntary hospitalization and treatment, statutory reporting requirements (eg, child abuse) and "duty to warn" obligations (eg, Tarasoff requirements).[21,22] Laws that define when a clinician can place a patient on involuntary status vary among jurisdictions but generally include risk to the safety of self or others, significant impairment in self-care or grave disability and the need for treatment, or risk of deterioration in the presence of a mental disorder. The clinician should be familiar with legal requirements in the jurisdiction in which he or she practices, as statutes and case law may vary widely.[18]

Documentation

Documentation of sources of information for the patient's history should be included in the medical record. Collateral information obtained in addition to attempts to elicit or review relevant information, even if not available, should be included. The patient's consent for discussion with collateral sources should be noted. If the patient refuses to give permission, reasons for contacting others should be clearly documented. The relevant decision-making process related to disposition and statutory reporting obligations should form part of the patient's medical record.[23]

Ultimately, the results of the psychiatric assessment of the agitated patient should be documented in an organized manner in the medical record. In addition to a relevant patient history and mental status examination, a clinical impression should summarize the case and describe *who* the patient is and *why* he is presenting with agitation at this point in time. A summary of the risk assessment, including a discussion of risk factors for suicide or other violence, as well as protective factors, should be included. In addition, steps that have been taken to mitigate risk or strengthen protective factors, or steps that may still need to be taken to do so, should also be discussed. The rationale for the preferred disposition and overall management plan should be included as part of the clinical impression.[24]

CONCLUDING REMARKS

Initial psychiatric assessment of the agitated patient can often be quite challenging. As outlined in the related articles within this issue, de-escalation and other strategies may need to be used before or at the same time psychiatric assessment is

started. The possibility of medical etiologies must be considered first and foremost. Particular attention should be paid to the patient's appearance and behavior, level of awareness, attentional deficits, and cognitive abilities to rule out delirium/medical causes for the agitation. Affective state, thought process, suicidal and homicidal ideation, the presence of psychotic symptoms, judgment/insight, executive functions, and reasoning and reliability must ultimately also be assessed. The clinician may need to gather a significant amount of information from collateral sources. The focus of the evaluation is on developing a reasonable differential diagnosis, ascertaining safety and self-care concerns, and deciding how to manage the agitation. Developing the most appropriate treatment and disposition plan with the information gathered is more important than making a definitive diagnosis.

Address for Correspondence: Keith R. Stowell, MD, MSPH, Western Psychiatric Institute and Clinic, 3811 O'Hara St, Pittsburgh, PA 15213-2593. E-mail: stowellkr@upmc.edu.

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Verbal De-escalation of the Agitated Patient: Consensus Statement of the American Association for Emergency Psychiatry Project BETA De-escalation Workgroup

Janet S. Richmond, MSW*
Jon S. Berlin, MD†
Avrim B. Fishkind, MD‡
Garland H. Holloman Jr, MD, PhD§
Scott L. Zeller, MD||
Michael P. Wilson, MD, PhD¶
Muhamad Aly Rifai, MD, CPE#
Anthony T. Ng, MD, FAPA**

* Tufts University School of Medicine, Department of Psychiatry, Boston, Massachusetts
† Medical College of Wisconsin, Departments of Psychiatry and Emergency Medicine, Milwaukee, Wisconsin
‡ JSA Health Telepsychiatry, LLC, Houston, Texas
§ University of Mississippi Medical Center, Department of Psychiatry, Jackson, Mississippi
|| Alameda County Medical Center, Department of Psychiatry, Oakland, California
¶ UC San Diego Health System, Department of Emergency Medicine, San Diego, California
# Drexel University/Blue Mountain Health System, Department of Psychiatry, Lehighton, Pennsylvania
** Acadia Hospital, Bangor, Maine

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Agitation is an acute behavioral emergency requiring immediate intervention. Traditional methods of treating agitated patients, ie, routine restraints and involuntary medication, have been replaced with a much greater emphasis on a noncoercive approach. Experienced practitioners have found that if such interventions are undertaken with genuine commitment, successful outcomes can occur far more often than previously thought possible. In the new paradigm, a 3-step approach is used. First, the patient is verbally engaged; then a collaborative relationship is established; and, finally, the patient is verbally de-escalated out of the agitated state. Verbal de-escalation is usually the key to engaging the patient and helping him become an active partner in his evaluation and treatment; although, we also recognize that in some cases nonverbal approaches, such as voluntary medication and environment planning, are also important. When working with an agitated patient, there are 4 main objectives: (1) ensure the safety of the patient, staff, and others in the area; (2) help the patient manage his emotions and distress and maintain or regain control of his behavior; (3) avoid the use of restraint when at all possible; and (4) avoid coercive interventions that escalate agitation. The authors detail the proper foundations for appropriate training for de-escalation and provide intervention guidelines, using the "10 domains of de-escalation." [West J Emerg Med. 2012;13(1):17–25.]

INTRODUCTION

Traditional methods of treating agitated patients, ie, routine restraints and involuntary medication, have been replaced with a much greater emphasis on a noncoercive approach. Experienced practitioners have found that if such interventions are undertaken with genuine commitment, successful outcomes can occur far more often than previously thought possible. In the new paradigm, a 3-step approach is used. First, the patient is verbally engaged; then a collaborative relationship is established; and, finally, the patient is verbally de-escalated out of the agitated state. In some ways, this is a return to Lazare's methods published in an article written more than 35 years ago.[1]

The traditional goal of "calming the patient" often has a

dominant-submissive connotation, while the contemporary goal of "helping the patient calm himself" is more collaborative. The act of verbally de-escalating a patient is therefore a form of treatment in which the patient is enabled to rapidly develop his own internal locus of control.

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These objectives may be challenging to pursue in some situations and settings. For example, in an emergency department, both the clinician and patient can slip into irrational thinking or expediency at the price of engaging each other. A clinician who has many patients to see and too little time may prematurely use medication to avoid verbal engagement. However, using medication too quickly may seem dismissive, rejecting, or humiliating to the patient[2] and can lead to more agitation and violence.

Agitation is a behavioral syndrome that may be connected to different underlying emotions. Associated motor activity is usually repetitive and non–goal directed and may include such behaviors as foot tapping, hand wringing, hair pulling, and fiddling with clothes or other objects. Repetitive thoughts are exhibited by vocalizations such as, "I've got to get out of here. I've got to get out of here."[3] Irritability and heightened responsiveness to stimuli may be present,[4] but the association of agitation and aggression has not been clearly established.[5]

Agitation exists on a continuum, eg, from anxiety to high anxiety, to agitation, to aggression.[6] The agitated patient may be unable to engage in any conversation, and may be on the edge of new or repeated violence, requiring vastly different management than a person who may be willing and able to engage. The Project BETA (Best practices in Evaluation and Treatment of Agitation) guidelines[7] discussed in this section will help shape a practical, noncoercive approach to de-escalating agitated patients regardless of etiology or capacity to engage in a therapeutic relationship.

CLINICIAN'S APPROACH TO AGITATION

Emergency psychiatry is a well-established mental health discipline. However, the number of emergency psychiatrists and the volume of psychiatric crises they see are limited when compared to the number of emergency department physicians evaluating psychiatric emergencies. Interventions must often proceed with the agitated patient with, at best, a tentative diagnosis.

A paradigm that can be useful for both psychiatrists and emergency physicians is one in which the clinician uses rapid assessment and decision-making skills in an effort to quickly provide symptom relief. This relief, through verbal de-escalation and/or medication, enhances a positive clinician-patient relationship, decreases the likelihood of restraints, seclusion, and hospital admissions,[8] and prevents longer hospitalization, since the use of restraints has been associated with increased length of stay.[9,10] After initial stabilization of the patient's agitation, the clinician can work with the patient to establish a final diagnosis.

Regardless of underlying etiology, agitation is an acute emergency and "requires immediate intervention to control symptoms and decrease the risk of injury" to the patient or others.[11] While voluntary medication and environment planning are also important, verbal de-escalation and nonverbal communication are usually key to engaging the patient and helping him become an active partner in de-escalation.

Finally, each clinician must remember the 4 main reasons for using noncoercive de-escalation. First, when staff members physically intervene to subdue a patient, it tends to reinforce the patient's idea that violence is necessary to resolve conflict. As such, noncoercive de-escalation is a success for the patient and staff, and is in effect a form of treatment. Second, patients who are put in restraints are more likely to be admitted to a psychiatric hospital[8] and have longer inpatient lengths of stay.[9,10] Third, the Joint Commission and the Centers for Medicare and Medicaid Services consider low restraint rates a key quality indicator, and fourth, staff and patients are less likely to get hurt when physical confrontation is averted.

DE-ESCALATION OF AGITATED PATIENTS IN THE EMERGENCY SETTING

General principles of verbal de-escalation can be found in specific psychotherapies, linguistic science, law enforcement, martial arts, and the nursing profession. Clinicians who work with agitated patients on a daily basis have perfected skills that frequently are in line with principles found in these resources. However, a review of the literature indicates that scientific studies and medical writings on verbal de-escalation are few and lack descriptions of specific techniques and efficacy.

There is indirect evidence from pharmacologic studies of agitation that verbal techniques can be successful in a substantial percentage of patients. In a recent study, patients were excluded from a clinical trial of droperidol if they were successfully managed with verbal de-escalation; however, the specific verbal de-escalation techniques were not identified or studied.[12]

The following guidelines were therefore developed by the consensus of the authors and a review of the limited available literature on verbal de-escalation.[13–15]

GUIDELINES FOR ENVIRONMENT, PEOPLE, PREPAREDNESS

Guideline: Physical Space Should Be Designed for Safety

The physical environment is important for the safe management of the agitated patient. Moveable furniture allows for flexible and equal access to exits for both patient and staff. The ability to quickly remove furniture from the area can expedite the creation of a safe environment. Some emergency

departments prefer stationary furniture, so that the patient cannot use the objects as weapons, but this may create a false sense of security. There should be adequate exits, and extremes in sound, wall color, and temperature of the environment should be avoided to minimize abrasive sensory stimulation. Be mindful, also, of the potential for an agitated patient's throwing objects that may cause injuries to others. Any objects, such as pens, sharp objects, table lamps, etc that may be used as weapons should be removed or secured. The clinician should closely monitor any objects that cannot be removed.

Guideline: Staff Should Be Appropriate for the Job

Clinicians who work in acute care settings must be good multitaskers and tolerate rapidly changing patient priorities. In this environment, tolerating and even enjoying dealing with agitated patients takes a certain temperament, and all clinicians are encouraged to assess their temperament for this work.

Agitated patients can be provocative and may challenge the authority, competence, or credentials of the clinician. Some patients, in order to deflect their own sense of vulnerability, are exquisitely sensitive in detecting the clinician's vulnerability and focusing on it. To work well with agitated patients, staff members must be able to recognize and control countertransference issues and their own negative reactions. These include the clinician's understanding of his own vulnerabilities, tendencies to retaliate, argue, or otherwise become defensive and "act-in" with the patient. Such behaviors on the part of the clinician only serve to worsen the situation. Clinicians need to also recognize their limits in dealing with an agitated patient, as it can be quite taxing, and sometimes the best intervention is knowing when to seek additional help.

Security and police officers, who work with agitated patients, must accept that a patient's abnormal behavior is a manifestation of mental illness and that de-escalation is the preferred treatment of choice. The Crisis Intervention Team (CIT) model is a police-based, first-responder program that has been implemented nationwide. Persons taken into custody because of suspected mental illness are taken to a psychiatric emergency service or other facility where the person can receive psychiatric evaluation and treatment. CIT officers usually volunteer for these teams so that an officer is not forced into taking on a role that he does not want. Training of officers is provided by mental health professionals, legal experts, and advocates.[16,17]

Natural skill at verbal de-escalation exists on a continuum. However, almost anyone can learn de-escalation techniques and use them successfully if he is well trained and adopts a certain skill set. The most essential skill is a good attitude, starting with positive regard for the patient and the capacity for empathy. Staff should be able to recognize that the patient is doing the best he can under the circumstances, ie, the patient is experiencing difficulty in conforming to what is expected of him. Clinicians in emergency settings also will need to be skilled at recognizing that the inability to conform is due to either cognitive impairment—for example, delirium, psychosis, intoxication, and intellectual disability—or the patient's lack of the skills needed to effectively get his needs met, eg, personality disorder.

Guideline: Staff Must Be Adequately Trained

Training in management of the agitated patient decreases the tendency of clinicians to avoid working with these patients. The American Psychiatric Association Task Force on Psychiatric Emergency Services[18] has recommended that staff receive annual training on managing behavioral emergencies. This training is analogous to advanced cardiovascular life support training, ie, knowledge about skills can be taught in a classroom or can be learned from a book, but skills come only with practice. De-escalation skills can be learned by role playing and can be practiced in day-by-day encounters with nonagitated patients who are considered to be difficult in the sense of not conforming to what the clinician expects.

All persons who work with agitated patients should receive training in de-escalation techniques. A person, who is appropriate for the job, as discussed earlier, should be the one who works directly with the patient. A psychiatrist, emergency physician, or any other healthcare worker can become proficient at de-escalation, and any of these can engage the patient and perform de-escalation.

De-escalation frequently takes the form of a verbal loop in which the clinician listens to the patient, finds a way to respond that agrees with or validates the patient's position, and then states what he wants the patient to do, eg, accept medication, sit down, etc. The loop repeats as the clinician listens again to the patient's response.[19] The clinician may have to repeat his message a dozen or more times before it is heard by the patient. Yet, beginning residents, and other inexperienced clinicians, tend to give up after a brief attempt to engage the patient, reporting that the patient won't listen or won't cooperate.[20]

The amount of time permitted for verbal de-escalation may vary depending on the setting and other constraints. However, it is the consensus of Project BETA De-escalation Workgroup members that verbal de-escalation frequently can be successful in less than 5 minutes. Its potential advantages in safety, outcome, and patient satisfaction indicate it should be attempted in the vast majority of agitation situations, even in very busy emergency settings.

Even the most complicated cases can be managed with a little additional time. Assuming that a single interaction of listening and responding takes less than a minute, then a dozen repetitions of the clinician's message would take 10 minutes at the most. De-escalation, when effective, can avoid the need for restraint. Taking the time to de-escalate the patient and working with him as he settles down can be much less time-consuming than placing him in restraints, which requires additional resources once he is restrained.

There are patients who cannot be effectively engaged and verbally de-escalated, eg, a delirious patient. However, training

Table 1. Behavioural Activity Rating Scale (BARS).[21]

1 = Difficult or unable to rouse
2 = Asleep but responds normally to verbal or physical contact
3 = Drowsy, appears sedated
4 = Quiet and awake (normal level of activity)
5 = Signs of overt (physical or verbal) activity, calms down with instructions
6 = Extremely or continuously active, not requiring restraint
7 = Violent, requires restraint

should emphasize that a patient may not respond to initial efforts to engage him in de-escalation and that persistence is indicated, especially when the patient is not showing signs of further escalation that is moving toward violence.

Guideline: An Adequate Number of Trained Staff Must Be Available

Working with an agitated patient is a team effort and there must be an adequate number of people to provide for verbal de-escalation, offer the possibility of voluntary medication, and maintain safety if the patient's agitation escalates to violence. There is also a benefit in having enough people to provide a nonverbal communication to the patient that violence on the part of the patient will not be acceptable behavior. In a busy emergency service, the de-escalation team should consist of 4 to 6 team members made up of nurses, clinicians, technicians, and police and security officers, if available.

Guideline: Use Objective Scales to Assess Agitation

The use of objective scales to measure agitation can help mitigate defensive behaviors on the part of staff that might result in their avoiding or "ignoring" early signs of agitation. One such scale that is quite simple and easy to implement is the Behavioural Activity Rating Scale (BARS; Table 1).[21]

The initial BARS score should be based not only on the patient's presentation, but also on his behavior before arrival at the emergency facility. Any score other than a 4 should trigger an evaluation by a clinician and establish the urgency of that evaluation. Other available scales include the Overt Aggression Scale,[22] the Scale for the Assessment of Aggressive and Agitated Behaviors,[23] and the Staff Observation Aggression Scale.[24]

GENERAL DE-ESCALATION GUIDELINES

Guideline: Clinicians Should Self-Monitor and Feel Safe When Approaching the Patient

A clinician cannot be effective if he has too much emotion or is frightened by the patient. Keeping the clinician safe is the first step toward patient safety. Approximately 90% of all emotional information and more than 50% of the total information in spoken English is communicated not by what one says but by body language, especially tone of voice.[25]

Table 2. Ten domains of de-escalation.[27]

1. Respect personal space
2. Do not be provocative
3. Establish verbal contact
4. Be concise
5. Identify wants and feelings
6. Listen closely to what the patient is saying
7. Agree or agree to disagree
8. Lay down the law and set clear limits
9. Offer choices and optimism
10. Debrief the patient and staff

When the clinician approaches the agitated patient, he must monitor his own emotional and physiologic response so as to remain calm and, therefore, be capable of performing verbal de-escalation.[26]

Guideline: 10 Domains of De-Escalation Exist That Help Clinicians' Care of Agitated Patients

Review of the literature establishes 10 domains of de-escalation (Table 2).[27]

Domain I: Respect Personal Space

Key Recommendation: Respect the Patient's and Your Personal Space. When approaching the agitated patient, maintain at least 2 arm's lengths of distance between you and the patient. This not only gives the patient the space he needs, but also gives the clinician the space needed to move out of the way if the patient were to kick or otherwise strike out. The clinician may want to give himself more distance in order to feel safe; and, if a patient tells you to get out of the way, do so immediately. Both the patient and the clinician should be able to exit the room without feeling that the other is blocking his way.

A high percentage of patients have a past history of trauma, and the emergency experience has the potential for repeating the traumatic experience when specific aspects of personal space are ignored. A person who lives on the street may be very sensitive about protecting his belongings. Those who have been sexually abused may be apprehensive about being unclothed, which can increase their sense of vulnerability and cause humiliation.

Domain II: Do Not Be Provocative

Key Recommendation: Avoid Iatrogenic Escalation. The clinician must demonstrate by body language that he will not harm the patient, that he wants to listen, and that he wants everyone to be safe. Hands should be visible and not clenched. Avoid concealed hands, which imply a concealed weapon.[20] Knees should be slightly bent. The clinician should avoid directly facing the agitated patient and should stand at an angle

to the patient so as not to appear confrontational. A calm demeanor and facial expression are important. Excessive, direct eye contact, especially staring, can be interpreted as an aggressive act. Closed body language, such as arm folding or turning away, can communicate lack of interest. It is most important that the clinician's body language be congruent with what he is saying. If not, the patient will sense that the clinician is insincere or even "faking it" and may become more agitated and angry. It is also important to monitor closely that other patients or individuals do not provoke the patient further.

According to Lazare and Levy,[28] humiliation is an aggressive act where a person has threatened another person's integrity and very self. In some cases, humiliation itself can be traumatic. Therefore, do not challenge the patient, insult him, or do anything else that can be perceived as humiliating.

Domain III: Establish Verbal Contact

Key Recommendation: Only 1 Person Verbally Interacts with the Patient. The first person to make contact with the patient should be the person designated to de-escalate the patient. If that person is not trained or is otherwise unable to take on this role, another person should be designated immediately.

Multiple people verbally interacting can confuse the patient and result in further escalation. While the designated person is working with the patient, another team member should alert staff to the encounter, while removing innocent bystanders.

Key Recommendation: Introduce Yourself to the Patient and Provide Orientation and Reassurance. A good strategy is to be polite. Tell the patient your title and name. Rapidly diminish the patient's concerns about your role by explaining that you are there to keep him safe and make sure no harm comes to him or anyone else in the emergency setting. If the patient is very agitated, he may need additional reassurance that the clinician wants to help him regain control. Orient the patient as to where he is and what to expect. If the patient's name is unknown, ask for his name. Judgment is required in deciding whether to call the person by his first or last name. Although some prefer calling all patients by their last names, this formality, in some situations, can add to a patient's suspicion and appear patronizing. When in doubt, it is best to ask the patient how he prefers to be addressed; this act communicates that he is important and, from the very beginning of the interaction, that he has some control over the situation.

Domain IV: Be Concise

Key Recommendation: Be Concise and Keep It Simple Since agitated patients may be impaired in their ability to process verbal information, use short sentences and a simple vocabulary. More complex verbalizations can increase confusion and can lead to escalation. Give the patient time to process what has been said to him and to respond before providing additional information.

Key Recommendation: Repetition Is Essential to Successful De-escalation. This involves persistently repeating your message to the patient until it is heard. Since the agitated patient is often limited in his ability to process information, repetition is essential whenever you make requests of the patient, set limits, offer choices, or propose alternatives. This repetition is combined with other assertiveness skills that involve listening to the patient and agreeing with his position whenever possible.[19]

Domain V: Identify Wants and Feelings

Examples of wants include succorance, the wish to ventilate to an empathic listener, a request for medication, some administrative intervention, such as a letter to an employer, or intervening with a difficult spouse or parent. Whether or not the request can be granted, all patients need to be asked what their request is.[1] A statement like, "I really need to know what you expected when you came here," is essential, as is the caveat "Even if I can't provide it, I would like to know so we can work on it."

Key Recommendation: Use Free Information to Identify Wants and Feelings. "Free information" comes from trivial things the patient says, his body language, or even past encounters one has had with the patient.[19] Free information can help the examiner identify the patient's wants and needs. This rapid connection based on free information allows the clinician to respond empathically and express a desire to help the patient get what he wants, facilitating rapid de-escalation of agitation.

A sad person wants something he has given up hope of having. A patient who is fearful wants to avoid being hurt. In a later discussion of aggression, it will be apparent that the aggressive patient has specific wants also, and identifying these wants is important for the management of the patient.

Domain VI: Listen Closely to What the Patient Is Saying

Key Recommendation: Use Active Listening. The clinician must convey through verbal acknowledgment, conversation, and body language that he is really paying attention to the patient and what he is saying and feeling. As the listener, you should be able to repeat back to the patient what he has said to his satisfaction. Such clarifying statements as "Tell me if I have this right. . ." is a useful technique. Again, this does not mean necessarily that you agree with the patient but, rather, that you understand what he is saying.

Key Recommendation: Use Miller's Law. Miller's law states, "To understand what another person is saying, you must assume that it is true and try to imagine what it could be true of."[25] If you follow this law, you will be trying to understand. If you are truly trying to imagine how it could be true, you will be less judgmental, and the patient will sense that you are interested in what he is saying and this will significantly improve your relationship with the patient. For example, if the patient's agitation is driven by the delusion that someone is

Table 3. Summary of strategies for broaching the topic of medication/escalating persuasion techniques.

What helps you at times like this?	STRATEGY: Invite the patient's ideas.
I think you would benefit from medication.	STRATEGY: Stating a fact.
I really think you need a little medicine.	STRATEGY: Persuading.
You're in a terrible crisis. Nothing's working. I'm going to get you some emergency medication. It works well and it's safe. If you have any serious concerns, let me know.	STRATEGY: Inducing.
I'm going to have to insist.	STRATEGY: Coercing. Great danger, last resort.

following him and intends to cause him harm, you can imagine how this is true from the patient's standpoint and engage the patient in conversation as to why this is happening to him and who would want to harm him. This will convey your interest and will result in the patient engaging in conversation about that which is driving his agitation. By engaging in conversation, the patient will begin to see that you care, which in turn, fosters de-escalation.

Domain VII: Agree or Agree to Disagree

Fogging is an empathic behavior in which one finds something about the patient's position with which he can agree.[19] It can be very effective in developing one's relationship with the patient. There are 3 ways to agree with a patient. The first is agreeing with the truth. If the patient is agitated after 3 attempts to draw his blood, one might say, "Yes, she has stuck you 3 times. Do you mind if I try?"The second is agreeing in principle. For the agitated patient who is complaining that he has been disrespected by the police, you don't have to agree that he is correct but you can agree with him in principle by saying, "I believe everyone should be treated respectfully." The third is to agree with the odds. If the patient is agitated because of the wait to see the doctor and states that anyone would be upset, an appropriate response would be, "There probably are other patients who would be upset also." Using these techniques, it is usually easy to find a way of agreeing, and one should agree with the patient as much as possible. Clinicians may find themselves in a position where they are being asked to agree with an obvious delusion or something else the clinician can obviously have no knowledge of. In this situation, acknowledge that you have never experienced what the patient is experiencing but that you believe that he is having that experience. However, if there is no way to honestly agree with the patient, agree to disagree.

Domain VIII: Lay Down the Law and Set Clear Limits

Key Recommendation: Establish Basic Working Conditions. It is critical that the patient be clearly informed about acceptable behaviors. Tell the patient that injury to him or others is unacceptable. If necessary, tell the patient that he may be arrested and prosecuted if he assaults anyone. This should be communicated in a matter-of-fact way and not as a threat.

Key Recommendation: Limit Setting Must Be Reasonable and Done in a Respectful Manner. Set limits demonstrating your intent and desire to be of help but not to be abused by the patient. If the patient is causing the clinician to feel uncomfortable, this must be acknowledged. Often telling the patient that his behavior is frightening or provocative is helpful if it is matched with an empathic statement that the desire to help can be interrupted or even derailed if the clinician feels angry, fearful, etc.

The bottom line is that good *"*working conditions*"* require that both patient and clinician treat each other with respect. Being treated with respect and dignity must go both ways. Violation of a limit must result in a consequence, which (1) is clearly related to the specific behavior; (2) is reasonable; and (3) is presented in a respectful manner.

Some behaviors, eg, punching a wall or even breaking a chair, may not automatically indicate the need for seclusion or restraint, and the patient can continue to be de-escalated with some increase in limit setting and consequences. Reassure the patient that you want to help him regain control and establish acceptable behavior.

Key Recommendation: Coach the Patient in How to Stay in Control. Once you have established a relationship with the patient and determined that he has the capability to stay in control, teach him how to stay in control. Use gentle confrontation with instruction: *"*I really want you to sit down; when you pace, I feel frightened, and I can't pay full attention to what you are saying. I bet you could help me understand if you were to calmly tell me your concerns.*"*

Domain IX: Offer Choices and Optimism

Key Recommendation: Offer Choices. For the patient who has nothing left but to fight or take flight, offering a choice can be a powerful tool. Choice is the only source of empowerment for a patient who believes physical violence is a necessary response. In order to stop a spiraling aggression from turning into an assault, be assertive and quickly propose alternatives to violence. While offering choices, also offer things that will be perceived as acts of kindness, such as blankets, magazines, and access to a phone. Food and something to drink may be a choice the patient is willing to accept that will stall aggressive behaviors. Be mindful that these choices must be realistic.

Never deceive a patient by promising something that cannot be provided for him. For example, a patient should not be promised a chance to smoke when the hospital has a no-smoking policy.

Key Recommendation: Broach the Subject of Medications. The goal of medicating the agitated patient is not to sedate but to calm him. As Allen and colleagues[11] point out, a calm, conscious patient is one who can participate in his own care and work with the crisis clinician toward an appropriate treatment disposition, which is of benefit to the patient and also to the staff. It can decrease length of stay and make the emergency department experience a positive one.

When medications are indicated, offer choices to the patient. Timing is essential. Do not rush to give medication but, at the same time, do not delay medication when needed. Using increasing strategies of persuasion is a sound technique (Table 3). For example, the first step is not to mention medication at all but to ask the patient what he needs, what works. Try to get the request for medication to come from the patient himself, or perhaps the patient has a better idea.

If the patient does not mention medication and the clinician believes it is indicated, then state clearly to the patient that you think he would benefit from medication. Ask the patient what medication has helped him in the past or state, *"*I see that you're quite uncomfortable. May I offer you some medication?*"*

Gentle confrontation may also be useful: *"*It's important for you to be calm in order for us to be able to talk. How can that be accomplished? Would you be willing to take some medication?*"*

Another step is one just short of involuntary medication. *"*Mr Smith, you're experiencing a psychiatric emergency. I'm going to order you some emergency medicine.*"* This strategy is authoritative, as in being knowledgeable and self-assured, possessing expertise, having the ability to explain one's thinking, and being persuasive. Giving the patient a choice in either oral or parenteral administration can help give the patient some control. He may willingly take medication if the means of administration is a choice, even if the administration of medication itself is not a choice. Appealing to the patient's desire to stay in control and the clinician's mandate to keep everyone safe, one might say to the patient: *"*I can't let any harm come to you or anyone else*"* or *"*I need to protect you from hurting someone, so I would like for you to take some medication to help you stay in control.*"* The clinician then says to the patient as many times as necessary, *"*Would you like to take medication by mouth or by a shot?*"* Emphasizing the protection aspect is very important and can be effective in empowering the patient to stay in control. *"*I feel medications can help, would you like a pill you can swallow, a pill that will melt in your mouth, or a liquid? If you agree to take a pill by mouth you can avoid taking a shot.*"* Even when there is no choice but to give an injection, the clinician can give a choice as to which drug is to be used, emphasizing that one has a more beneficial side-effect profile.

Finally, when verbal attempts to de-escalate fail, more coercive measures such as restraints or injectable medication may be necessary to ensure safety but always as a last resort.

Key Recommendation: Be Optimistic and Provide Hope. Be optimistic but in a genuine way. Let patients know that things are going to improve and that they will be safe and regain control. Give realistic time frames for solving a problem and agree to help the patient work on the problem. When the patient states, *"*I want to get out of here,*"* the clinician can respond, *"*I want that for you as well; I don't want you to have to stay here any longer than necessary; how can we work together to help you get out of here?*"*

Domain X: Debrief the Patient and Staff

Key Recommendation: Debrief the Patient. After any involuntary intervention with an agitated patient, it is the responsibility of the clinician who ordered these interventions to restore the therapeutic relationship to alleviate the traumatic nature of the coercive intervention and to decrease the risk of additional violence.

Start by explaining why the intervention was necessary. Let the patient explain events from his perspective. Explore alternatives for managing aggression if the patient were to get agitated again. Teach the patient how to request a time out and how to appropriately express his anger. Explain how medications can help prevent acts of violence and get the patient's feedback on whether his concerns have been addressed. Finally, debrief the patient's family who witnessed the incident.

Once the patient is calm, the clinician can acknowledge and work with the patient on a deeper level, help put the patient's concerns into perspective, and assist him in problem solving his initial precipitating situation. Since prevention of agitation is the best way to treat it, planning with the patient is best: *"*What works when you are very upset as you were today? What can we/you do in the future to help you stay in control?*"*

Key Recommendation: Debrief the Staff. If restraint or force needs to be used, it is important that the staff be debriefed on the actions after the event. Staff should feel free to suggest both what went well during the episode, and what did not, and recommend improvements for the next episode.

THE AGGRESSIVE PATIENT

As previously noted the extent of aggression associated with agitation has not been clearly established.[5] However, some agitated patients are aggressive and the approach to the patient depends upon the type of aggression. Moyer[29] has defined several types of aggression, some of which are commonly seen in the emergency setting. Types of aggression also have been identified in the setting of a correctional facility[30] and by martial arts instructors.[31] These identified types can be placed in Moyer's

classification and are important because principles of management have been developed for each of the different types of aggression. Some of the management techniques used in correctional facilities and taught in the martial arts are not recommended for use in the healthcare setting. However, the principles allow us to develop techniques appropriate to the healthcare setting and are discussed here. It will be apparent that there is always something the patient wants. As discussed earlier, identifying the patient's wants is important and, in this case, determines how the patient is managed.

Instrumental aggression is used by those who have found they can get what they want by violence or threats of violence. This aggression is not driven by emotion and can be handled by using unspecified counter offers to the aggressor's threat. If a patient threatens to hurt someone if he doesn't get a cigarette, a counter offer might be, *"*I don't think that's a good idea.*"* The patient's next response may be, *"*What do you mean?*"* A counter offer would be, *"*Let's not find out.*"*

Fear driven aggression is not self defense. The patient wants to avoid being hurt and may attack to prevent someone from hurting him. Give the fearful patient plenty of space. Do not have a show of force or in any other way intimidate the patient or make him feel threatened, as this will feed into the patient's belief that he is going to be hurt. De-escalation involves matching the patent's pace until he begins to focus on what is being said rather than his fear. If the patient says, *"*Don't hurt me. Don't hurt me.*"* Counter with the same pace by saying, *"*You're safe here. You're safe here.*"* Try to decrease the pace to help the patient calm down.

Irritable aggression comes in 2 forms. The first is the patient who has had boundaries violated. Someone has cheated him, humiliated him, or otherwise emotionally wounded him. He is angry and trying to put his world back together, ie, he is trying to regain his self-worth and integrity. This patient wants to be heard and have his feelings validated. This type of aggression is identified by the patient's telling you what has made him angry. De-escalation involves setting conditions for the patient to be heard. Fogging and the broken record approach[19] are most helpful. A typical scenario is the patient who found out that his girlfriend had cheated on him. His friends kidded him and a fight ensued. He was brought in by police. On arrival the patient is furious. He states that his girlfriend had cheated on him and that the police are treating him unfairly. The initial response is to agree in principle that the patient's anger is justified. This is followed by telling the patient that you want to know more but cannot until he regains control so that *"*we can talk.*"* The patient may respond that nobody understands. The response is that he may be right but you would like to try to understand. This loop may need repeated a dozen or more times before the patient complies.

The second form of irritable aggression occurs in persons who are chronically angry at the world and are looking for an excuse to *"*go off.*"* They give no reason for their anger. They want to release the constant pressure resulting from their world view. They make unrealistic and erratic demands and use these as an excuse to attack when their demands are not met. They get enjoyment out of creating fear and confusion and may make feigned attacks to intimidate those who are working with them. Do not react in a startled or defensive way. These patients are looking for an emotional response from anyone who is an audience. Don't give them one and remove all other patients, unnecessary staff members, and bystanders from the area. Use emotionless responses. De-escalation involves giving the patient choices other than violence to get what he wants. As he makes erratic demands, use the broken record to return to the options you can offer. Let him know you will work with him but only when he is willing to be cooperative. Set firm limits to protect staff and other patients and intervene with restraint if the limit is violated. Unfortunately, many of these patients will test the limit by doing just what you have asked them not to do and end up in restraints.

SUMMARY

Verbal de-escalation techniques have the potential to decrease agitation and reduce the potential for associated violence, in the emergency setting. But while much has been written on the psychopharmacologic approaches to agitated patients, until now there has been relatively little discussion about verbal methods.

Modern clinical thinking endorses less coercive interventions, in which the patient becomes a collaborative partner with staff members in managing behavior. These approaches may result in many benefits over traditional procedures. Patients spiraling into agitation can be calmed without forced medication or restraint; most importantly, such benign treatment can empower the patient to stay in control while building trust with caregivers. This may help patients to confidently seek help earlier in the future, and avoid subsequent episodes of agitation altogether.

Address for Correspondence: Janet S. Richmond, MSW, 575 Chestnut St, Waban, MA 02468. E-mail: JanetRichmond@att.net.

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The Psychopharmacology of Agitation: Consensus Statement of the American Association for Emergency Psychiatry Project BETA Psychopharmacology Workgroup

Michael P. Wilson, MD, PhD*
David Pepper, MD†
Glenn W. Currier, MD, MPH‡
Garland H. Holloman Jr, MD, PhD§
David Feifel, MD, PhD||

* UC San Diego Health System, Department of Emergency Medicine, San Diego, California
† Hartford Hospital/Institute of Living, Department of Psychiatry, Hartford, Connecticut
‡ University of Rochester Medical Center, Departments of Psychiatry and Emergency Medicine, Rochester, New York
§ University of Mississippi Medical Center, Department of Psychiatry, Jackson, Mississippi
|| UC San Diego Health System, Department of Psychiatry, San Diego, California

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Agitation is common in the medical and psychiatric emergency department, and appropriate management of agitation is a core competency for emergency clinicians. In this article, the authors review the use of a variety of first-generation antipsychotic drugs, second-generation antipsychotic drugs, and benzodiazepines for treatment of acute agitation, and propose specific guidelines for treatment of agitation associated with a variety of conditions, including acute intoxication, psychiatric illness, delirium, and multiple or idiopathic causes. Pharmacologic treatment of agitation should be based on an assessment of the most likely cause of the agitation. If agitation results from a delirium or other medical condition, clinicians should first attempt to treat the underlying cause instead of simply medicating with antipsychotics or benzodiazepines. [West J Emerg Med. 2012;13(1):26–34.]

INTRODUCTION

The proper management of an agitated patient is essential to keep staff safe and ensure appropriate treatment for the patient. Most emergency physicians think of agitation as one of the simplest cases to treat, with haloperidol being a common approach in many emergency departments.[1–4] In most circumstances, nonpharmacologic methods of behavior control, such as a verbal intervention, de-escalation, or even nicotine replacement therapy, may be helpful initially to manage agitated patients.[5,6] When medications are required, second-generation antipsychotics, preferred by many psychiatrists over first-generation antipsychotics for long-term management of psychiatric illnesses, have also become increasingly used in the acute setting for management of agitation.[7] This paper represents consensus recommendations from a workgroup of the American Association for Emergency Psychiatry. This workgroup convened in 2010–2011 to recommend best practices in the use of medication to manage agitated patients in the emergency setting.[8]

THE RATIONALE FOR USING MEDICATION

Agitation is prevalent in the emergency setting. The National Emergency Department Safety Study, for instance, documented that at least 25% of emergency department staff felt safe at work "sometimes," "rarely," or "never."[9] In addition, the 2010 Emergency Nurses Association study on violence in the workplace reported that more than half of emergency nurses had been verbally or physically threatened at work within the preceding 7 days.[10] Agitation can also have effects on patients as well, with case reports of death due to untreated excited delirium.[11,12]

Calming of agitated patients therefore is of primary

importance. When initial verbal methods have failed to calm the patient, medications may become necessary. One of the first crucial steps in prescribing medication is the establishment of a provisional diagnosis as to its cause. It is often not possible to make a definitive diagnosis but clinicians should attempt a diagnosis of the most likely cause, since this can guide the choice of medication following guidelines discussed later. The timing of the administration of medication can be crucial to the outcome of successfully managing an agitated patient. If an agitated patient is medicated too aggressively or too early, it may hinder psychiatric evaluation. If the patient is medicated too late, it places the patient, staff, and others at increased risk for harm. In addition, the agitation may also become more pronounced, and greater doses or repeated medication administration may be required to abort the agitation.

THE GOALS OF USING MEDICATION

The goal of using medication is to calm the patient so that he or she can be more accurately assessed by clinicians. Medication used in this manner is consistent with current guidelines on medication administration, which state that the proper endpoint of medication administration is calming without inducing sleep.[7,13] In the acute setting, this more easily permits a diagnosis of the underlying cause of the agitation and allows patients to have some participation in their own care. More practically, however, patients who are not asleep are easier to discharge from the emergency department. Whether this matters for waiting times is controversial, with recent research indicating that the longest length of time that patients with psychiatric illnesses spend in the emergency department actually occurs between consultant disposition and discharge.[14] However, between emergency departments, the most variable length of time that patients spend is between triage and contacting of psychiatry consultants, thus potentially allowing for improvements in these times by careful use of medication.

TYPES OF MEDICATION

There is no type of medication considered to be "best" in all cases of agitation but 3 general classes of medication have been studied and used most frequently for agitation, including first-generation antipsychotics, second-generation antipsychotics, and benzodiazepines. Three routes of administration are possible (though not for each medication): oral/oral fast-dissolving tablets, intramuscular, or intravenous. The workgroup believes that patients should be involved, if possible, in both the selection of the type and the route of any medication.

Although antipsychotics and benzodiazepines may manage the level of agitation a patient exhibits, this does not imply that these medications are doing so by directly addressing the underlying etiology of the agitation. For example, a large number of physiologic (eg, hypoxia) and metabolic (eg, hypoglycemia) perturbations that compromise brain function can produce delirium that is associated with agitation. Treatment to correct the specific underlying medical disturbance is the definitive and preferred treatment of agitation in such cases, but this article will not attempt to address the optimal treatments for such medical disturbances or the other varied etiologies of agitation. Rather, this article will discuss best-practice pharmacologic approaches to use when agitation requires emergent management before stabilization of the underlying etiology.

The Use of First-Generation Antipsychotics

Typical or first-generation antipsychotics (FGA) have a long history of use for treatment of agitation. The exact mechanism of calming with FGAs is unknown but most likely due to their inhibition of dopamine transmission in the human brain, which reduces the underlying psychotic symptoms causing the agitation. In addition, some FGAs are structurally similar to the human inhibitory neurotransmitter gamma-aminobutyric acid (GABA) and interact with the human GABA receptor at high doses.[15]

The phenothiazines, a class of medication that includes low-potency antipsychotics such as chlorpromazine (Thorazine), the first FGA approved and marketed by the US Food and Drug Administration (FDA), have a propensity to cause more hypotension, more anticholinergic side effects, and lower the seizure threshold, compared to FGAs such as haloperidol.[16] Thus, phenothiazines are not preferred for the treatment of acute agitation.

Haloperidol, an FGA belonging to the butyrophenone class, is a highly potent and selective antagonist of the dopamine-2 (D2) receptor. Haloperidol, which is FDA approved for oral or intramuscular use in schizophrenia, has a long track record of effective and safe use for the treatment of agitation in the acute setting. This drug is by far the most common FGA currently used to treat acute agitation.[3,17] Droperidol, another butyrophenone with D2 receptor–blocking effects, has not been approved for psychiatric use but is approved as a preanesthetic to reduce nausea and vomiting associated with anesthesia. It has also been used widely in acute settings to treat agitation.

Both haloperidol and droperidol have minimal effects on vital signs, negligible anticholinergic activity, and minimal interactions with other nonpsychiatric medications. Unfortunately, both medications have important side effects. Notably, droperidol and haloperidol have a propensity to lengthen QTc intervals. Cases of torsades de pointes (TdP) have been reported with both drugs. There is much controversy regarding the degree and clinical significance of this QTc prolongation, and much research has indicated that clinically adverse cardiac effects are rare occurrences. Nevertheless, both drugs carry warnings about QTc prolongation in labeling of which physicians should be aware. The haloperidol label warning, for instance, indicates that "Higher doses and intravenous administration of haloperidol appear to be associated with a higher risk of QT prolongation and TdP."[18]

This has led to an increasing number of hospitals implementing restrictive guidelines on the use of intravenous haloperidol, typically requiring electrocardiogram (ECG) monitoring during administration. Regardless of the true clinical risk, however, it seems prudent for physicians to avoid intravenous administration of haloperidol (which is not an FDA-approved route of administration for this medication), especially for patients who are taking other medication that can prolong QTc, who have a preexisting long QTc, or who have other conditions predisposing to TdP or QTc prolongation, such as underlying cardiac abnormalities, electrolyte imbalances (particularly hypokalemia and hypomagnesemia), or hypothyroidism. When haloperidol must be administered intravenously, the dose should be limited to 5 to 10 mg/day and administered in conjunction with continuous ECG monitoring.

Droperidol carries even more stringent warnings from the FDA about QTc prolongation and TdP. In 2007, this warning was upgraded to a black box, the most serious warning the FDA can require. This warning has subsequently proved highly controversial, as many reviews of the FDA data have claimed that it was based upon a very limited number of adverse events, mostly involving doses of droperidol much higher than those typically used to treat agitation (for an excellent review of the data see Jackson et al[19]). Other evidence also suggests that droperidol does not warrant such strong safety concerns. For instance, Isbister et al[20] found that doses of up to 10 mg of droperidol had fewer adverse events than the use of midazolam in agitated emergency department patients, and Shale et al[21] did not find a single case of a clinically significant adverse cardiac event in more than a decade of treating psychiatric emergencies with droperidol (typically 5 mg) in a busy emergency psychiatry unit. Emergency department–based studies, such as by Martel and colleagues,[22] have even indicated that droperidol may have better efficacy and fewer side effects than ziprasidone, a second-generation antipsychotic approved for agitation. The FDA has indicated that it would revisit the evidence for droperidol's black-box warning either through an internal review or through review of an external study.[19] Until such time as the FDA warning is modified or removed, however, it is prudent for clinicians to avoid using droperidol for agitation, especially because it is not FDA approved for psychiatric use.

In addition to cardiac effects, haloperidol and droperidol carry a risk of inducing acute extrapyramidal side effects (EPS) such as dystonia or neuroleptic malignant syndrome. High doses of these drugs can also cause catatonic reactions due to excessive central dopamine blockade. Although the true incidence of such EPS events is not clear, 1 study noted that EPS symptoms occurred in 20% of agitated patients treated with haloperidol alone but in only 6% of agitated patients treated with a combination of haloperidol and lorazepam.[23] This combination treatment was also found to produce more rapid reduction in agitation. Other studies have found that adding promethazine to haloperidol can similarly reduce the incidence of extrapyramidal side effects.[24,25] In part because of these studies, haloperidol is frequently administered in combination with another medication such as lorazepam, promethazine, or diphenhydramine.[3] However, using multiple medications to control agitation may increase the risk both of oversedation and interactions with other medications. In addition, studies on patient preference have indicated that FGAs sometimes cause dysphoria after use.[26,27] Given that most second-generation antipsychotics have demonstrated good efficacy in treating acute agitation, have low rates of extrapyramidal side effects (see upcoming text), and are subjectively preferred by patients over FGAs,[26,27] the workgroup considers haloperidol to be less preferred than second-generation antipsychotics when an antipsychotic is indicated.

One common clinical scenario where haloperidol may still be the medication of choice is agitation in the context of acute alcohol intoxication. In agitation secondary to alcohol intoxication, medications to manage agitation should be generally avoided if possible, with nonpharmacologic methods, such as reduced environmental stimulation, being the preferred method of treatment.[4,7] If medication is required, previous expert consensus documents have recommended benzodiazepines, given the possibility that a component of withdrawal may be contributing to the agitation.[7] However, alcohol intoxication and withdrawal are distinct nonoverlapping presentations, which clinicians are generally able to differentiate. In addition, although there is no clear scientific evidence of respiratory depression with benzodiazepine use, there is a potential for clinically significant respiratory depression when benzodiazepines are administered to alcohol-intoxicated patients, as both agents are central nervous system (CNS) depressants.[22] As such, this workgroup recommends the use of antipsychotics instead of benzodiazepines to treat agitation in the context of alcohol intoxication but the opposite in alcohol withdrawal (see upcoming text). There are both widespread clinical experience and published literature on the safe and effective use of haloperidol in intoxicated patients. Second-generation antipsychotics, however, have not been well studied in this situation. Thus, haloperidol remains preferred by the workgroup in this clinical scenario, although further study is needed.

THE USE OF SECOND-GENERATION ANTIPSYCHOTICS

Atypical antipsychotics, also called second-generation antipsychotics (SGA), were mostly developed in the 1990s and beyond. Several of these medications are commonly used in the acute setting. Olanzapine (Zyprexa), ziprasidone (Geodon), and aripiprazole (Abilify) come in both intramuscular and oral preparations. Risperidone (Risperdal) and quetiapine (Seroquel) are available in an oral formulation only.

As a class, these medications act as antagonists at the D2 receptor, as do FGAs, but also have comparable or stronger antagonism of other receptor subtypes, particularly serotonin-

2A (5-HT2A) receptors. In addition, this class of medication has actions at other receptor types, such as histamine, norepinephrine, and α-2 receptors. Ziprasidone, for instance, has a high affinity for serotonin receptors compared to D2 receptors,[28,29] while olanzapine and quetiapine have relatively higher affinities for the histamine receptor. In general, when compared with older drugs, SGAs have a reduced risk of near-term side effects such as dystonia or akathisia,[30–32] with reported rates of less than 1%.[30–32] This is lower than that reported with haloperidol alone[23] and is some 10 times lower than even the combination of haloperidol + lorazepam.[23,33]

With the exception of risperidone, most randomized controlled trials of second-generation antipsychotics have been conducted in a psychiatric emergency department or inpatient ward, and not typical acute adult/pediatric emergency departments. Most of this research has generally indicated that most members of the class are effective in reducing agitation when compared to placebo, and are at least as calming as haloperidol.[34–40] This is true of oral and oral rapid-dissolving formulations as well. In the limited number of studies that have compared oral antipsychotics, the combination of oral risperidone + lorazepam is as efficacious as intramuscular haloperidol + lorazepam, and oral risperidone alone is as efficacious as intramuscular haloperidol alone.[37–40] Although there are no comparisons of oral olanzapine or oral ziprasidone with intramuscular haloperidol + lorazepam, oral olanzapine is as efficacious as oral risperidone alone.[39] With the exception of risperidone, however, none of the SGAs have been compared against the more common regimen of haloperidol + lorazepam.[37,38] Further, many of the published SGA investigations were industry-sponsored studies.

Although there have been no head-to-head trials of SGAs in the acute setting, published reviews have attempted to compare the effectiveness of different drugs in the class on a common scale, such as number-needed-to-treat.[36] These reviews have generally indicated that most SGAs are equally effective at reducing agitation, with 3 possible exceptions. First, aripiprazole, the only partial D2 agonist approved for agitation, appears slightly less efficacious than other SGAs.[36] Second, research on quetiapine has indicated that while this medication is useful in inpatient settings, it has an unacceptably high risk of orthostatic hypotension in the emergency department where patients are often volume depleted.[41] Third, clozapine is only FDA approved for treatment-resistant schizophrenia and is not generally a first-line agent. Thus, although more study is needed, the use of aripiprazole, quetiapine, or clozapine cannot be recommended as first-line agents in the acute control of agitation. Other agents, such as lurasidone, iloperidone, and asenapine, are promising but have not yet been tested for acute agitation.

Most published studies of second-generation antipsychotics in agitated patients have not investigated their use either with benzodiazepines or in alcohol-intoxicated patients. Marder et al[42] described a number of adverse events in patients who were administered the combination of olanzapine with benzodiazepines, and this combination is not currently recommended by the manufacturer. In 2 small retrospective studies, Wilson and colleagues[43,44] noted that the combination of olanzapine + benzodiazepines did not cause vital sign abnormalities in patients who had not ingested alcohol. In some patients who had ingested alcohol, however, intramuscular olanzapine + benzodiazepines were associated with decreased oxygen saturations. These studies were too small, however, to provide conclusive evidence for the safety of olanzapine + benzodiazepines in nonintoxicated patients; thus, this combination should be avoided. Similarly, as little research has been conducted on other second-generation antipsychotics in alcohol-intoxicated patients, a first-generation antipsychotic may be a safer choice, especially if clinicians anticipate using a benzodiazepine as well.

A summary of dosing for medications recommended in the treatment of agitation is provided in the Table.

BENZODIAZEPINES

Benzodiazepines such as diazepam, lorazepam, and clonazepam act on the GABA receptor, the main inhibitory neurotransmitter in the human brain. These medications have a long record of efficacy for agitation, and are often preferred by clinicians when the patient is known to be suffering from stimulant intoxication, ethanol withdrawal, or when the etiology of agitation is undetermined. However, in agitation involving psychosis, benzodiazepines alone may only sedate a patient while not addressing the underlying disease that is producing the agitation. In addition, these medications may be oversedating and have the potential for respiratory depression or hypotension when used parenterally in patients with underlying respiratory conditions or in combination with other CNS depressants such as alcohol. In a minority of patients who chronically abuse stimulants, particularly amphetamines, psychotic symptoms develop as a result of their amphetamine use. In these patients, a first- or second-generation antipsychotic is often useful in addition to, or in place of, a benzodiazepine.[45]

SPECIFIC GUIDELINES FOR MEDICATION USE

A recommended protocol for the treatment of agitation is shown in an algorithm in the Figure.

General Recommendations

1. The use of medication as a restraint (ie, to restrict movement) should be discouraged. Rather, clinicians should, to whatever extent possible, attempt a provisional diagnosis of the most likely cause of the agitation and target medication to the most likely disease.
2. Nonpharmacologic approaches, such as verbal de-escalation and reducing environmental stimulation

Table. Medications recommended in the treatment of agitation.

	Initial dose, mg	Tmax*	Can repeat†	Maximum dose (per 24 hours), mg
Oral medication				
Risperidone	2	1 h	2	6
Olanzapine	5–10	6 h	2	20
Haloperidol‡	5	30–60	15 min	20
Lorazepam	2	20–30	2	12
Intramuscular medication				
Ziprasidone	10–20	15	10 mg q 2 h, 20 mg q 4 h	40
Olanzapine	10	15–45	20 min	30
Aripiprazole	9.75	1 h	2	30
Haloperidol‡	5	30–60	15 min	20
Lorazepam	2	20–30	2	12
Intravenous medication				
Haloperidol§	2–5	Immediate	4	10§

q 2 h, every 2 hours; *q 4 h*, every 4 hours.
* Values are expressed as minutes unless otherwise indicated.
† Values are expressed as hours unless otherwise indicated.
‡ Likely to cause higher incidence of extrapyramidal side effects than other recommended drugs.
§ Administering haloperidol intravenously increases risk of QT prolongation. Therefore, avoid if possible, especially in patients with borderline QT or taking other medication that can prolong QT. If given intravenously, limit dose and provide cardiac monitoring.

(quiet room, low lighting), should be attempted, if possible, before medications are administered.
3. Medication should be used to calm patients, not to induce sleep.
4. Patients should be involved in the process of selecting medication to whatever extent possible (eg, oral vs intramuscular).
5. If the patient is able to cooperate with taking oral medications, these are preferred over intramuscular preparations.

Agitation Due to Intoxication

1. Drugs: For intoxication with most recreational drugs, especially stimulants, benzodiazepines are generally considered first-line agents.[47] A minority of chronic amphetamine users develop psychotic symptoms from their amphetamine use.[45] In these patients, a second-generation antipsychotic may be useful in addition to a benzodiazepine.
2. Alcohol: Medication to treat agitation associated with alcohol intoxication should be used sparingly if at all. If medication is required, benzodiazepines should be avoided because of the potential to compound the risk of respiratory depression. Thus, antipsychotics are preferred. Haloperidol has the longest track record of safety and efficacy and has minimal effects on respiration. Second-generation antipsychotics, such as olanzapine and risperidone, have not been well studied for alcohol intoxication but may be a reasonable alternative to haloperidol for agitation in the context of alcohol intoxication. Of note, it is important to distinguish agitation secondary to alcohol intoxication versus agitation secondary to alcohol withdrawal, as benzodiazepines are preferred over antipsychotics in alcohol withdrawal (see the "Agitation Associated with Delirium" section). Agitation in a chronic alcohol user who exhibits features of delirium, such as tachycardia, diaphoresis, tremors, and a low or undetectable alcohol blood level, should be presumed to be due to withdrawal and treated accordingly.

Agitation Due to a Psychiatric Illness

1. For psychosis-driven agitation in a patient with a known psychiatric disorder (eg, schizophrenia, schizoaffective disorder, bipolar disorder), antipsychotics are preferred over benzodiazepines because they address the underlying psychosis.
2. Second-generation antipsychotics with supportive data for their use in acute agitation are preferred over haloperidol either alone or with an adjunctive medication. If the patient is willing to accept oral medication, oral risperidone has the strongest evidence for safety

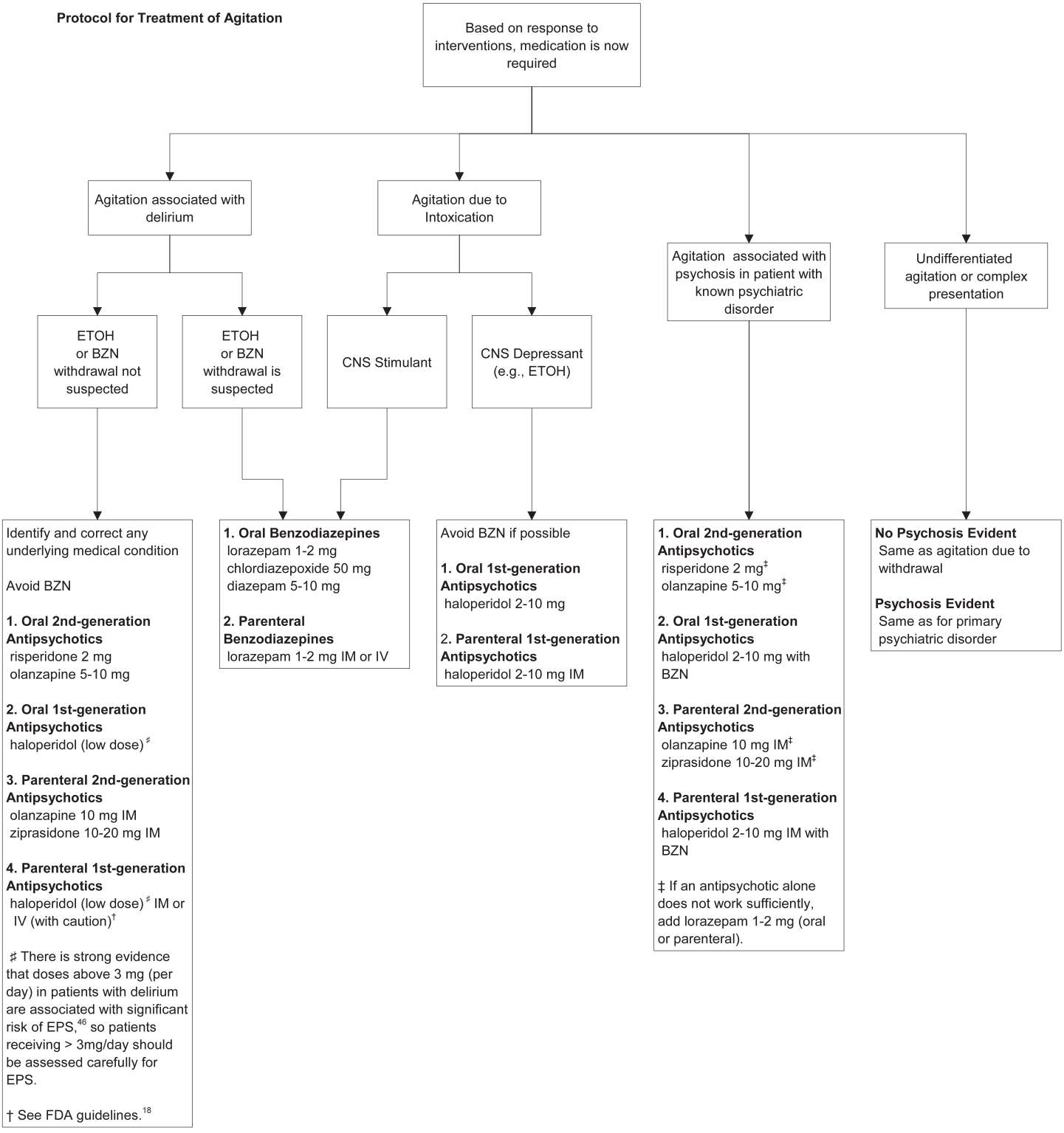

Figure. Protocol for treatment of agitation. *BZN*, benzodiazepine; *EPS*, extrapyramidal side effects; *ETOH*, alcohol; *IM*, intramuscular.

and efficacy, with a smaller number of studies supporting the use of oral antipsychotics such as olanzapine. If the patient cannot cooperate with oral medications, intramuscular ziprasidone or intramuscular olanzapine is preferred for acute control of agitation.

3. If an initial dose of antipsychotic is insufficient to control agitation, the addition of a benzodiazepine such as lorazepam is preferred to additional doses of the same antipsychotic or to a second antipsychotic.

Agitation Associated with Delirium

1. Delirium is a distinct clinical syndrome that frequently is associated with psychosis and agitation. It is important for clinicians to be able to recognize agitation associated with delirium for 2 reasons. First, the presence of delirium signals an underlying medical perturbation affecting brain function or a rapid change in the established environment of the brain. This can occur with sudden withdrawal from a chronically ingested agent (eg, alcohol or medication) or recent ingestion of a drug or medication, such as an anticholinergic agent in an elderly patient. Thus, the presence of delirium should impel the treating physician to identify the cause and correct it. Second, the symptomatic control of agitation secondary to delirium necessitates different choices of calming agents than agitation from other causes.
2. Hallmarks of delirium include a decreased level of awareness and disturbances in attention and cognition (eg, memory) that develop over an acute time course (hours to days). The disturbances in cognition and awareness typically fluctuate over the course of hours (ie, wax and wane). Prominence of visual hallucinations or visual perceptual disturbances is a particularly characteristic feature of delirium.
3. If alcohol or benzodiazepine withdrawal is the suspected cause of delirium, then a benzodiazepine is the agent of choice,[48] since rapid loss of chronic GABA receptor inhibition is implicated in the delirium produced in these circumstances. Clonidine can also be helpful in reducing the sympathetic overdrive of alcohol or benzodiazepine withdrawal, thereby easing delirium and agitation.[49]
4. If withdrawal from another agent is suspected, replacement of the agent with another that has similar pharmacologic properties should be attempted if safe and appropriate (eg, nicotine for nicotine withdrawal).
5. If the recent ingestion of a new agent (or an increased dose of a chronically ingested agent) is the suspected cause of the delirium, then the delirium will be self-limiting. However, agitation may require temporary pharmacologic management (see No. 7).
6. When an underlying medical abnormality (eg, hypoglycemia, electrolyte imbalance, hypoxia) is the likely cause of delirium, the definitive treatment of the delirium and its associated agitation is correction of the underlying medical condition.
7. If immediate pharmacologic control of agitation is needed in a patient with delirium that is not due to alcohol, benzodiazepine withdrawal, or sleep deprivation, second-generation antipsychotics are the preferred agents. Haloperidol is also acceptable in low doses.[46] Benzodiazepines should be generally avoided because they can exacerbate the delirium.[50]

Agitation from Unknown or Complex (More Than 1 Cause) Reasons

If medication is needed to control agitation in a nondelirious patient for whom the underlying etiology of the agitation is not clear, there is little in the way of formal evidence to guide the decision of which agent to use. In patients who do not display psychosis (hallucinations, delusional thinking, paranoia), a benzodiazepine is recommended as first-line treatment. An antipsychotic is recommended in patients who are displaying psychotic features. See the Table for additional dosing information.

CONCLUSIONS

After reviewing available evidence, the workgroup makes the following recommendations. Best practices for treating agitation include the following (please see specific recommendations for detailed recommendations in different clinical scenarios):

1. Pharmacologic treatment of agitation should be based on an assessment of the most likely cause for the agitation. If the agitation is from a medical condition or delirium, clinicians should first attempt to treat this underlying cause instead of simply medicating with antipsychotics or benzodiazepines.
2. Oral medications should be offered over intramuscular injections if the patient is cooperative and no medical contraindications to their use exist.
3. Antipsychotics are indicated as first-line management of acute agitation with psychosis of psychiatric origin.
4. When an antipsychotic is indicated for treatment of agitation, certain SGAs (such as olanzapine, risperidone, or ziprasodone), with good evidence to support their efficacy and lack of adverse events, are preferred over haloperidol or other FGAs. Agitation secondary to intoxication with a CNS depressant, such as alcohol, may be an exception in which haloperidol is preferred owing to few data on second-generation antipsychotics in this specific clinical scenario.
5. If haloperidol is used, clinicians should consider administering it with a benzodiazepine to reduce extrapyramidal side effects unless contraindications to use of this medication exist.

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Address for Correspondence: David Feifel, MD, PhD, UC San Diego Health System, Department of Psychiatry, 200 W Arbor Dr, San Diego, CA 92103. E-mail: dfeifel@ucsd.edu.

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Use and Avoidance of Seclusion and Restraint: Consensus Statement of the American Association for Emergency Psychiatry Project BETA Seclusion and Restraint Workgroup

Daryl K. Knox, MD*
Garland H. Holloman Jr, MD, PhD†

* Mental Health and Mental Retardation Authority of Harris County, Comprehensive Psychiatry Emergency Program, Houston, Texas
† University of Mississippi Medical Center, Department of Psychiatry, Jackson, Mississippi

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Issues surrounding reduction and/or elimination of episodes of seclusion and restraint for patients with behavioral problems in crisis clinics, emergency departments, inpatient psychiatric units, and specialized psychiatric emergency services continue to be an area of concern and debate among mental health clinicians. An important underlying principle of Project BETA (Best practices in Evaluation and Treatment of Agitation) is noncoercive de-escalation as the intervention of choice in the management of acute agitation and threatening behavior. In this article, the authors discuss several aspects of seclusion and restraint, including review of the Centers for Medicare and Medicaid Services guidelines regulating their use in medical behavioral settings, negative consequences of this intervention to patients and staff, and a review of quality improvement and risk management strategies that have been effective in decreasing their use in various treatment settings. An algorithm designed to help the clinician determine when seclusion or restraint is most appropriate is introduced. The authors conclude that the specialized psychiatric emergency services and emergency departments, because of their treatment primarily of acute patients, may not be able to entirely eliminate the use of seclusion and restraint events, but these programs can adopt strategies to reduce the utilization rate of these interventions. [West J Emerg Med. 2012;13(1):35–40.]

INTRODUCTION

A major focus of Project BETA (Best practices in Evaluation and Treatment of Agitation)[1] is noncoercive de-escalation, with the goal being to calm the agitated patient and gain his or her cooperation in the evaluation and treatment of the agitation. Some healthcare providers may view forced medication, seclusion, and restraint as the safest and most efficient intervention for the agitated patient but are relatively unaware that these interventions are associated with an increased incidence of injury to both patients and staff. These injuries are both physical and psychological. In addition, the use of drugs for the purpose of restraint results in side effects that can be problematic. Both physical interventions and drugs for the purpose of restraint have short-term and long-term detrimental implications for the patient and the physician-patient relationship. Because of this, regulatory agencies and advocacy groups are pushing for a reduction in the use of restraint. However, there are clinical situations for which verbal and behavioral techniques are not effective and the use of seclusion and/or restraint becomes necessary to prevent harm to the patient and/or staff. When use of restraint and seclusion is unavoidable, there are measures that can be taken to mitigate some of the negative consequences that may result when such actions are taken.

The Centers for Medicare and Medicaid Services (CMS) has adopted Conditions of Participation for Hospitals. These same conditions have been endorsed by The Joint Commission (TJC). In doing so, the following definitions are used:

- Seclusion is the involuntary confinement of a patient alone in a room or area from which the patient is

Table. Patient-reported psychological distress due to common interventions.[6]

Intervention	Patients (%)	
	Experiencing intervention	Experience severely distressing
"Taken down"	29	46
Placed in seclusion	59	48
Put in restraints	34	52
Forced to take medication	27	58
Any other physical force	21	66

physically prevented from leaving. Seclusion may be used only for the management of violent or self-destructive behavior.[2]

- A restraint is any manual method, physical or mechanical device, material, or equipment that immobilizes or reduces the ability of a patient to move his or her arms, legs, body, or head freely.[2]
- A drug is considered a restraint when it is used as a restriction to manage the patient's behavior or restrict the patient's freedom of movement and is not a standard treatment or dosage for the patient's condition.[2]
- Seclusion and restraint must be discontinued at the earliest possible time.[2]
- Within 1 hour of the seclusion or restraint, a patient must be evaluated face-to-face by a physician or other licensed independent practitioner or by a registered nurse or physician assistant who has met specified training requirements.[2]

Specified also are the following patient's rights:

- Seclusion or restraint may be used only when less restrictive interventions have been determined to be ineffective to protect the patient, a staff member, or others from harm.[2]
- All patients have the right to be free from restraint or seclusion, of any form, imposed as a means of coercion, discipline, convenience, or retaliation by staff.[2]
- Restraint or seclusion may only be imposed to ensure the immediate physical safety of the patient, a staff member, or others.[2]

In addition to the requirement to conform to these regulations, there are medicolegal reasons to avoid seclusion and restraint. A National Association of State Mental Health Program Directors document on risk management concludes as follows:

> *"Every episode of restraint or seclusion is harmful to the individual and humiliating to staff members who understand their job responsibilities. The nature of these practices is such that every use of these interventions leaves facilities and staff with significant legal and financial exposure.*
> *Public scrutiny of restraint and seclusion is increasing and legal standards are changing, consistent with growing evidence that the use of these interventions is inherently dangerous, arbitrary, and generally avoidable. Effective risk management requires a proactive strategy focused on reducing the use of these interventions in order to avoid tragedy, media controversy, external mandates, and legal judgments*."[3]

The purpose of this article is twofold. First, we will review information that supports the need to avoid physical restraint if at all possible. Second, we will provide guidelines for the use of seclusion and restraint when other methods fail. We will also offer recommendations to lessen the psychological impact on patients and staff that often ensues in the aftermath of a seclusion and restraint episode.

USE OF PHYSICAL RESTRAINT

There is much controversy regarding the use of restraints and seclusion. In 1994, Fisher[4] reviewed the literature and concluded that restraint and seclusion were useful for preventing injury and reducing agitation and that it was impossible to run a program that dealt with seriously ill individuals without the use of these restrictive interventions. However, he did acknowledge that use of these interventions caused adverse physical and psychological effects on both staff and patients and pointed out that nonclinical factors, such as cultural biases, role perceptions, and attitude, are substantial contributors to the frequency of seclusion and restraint.

A review by Mohr et al[5] concluded that the use of restraints puts patients at risk for physical injury and death and can be traumatic even without physical injury. Acknowledging the lack of empirical studies, they also concluded that physical injuries to patients were caused by a variety of complications from the use of physical restraint.

The Table shows several items from the data of a survey of 142 patients, using a questionnaire designed to identify the frequency of potentially harmful events and the associated psychological distress experienced by the patient. This clearly shows that commonly used interventions are traumatic to patients.[6]

If patients experience physical and psychological effects from restraints, what effects do healthcare providers experience when working with agitated patients? Healthcare workers are at a considerably higher risk for workplace violence than other professions. Nurses are at greater risk than physicians (2.19% vs 1.62%), but the risk is even greater for mental health professionals (6.82%).[7] In a survey of 242 emergency department workers at 5 hospitals, approximately 48% had been physically assaulted.[8] In a randomized sample of 314 nurses, 62.1% had been exposed to aggression by patients. Of these, 40% experienced psychological distress and 10%

experienced moderate to severe depression.[9] None of these studies looked at the injury occurring during attempted restraint. However, in a study of the prehospital, emergency medical services (EMS) setting, 4.5% of cases involved violence toward EMS personnel.[10] When physical restraint was used in the prehospital setting, 28% involved assault on EMS personnel.[11]

Even if restraint and seclusion can prevent injury to patients and staff, a physical altercation with a patient can result in a variety of injuries to both, and these injuries could be avoided if effective ways were available to manage the patient without their use. This can happen, but it will require a change in attitude on the part of clinicians who work with agitated patients, as well as change in the staff development training and culture of the institutions in which they practice. In a summary report, the Substance Abuse and Mental Health Services Administration states, "The culture must change from one in which seclusion and restraint are viewed as positive and therapeutic to one in which they are regarded as violent acts that result in traumatization to patients, observers, and others."[12] The following studies show that this is possible.

A public psychiatric inpatient service was able to reduce restraint without an increase in patient-to-patient assaults. There was an initial increase in patient-to-staff assaults but when the initial period was excluded, there was no statistical change.[13]

In a retrospective analysis of a large inner city hospital's efforts to implement the mandates of CMS and TJC, Khadivi et al[14] found a significant decrease in the use of restraints but an increase in assaults on patients and staff. However, they noted that "staff did not receive any specific training in the management of violent patients, which may have increased the rate of assaults on staff members and diminished their ability to reduce other-directed assaults."

Another large study took place in 9 Pennsylvania state hospitals during an 11-year period. According to the authors, "the rate of seclusion decreased from 4.2 to 0.3 episodes per 1,000 patient-days. The average duration of seclusion decreased from 10.8 to 1.3 hours. The rate of restraint decreased from 3.5 to 1.2 episodes per 1,000 patient-days. The average duration of restraint decreased from 11.9 to 1.9 hours." At the time of the study, 1 hospital had gone 2 years without using restraint; and, since 2005, the system as a whole, which provides more than 60,000 days of care per month, had used seclusion 19 times and restraints 143 times for a total of 160 hours. Data on staff injury indicated that staff members were not at increased risk of assault. The authors attributed part of the success to administration recognizing that "seclusion and restraint are not treatment modalities but treatment failures." Other major reasons were changes in attitude, culture, and environment within the hospitals.[15]

Donat[16] reviewed several initiatives aimed at reducing seclusion and restraint taken during a 5-year period at a public psychiatric hospital. These initiatives included "changes in the criteria for administrative review of incidents of seclusion and restraint, changes in the composition of the case review committee, development of a behavioral consultation team, enhancement of standards for behavioral assessments and plans, and improvements in the staff–patient ratio." He applied a multiple regression analysis to the results and discovered that the most significant variable leading to the 75% reduction in seclusion and restraint incidents was "changes in the process for identifying critical cases and initiating a clinical and administrative case review."

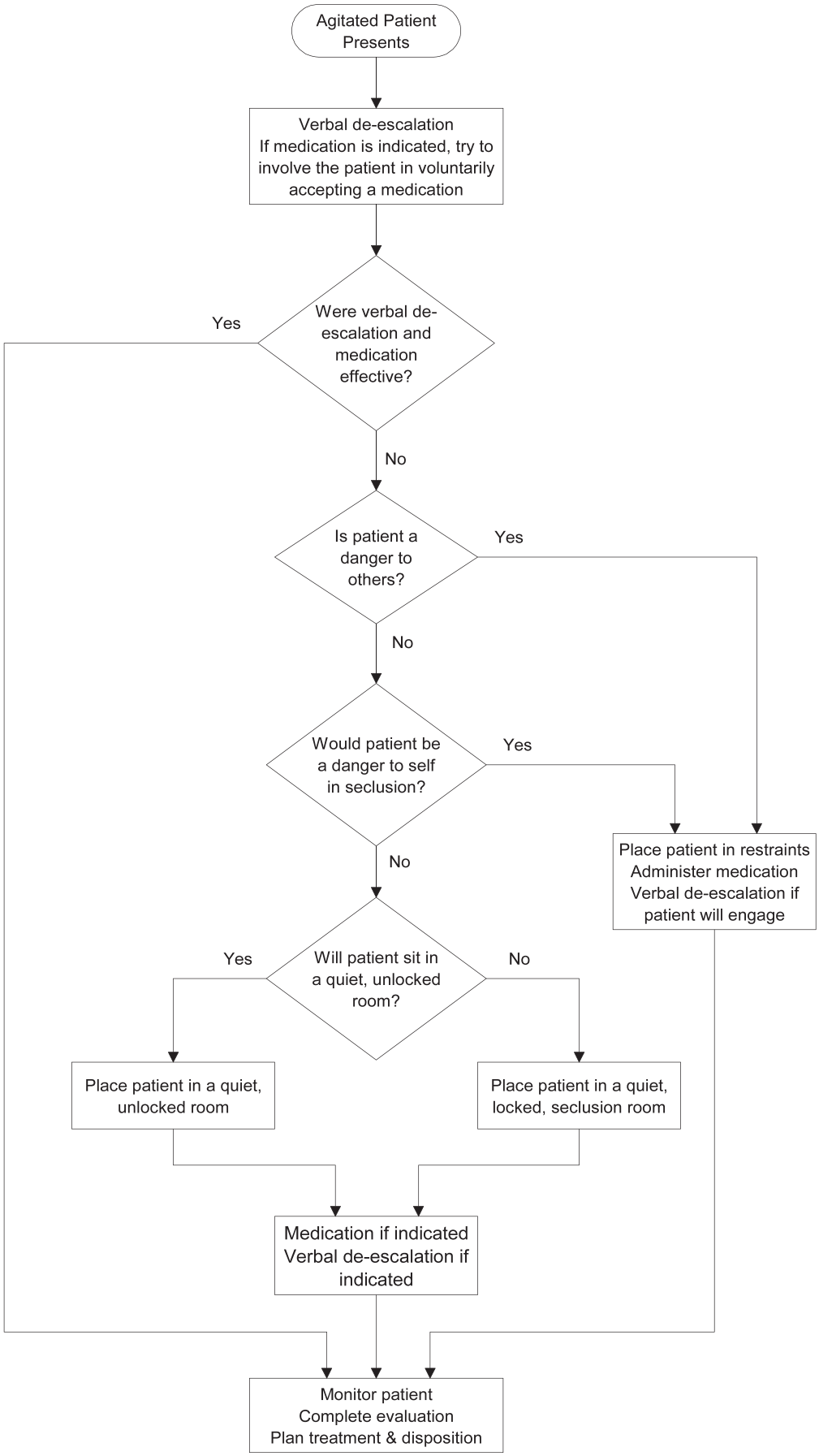
The above strategies for decreasing seclusion and restraint worked well in inpatient hospital environments, and there are several other reports on successful reduction of seclusion or restraint.[17–20] However, it may be unrealistic to expect these results in a psychiatric emergency service (PES) or emergency department (ED) setting, as they differ in clinical structure, purpose, and length of stay from an inpatient hospital unit.

Zun,[21] in a prospective study of complications of restraint use in emergency departments, found that use of restraints "is significantly higher than in an inpatient facility." Hospital inpatient units are seldom as hectic as an ED or PES. In inpatient facilities, patients typically have a chance to develop rapport with staff over a period of days, and most units provide ample space and a place such as a bedroom for patients to retreat when unit activity becomes stressful. The volume of admissions and discharges from an inpatient unit occurs more sporadically than in an ED or PES, where there are constant admissions and discharges within a day, and the acuity level can be constantly high and intense. Arguably, these differences between the emergency setting and an inpatient unit make it less likely that episodes of seclusion and restraint can be eliminated totally in this setting. However, review of seclusion and restraint cases, including feedback to staff, and institutional changes in culture and attitude, can be important factors in reducing occurrence of these incidents in more acute settings.

In the introduction to a special session on seclusion and restraint, Busch[22] states that programs for reduction of restraint have been successful without increasing the risk to staff. She asks, "Can we do a better job of preventing or de-escalating these situations so that we do not need to use seclusion, restraint, or emergency medication?" She points out that literature tells us that we can.

Even with these and other success stories, the use of seclusion and restraint is still a common practice. Seclusion is used as an intervention in 25.6% of emergency departments.[23] In another survey of emergency departments, 30% of respondents used physical restraint alone and another 30% used physical restraint combined with pharmacotherapy.[24]

Ashcraft and Anthony[25] state that successful seclusion and restraint reduction programs are based on strong leadership direction, policy and procedural change, staff training, consumer debriefing, and regular feedback. Forster and colleagues[26] focused their training on increasing awareness of


Agitated Patient Presents
Verbal de-escalation
If medication is indicated, try to involve the patient in voluntarily accepting a medication
Were verbal de-escalation and medication effective?
Yes
No
Is patient a danger to others?
Yes
No
Would patient be a danger to self in seclusion?
Yes
No
Place patient in restraints
Administer medication
Verbal de-escalation if patient will engage
Will patient sit in a quiet, unlocked room?
Yes
No
Place patient in a quiet, unlocked room
Place patient in a quiet, locked, seclusion room
Medication if indicated
Verbal de-escalation if indicated
Monitor patient
Complete evaluation
Plan treatment & disposition

factors that lead to agitation and violence, teaching less restrictive interventions, and the teaching of safe reactions to patient violence. Borckardt and colleagues[27] implemented an engagement model that includes trauma-informed care training, changes in rules and language, patient involvement in treatment planning, and changes to the physical characteristics of the therapeutic environment. Project BETA believes that the culture that promotes the use of restraint and seclusion can be changed. This will require implementing programs with the above features, plus specific training in verbal, de-escalation techniques.

GUIDELINES FOR THE USE OF SECLUSION AND RESTRAINT

When seclusion or restraint is necessary, the least restrictive intervention should be chosen. The Figure shows a recommended algorithm. Unless the patient is actively violent, verbal de-escalation should be tried first. The clinician should offer medication and try to involve the patient in decisions about medication. If the patient is an immediate danger to others, restraint is indicated. If the patient is not a danger to others, seclusion should be considered. However, if the patient would be a danger to himself while in seclusion, restraint is appropriate. If the restrained patient will engage in a reasonable dialog, verbal de-escalation efforts should continue, including getting the patient's input on medication. Either way, medication should be administered to calm a patient who has been placed in restraints. If restraint is not indicated and the patient is willing to sit in a quiet, unlocked room, then an unlocked seclusion room should be used. If not, then forced seclusion is indicated. For some patients, seclusion with decreased stimulation is adequate for them to regain control. For others, medication should be considered, and ongoing efforts at verbal de-escalation may be beneficial. All patients in restraint or seclusion should be monitored to assess response to medication and to prevent complications from these interventions. Treatment should be directed toward minimizing time in forced seclusion or restraint. Once the patient has regained control, a more thorough evaluation can be done, followed by further treatment planning and determining disposition.

In summary, approaches for reducing seclusion and restraint episodes that may be applied to ED/PES settings include change in organization culture where restraint is viewed as a treatment failure, implementing an administrative quality management review process aimed at improving outcomes in manging aggrerssive behavior, regular staff feedback, early identification and intervention using de-escalation techniques, and the use of protocols or aggressive mangement algorithms to guide clinical interventions.

In addition, it is important, as well as legally mandated, that CMS guidelines be followed and incorporated into the program's policies and procedures. All clinical staff in an ED or PES must have training on an annual basis at a minimum on verbal de-esclation techniques and the prevention and management of aggressive behavior. All staff members, including physicians, should be familiar with the types of restraints used in their programs and how to appropriately apply, monitor, and assess potential bodily injury that might result from application of the restraints. Use of video cameras in the clinical areas that are used by clinical staff to monitor the clinical environment can also be used in an instructive manner to review the restraint or seclusion episode to see if other, less forecful, interventions could have been tried. Where possible, time set aside to debrief staff and patients on the seclusion and restraint episode can provide valuable learning opportunites as well as a way to verbalize and process feelings surrounding the event.

CONCLUSIONS

While it may not be possible to eliminate incidents of seclusion and restraint in the PES or ED setting, more can be done to reduce the current rate of these incidents. It is important to keep in mind that often a patient's first entry into the mental health system can be through the doors of an emergency department. Patients may be at their lowest point of functioning, whereby their perceptions are altered, their sense of reality is grossly impaired, and they are being forced into treatment. It is in this atmosphere that emergency clinicians must make the most of a very unpleasant experience for the patient by endeavoring to make the experience as therapeutic as possible, with the goal of getting that patient into ongoing psychiatric treatment to minimize the likelihood of another decompensation and emergency setting encounter. "The new psychiatric emergency department is a place to start treatment and not one whose primary purpose is restraint, triage or referral."[28]

Address for Correspondence: Daryl K. Knox, MD, Mental Health and Mental Retardation Authority of Harris County, Comprehensive Psychiatry Emergency Program, 1502 Taub Loop, Houston, TX 77030. E-mail: daryl.knox@mhmraharris.org.

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←
Figure. Recommended seclusion and restraint algorithm.

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Evaluation of an Emergency Department Educational Campaign for Recognition of Suicidal Patients

Glenn W. Currier, MD, MPH*†
David Litts, OD‡
Patrick Walsh, MPH*
Sandra Schneider, MD†
Thomas Richardson, PhD†
William Grant, EdD§
Wayne Triner, DO||
Nancy Robak, RN, MPH||
Ronald Moscati, MD¶

* University of Rochester Medical Center, Department of Psychiatry, Rochester, New York
† University of Rochester Medical Center, Department of Emergency Medicine, Rochester, New York
‡ Suicide Prevention Resource Center, Washington, DC
§ Upstate Medical University, Department of Emergency Medicine, Syracuse, New York
|| Albany Medical College, Department of Emergency Medicine, Albany, New York
¶ State University at Buffalo, Department of Emergency Medicine, Buffalo, New York

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Introduction: To evaluate the impact of a simple emergency department (ED)–based educational intervention designed to assist ED providers in detecting occult suicidal behavior in patients who present with complaints that are not related to behavioral health.

Methods: Staff from 5 ED sites participated in the study. Four ED staff members were exposed to a poster and clinical guide for the recognition and management of suicidal patients. Staff members in 1 ED were not exposed to training material and served as a comparator group.

Results: At baseline, only 36% of providers reported that they had sufficient training in how to assess level of suicide risk in patients. Greater than two thirds of providers agreed that additional training would be helpful in assessing the level of patient suicide risk. More than half of respondents who were exposed to the intervention (51.6%) endorsed increased knowledge of suicide risk during the study period, while 41% indicated that the intervention resulted in improved skills in managing suicidal patients.

Conclusion: This brief, free intervention appeared to have a beneficial impact on providers' perceptions of how well suicidality was recognized and managed in the ED. [West J Emerg Med. 2012;13(1):41–50.]

INTRODUCTION

Suicidal patients represent an increasing proportion of emergency department (ED) volumes.[1] In 2007, 472,000 people were treated in US EDs for self-inflicted injury.[2] While the National Strategy for Suicide Prevention recognizes the ED as a practical setting for suicide prevention,[3] for a variety of reasons, ED clinicians may not screen for or recognize suicidal patients.[4] Suicide ideation is often not disclosed by ED patients and is often undetected during visits.[5] Mental health patients that commit suicide often have attended an ED 1 or several times in the year prior to death.[6] ED providers can play a pivotal role in suicide prevention, particularly in the identification of suicidal risk and behavior and linkage with treatment.[7] Brief training has been shown to improve ED provider knowledge regarding suicidal behavior.[8,9] This highlights the need for suicide prevention training and protocol enhancement for ED providers. However, ED-based efforts must be focused, clinically relevant, and delivered in a means that is acceptable to busy providers.

The uptake of new information by healthcare providers is

critical to the advancement of clinical care. The translation of knowledge into effective patient care and policy, however, involves barriers at both practitioner and institutional levels, including the time constraints in acute care settings and the volume of information provided to practitioners.[10] In emergency medicine, translating research to practice has been inconsistent.[11] Implementing and uptake of practice guidelines can be complicated by the values and characteristics of the practitioners and patients, the clinical setting, and complexities of the specific practice guidelines.[11–13]

The objective of this multisite study is to evaluate the impact of a simple ED-based educational intervention designed to assist ED providers (attending and resident physicians, midlevel providers, and nurses) in detecting and addressing occult suicidal behavior in patients who present with complaints that are not related to behavioral health. We hypothesize that exposure to relevant educational material would result in increased provider awareness of potential ED patient suicidality and increased provider perception of their knowledge and skills to identify and treat suicidal ED patients.

METHODS

The educational intervention includes the use of a poster and clinical guide sponsored collaboratively by the Suicide Prevention Resource Center and the American Association of Suicidology and developed by a task force of behavioral health and ED clinician-researchers. The development process included multiple rounds of reviews and focus group testing by practicing ED physicians and nurses. The final product packet was composed of a poster, clinical triage guide, and implementation instructions distributed through the Emergency Nurses Association. Additionally, the materials have been distributed through state hospital associations as well as suicide prevention organizations. The study was supported by the Suicide Prevention Resource Center and was a cooperative effort of the Emergency Research Network in the Empire State (ERNES), a group of academic and community EDs throughout Western and Upstate New York and Northern Pennsylvania. During a 6-month period beginning in August 2009, providers in 4 ERNES EDs completed surveys detailing recognition and care of suicidal patients before and after exposure to training materials. Providers in 1 ED served as a comparator group, and completed the presurveys and postsurveys but did not receive the educational materials. Attitudes toward suicide and suicide prevention, related practice patterns, and perceived skills in suicide assessment were evaluated before and after dissemination of the training materials.

The study consisted of 3 phases including completion and collection of baseline surveys (phase 1, lasting 3 weeks), exposure to educational materials (phase 2, lasting 4 weeks), and completion and collection of follow-up surveys (phase 3, lasting 3 weeks). Surveys were made available to ED providers at each site in both paper form and online via Survey Monkey (SurveyMonkey.com, LLC, Palo Alto, California), an online survey tool, to facilitate as many responses as possible.

Before phase 1 and phase 3, study coordinators at each site provided instructions, distributed survey hard copies and invitation letters, and notified providers of the intervention. All providers were free to decline participation in the study. To obtain similar sample sizes across sites with varying numbers of providers, participation targets included a minimum of 80 providers at each site, including approximately one-third physicians, one-third midlevel providers, and one-third nurses. Surveys were anonymous; however, participants provided their own unique identification code to link baseline and follow-up surveys.

The director of each ED, or a designated study coordinator, distributed educational materials and managed each site's adherence to the study protocol, including the dissemination of study materials. After preliminary analysis, a postintervention survey was designed to assess if there were any additional trainings or enhancements to suicide prevention policy or processes at any ED sites during the study period. In this survey, ED directors or study coordinators were asked if they had done "anything during the intervention to highlight" or "improve" protocols for suicidal patients. The study was approved by the institutional review board of each site: Albany Medical Center (AMC), Erie County Medical Center in Buffalo, New York (ECMC), Robert Packer Hospital of Sayre, Pennsylvania (Guthrie Healthcare), SUNY Upstate University Hospital at Syracuse, New York (Syracuse), and the University of Rochester Medical Center (URMC).

Description of the Intervention

The intervention consisted of (1) a brightly colored, 11 × 17-inch poster mounted in the chart room or break room of each ED, and (2) distribution of an accompanying clinical guide to all ED providers. The "Is Your Patient Suicidal?" poster (Figure 1) provides suicide prevention information including signs of acute suicide risk, statistics, questions for use in detecting and discussing suicide ideation and prior attempts, and the National Suicide Prevention Lifeline number. The clinical guide, "Suicide Risk: A Guide for ED Evaluation and Triage," (Figures 2 and 3) is a 1-page, double-sided companion resource to the poster that describes the poster content; additional questions for assessing suicidal ideation, plans, and intent; information on triage (high-risk patients, moderate-risk patients, low-risk patients, and recommended interventions); and discharge and documentation checklists. The posters were mounted for at least 4 weeks, the duration of phase 2.

Inclusion Criteria

All physicians, physician assistants, nurse practitioners, and registered nurses at the ERNES ED sites were invited to participate in the baseline and follow-up surveys. Both male and female subjects were included in this study and all subjects

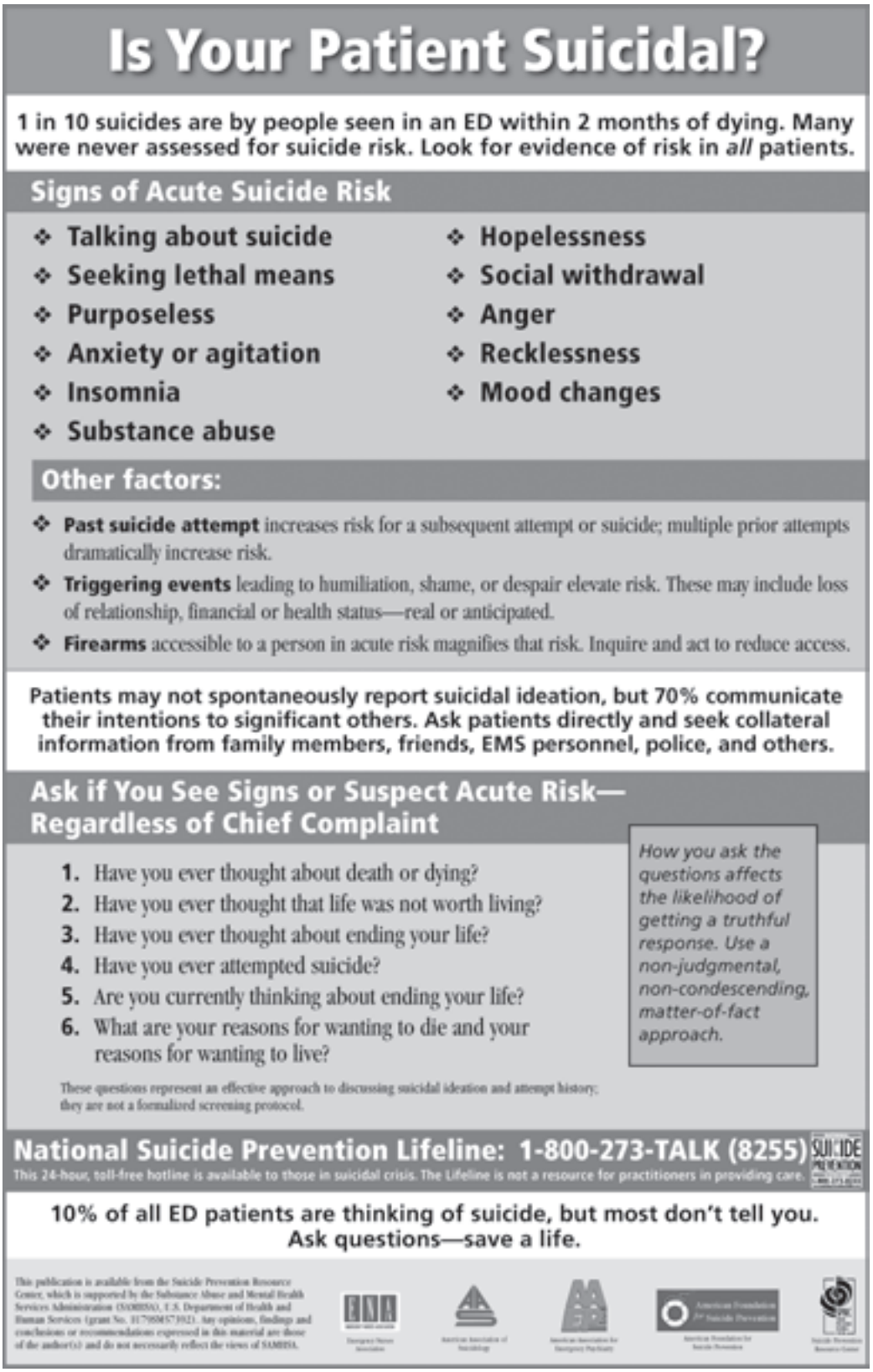
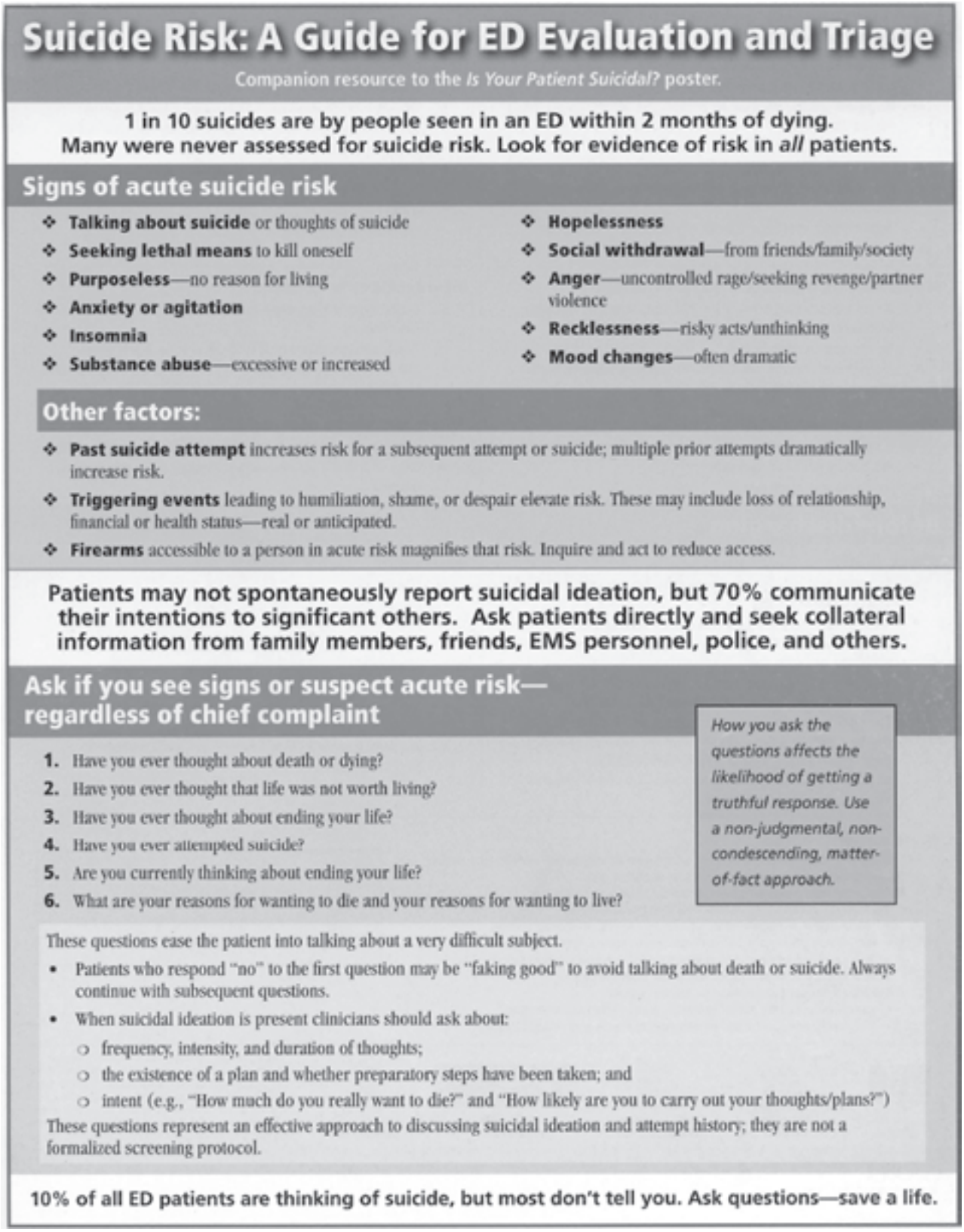


Figure 1. The "Is Your Patient Suicidal?" poster. *ED*, emergency department; *EMS*, emergency medical services.



Suicide Risk: A Guide for ED Evaluation and Triage

Companion resource to the *Is Your Patient Suicidal?* poster.

1 in 10 suicides are by people seen in an ED within 2 months of dying. Many were never assessed for suicide risk. Look for evidence of risk in *all* patients.

Signs of acute suicide risk

- **Talking about suicide** or thoughts of suicide
- **Seeking lethal means** to kill oneself
- **Purposeless**—no reason for living
- **Anxiety or agitation**
- **Insomnia**
- **Substance abuse**—excessive or increased
- **Hopelessness**
- **Social withdrawal**—from friends/family/society
- **Anger**—uncontrolled rage/seeking revenge/partner violence
- **Recklessness**—risky acts/unthinking
- **Mood changes**—often dramatic

Other factors:

- **Past suicide attempt** increases risk for a subsequent attempt or suicide; multiple prior attempts dramatically increase risk.
- **Triggering events** leading to humiliation, shame, or despair elevate risk. These may include loss of relationship, financial or health status—real or anticipated.
- **Firearms** accessible to a person in acute risk magnifies that risk. Inquire and act to reduce access.

Patients may not spontaneously report suicidal ideation, but 70% communicate their intentions to significant others. Ask patients directly and seek collateral information from family members, friends, EMS personnel, police, and others.

Ask if you see signs or suspect acute risk—regardless of chief complaint

1. Have you ever thought about death or dying?
2. Have you ever thought that life was not worth living?
3. Have you ever thought about ending your life?
4. Have you ever attempted suicide?
5. Are you currently thinking about ending your life?
6. What are your reasons for wanting to die and your reasons for wanting to live?

How you ask the questions affects the likelihood of getting a truthful response. Use a non-judgmental, non-condescending, matter-of-fact approach.

These questions ease the patient into talking about a very difficult subject.

- Patients who respond "no" to the first question may be "faking good" to avoid talking about death or suicide. Always continue with subsequent questions.
- When suicidal ideation is present clinicians should ask about:
  - frequency, intensity, and duration of thoughts;
  - the existence of a plan and whether preparatory steps have been taken; and
  - intent (e.g., "How much do you really want to die?" and "How likely are you to carry out your thoughts/plans?")

These questions represent an effective approach to discussing suicidal ideation and attempt history; they are not a formalized screening protocol.

10% of all ED patients are thinking of suicide, but most don't tell you. Ask questions—save a life.

Figure 2. Front view of clinical guide for "Suicide Risk: A Guide for ED Evaluation and Triage". *ED*, emergency department; *EMS*, emergency medical services.

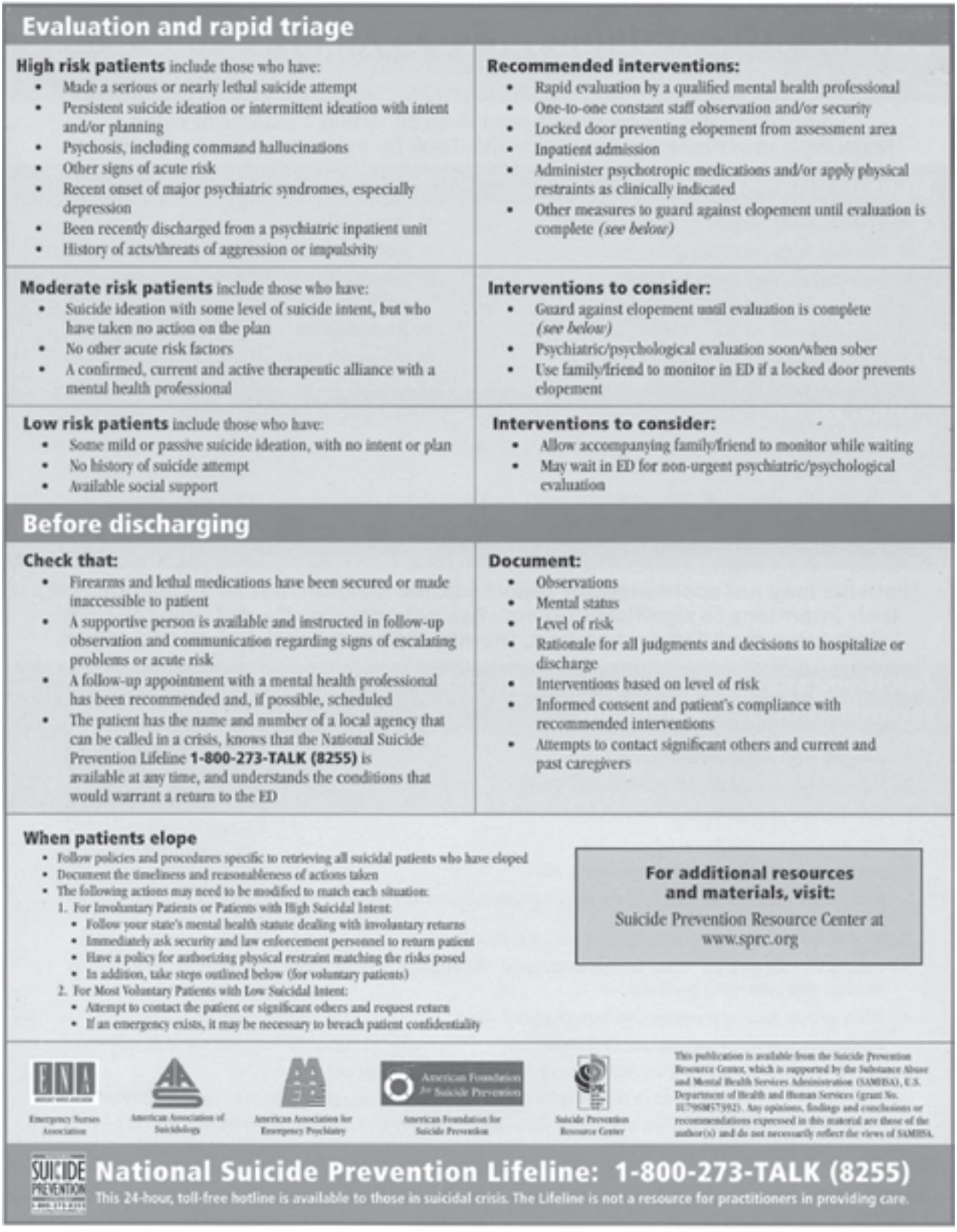


Figure 3. Back view of clinical guide for ''Suicide Risk: A Guide for ED Evaluation and Triage''. *ED*, emergency department.

were older than 18 years. Subjects' racial and ethnic origins reflected that of ED providers.

Exclusion Criteria

There were no exclusion criteria for this study.

Data Analysis

The results of each survey question were tabulated and reported in absolute numbers and proportions. Attending physicians were categorized as "physicians"; residents and fellows, physician assistants, and nurse practitioners were categorized as "supervised providers"; and nurses were categorized as "nurses." Chi-square tests were used to compare differences in proportions for dichotomous categorical variables. To conserve sample size, in some instances 5-point Likert scales (1, strongly disagree–5, strongly agree) were dichotomized to agree (4, 5)/not agree (1, 2, 3) or disagree (1, 2)/not disagree (3, 4, 5). We further performed analyses of respondents who reported recalling exposure to the training materials (exposed) versus those who did not (unexposed). Postintervention responses were compared between participants at intervention sites and participants at the comparator site. All tests were 2-tailed and used a 0.05 significance level. Statistical analyses were conducted with SPSS 16.0 (SPSS Inc, Chicago, Illinois).

RESULTS

Five EDs participated in the study: AMC, ECMC, Guthrie Healthcare, Syracuse, and URMC.

A total of 362 subjects completed the baseline survey and 250 subjects (69.1%) completed the follow-up survey. The 5 participating ED sites had approximately 650 physician, midlevel, and nurse providers in total. The overall baseline response rate for the study was thus approximated to be 55.7% (362/650). Response rates per provider type were approximately 58.4% (73/125) for physicians, 61.4% (118/192) for mid-level providers, and 51.4% (171/333) for nurses. More than one half of baseline surveys (51.1%) and follow-up surveys (54.0%) were completed online. Combined totals for the baseline and follow-up surveys per site ranged from 203 at AMC to 49 at Syracuse, with URMC completing 166, ECMC completing 136, and Guthrie completing 58 surveys each.

Fewer than 1% of subjects reported specialty training in psychiatry. About 60% reported providing direct care primarily to adults, 8% primarily to children, and 32% to both children and adults.

Other selected baseline provider characteristics and baseline provider experience variables are displayed in Table 1. Approximately 80% of providers reported that in their careers they had provided care to at least 10 "patients presenting for an acute suicide attempt." At baseline, 36.4% of respondents endorsed having detected acute suicidal thoughts in several patients who presented to the ED for medical complaints. About one half of providers had 5 or more years of ED experience.

Table 2 shows totals of baseline data on provider training, attitudes, and beliefs about care of suicidal patients. Only 36% of providers reported that they had "sufficient training in how to assess level of suicide risk in patients." Greater than two thirds of providers agreed that additional training "would be helpful" in assessing the level of patient suicide risk.

Of the 218 subjects at intervention sites that completed follow-up surveys, 93 recalled exposure to either the poster or the clinical guide (42.7%). Table 3 shows significant results for comparisons between *exposed* and *unexposed* (n = 157) follow-up subjects. Exposed subjects more readily endorsed that if they suspect emotional distress in their patient, they "always ask them about risk factors for suicide" (58.1% vs 41.3%; $\chi^2 = 6.3$, $P = 0.012$) and that they "always ask them directly if they are having suicidal thoughts" (73.1% vs 59.4%; $\chi^2 = 4.6$, $P = 0.032$). Approximately 10% of providers in both groups reported they had given a patient a suicide prevention hotline number. Significantly more exposed providers reported using an assessment guide to help determine level of suicide risk than unexposed providers (27.2% vs 9.2%; $\chi^2 = 13.3$, $P < 0.001$). Also, significantly more exposed providers reported using a guide to help manage suicidal patients than unexposed providers (28.3% vs 14.8%; $\chi^2 = 6.3$, $P = 0.012$).

The comparator group (ED not provided education materials at phase 2) included 22 follow-up subjects. As shown in Table 4, slightly more than half of intervention site subjects reported they "suspected underlying or concealed suicidal ideation in a patient who presented without a mental health–related chief complaint" in the past month, compared to fewer than one fifth of clinicians in the comparator site (51.8% vs 18.2%; $\chi^2 = 9.1$, $P = 0.003$). Interestingly, a higher proportion of intervention site subjects relative to comparator subjects agreed with the statement, "The ED where I work has a very good protocol for managing suicidal patients when they are identified" (74.1% vs 52.6%; $\chi^2 = 4.0$, $P = 0.044$).

Table 5 shows the impact of the intervention on knowledge and skills for managing suicide for subjects who recalled exposure to the intervention. More than half of exposed follow-up subjects (51.6%) reported that, as a result of the intervention, they had "an increased knowledge of signs of acute suicide risk"; 45.9% reported that their "skills for asking about underlying or concealed suicidal ideation have improved"; and 41.0% reported that their "skills in managing suicidal patients have improved." In response to the postintervention survey, no directors or study coordinators reported any changes or emphasis on protocols during the intervention.

DISCUSSION

Our findings suggest that significant improvements in self-reported practice patterns can be achieved through the simple intervention of hanging a wall poster and distributing a 1-page clinical guide to ED clinicians. For instance, providers that

Table 1. Baseline provider characteristics and experience.*

Item	Response	Total No. (%)
Position category	Nurse	171 (47.2)
	Midlevel	118 (32.6)
	Physician	73 (20.2)
Gender	Male	152 (42.3)
Years worked in emergency medicine	<1	55 (15.2)
	1–4	118 (32.6)
	5–9	62 (17.1)
	10+	126 (34.8)
In my career . . .		
. . . I have identified an acute suicide ATTEMPT in patients presenting without a mental health chief complaint.	Never	89 (24.7)
	Once	27 (7.5)
	A few times	137 (38.0)
	Several times	108 (29.8)
. . . I estimate that I have provided care to _____ patients presenting for an acute suicide ATTEMPT.	0	3 (0.8)
	<10	72 (19.9)
	10–50	140 (38.7)
	>50	147 (40.6)
. . . I estimate that I have provided care to _____ patients with a presenting complaint of suicidal IDEATION.	0	1 (0.3)
	<10	31 (8.6)
	10–50	95 (26.4)
	>50	233 (64.7)
. . . I have identified underlying or concealed suicidal IDEATION in patients presenting without a mental health chief complaint.	Never	55 (15.3)
	Once	21 (5.8)
	A few times	153 (42.5)
	Several times	131 (36.4)
In the past month . . .		
. . . I have SUSPECTED underlying or concealed suicidal ideation in a patient who presented without a mental health–related chief complaint.	Yes	192 (54.2)
. . . I have INQUIRED about suicidal ideation in a patient presenting without a mental health–related chief complaint.	Yes	260 (72.6)
. . . I have given a patient the phone number for a suicide prevention hotline.	Yes	53 (14.8)
. . . I used an assessment guide to help determine LEVEL OF SUICIDE RISK.	Yes	64 (17.8)
. . . I used a guide to help in the MANAGEMENT of suicidal patients.	Yes	74 (20.8)

* Totals do not always equal 362 due to missing data.

were exposed to the educational materials in this intervention were more likely to report that they inquired about suicide risk and suicidal thoughts. Subjects at intervention sites compared to comparator sites more frequently reported suspecting concealed suicide ideation in their patients. Clinicians exposed to the educational material were also more likely to directly inquire about suicide thoughts in patients they suspected were in emotional distress and were more likely to use a guide in making risk assessments and managing suicidal patients. These differences were evident despite low reporting of exposure to the educational materials at intervention sites, suggesting that introducing an educational intervention on suicide in an ED can influence provider attitudes and behaviors for those not reporting direct exposure to the material. This could suggest informal augmentation of suicide prevention awareness and attention to identification among ED providers.

Survey responses generally underscore the importance of assessing and implementing suicide prevention in the ED. Clinicians generally indicated feeling comfortable asking patients about concealed symptoms of depression and

Table 2. Baseline provider training/attitudes/beliefs.*

Item	Agree, total No. (%)
I have sufficient training in how to . . .	
. . . ASK patients about suicidal thoughts and behavior.	194 (55.0)
. . . ASSESS level of suicide risk in patients.	128 (36.4)
Additional training in how to. . .	
. . . ASK patients about suicidal thoughts and behavior would be helpful.	231 (65.3)
. . . ASSESS level of suicide risk in patients would be helpful.	239 (67.7)
Documentation in ED patient charts will accurately reflect the level to which ED providers inquire about suicidal thoughts or behaviors.	75 (21.5)
The ED is an important setting for identifying persons who may have underlying or concealed suicidal thoughts and behaviors.	310 (87.6)
As an ED healthcare provider, I play an important role in identifying/assessing underlying or concealed suicidal ideation in my patients.	302 (85.1)
I feel CONFIDENT in my abilities to detect underlying or concealed suicidal ideation in my patients.	156 (44.7)
I feel COMFORTABLE asking patients without mental health complaints about SYMPTOMS of . . .	
. . . depression.	225 (63.6)
. . . suicide ideation.	201 (56.8)
Detecting underlying or concealed suicidal thoughts in ED patients can help reduce the risk of future suicide attempts.	249 (71.3)
The ED where I work has a very good protocol for managing suicidal patients when they are identified.	228 (64.4)
When I suspect that my patient may have. . .	
. . . ATTEMPTED suicide, if available, I usually approach the patient's FAMILY or close FRIENDS, to ask about my patient's mental health and signs of suicidal behavior.	169 (49.3)
. . . ATTEMPTED suicide, if available, I usually approach the EMTs, to ask about my patient's mental health and signs of suicidal behavior.	214 (62.4)
. . . suicidal IDEATIONS, if available, I usually approach the patient's FAMILY or close FRIENDS to ask about my patient's mental health and signs of suicidal behavior.	172 (50.3)
. . . suicidal IDEATIONS, if available, I usually approach the EMTs to ask about my patient's mental health and signs of suicidal behavior.	192 (56.1)
If I suspect emotional distress in my patients, I always ask them directly if they are having suicidal thoughts.	196 (57.3)
I look and listen for signs/symptoms of emotional distress in all of my patients.	251 (72.8)

ED, emergency department; *EMT*, emergency medical technician.
* Totals do not always equal 362 due to missing data.

suicidality. Yet, the responses also revealed that subjects felt ED providers may be unaware of potential mental health issues if the patient does not present with specific mental health complaints. More than 1 of 7 providers (15.3%) reported they had never identified underlying or concealed suicide ideation in patients who did not present to the ED with a chief mental health complaint. Providers did not feel the extent of suicidality assessments was accurately documented in ED records. Overall, most providers agreed that additional training in how to ask about and assess suicidal thoughts and risk would be helpful.

Of note, clinicians at intervention sites were more likely to report that their ED had good protocols in place for managing suicidal patients. The results of the postintervention survey indicated this was not due to any enhancements.

LIMITATIONS

There are several limitations to this study that may reduce generalizability of findings to other settings. We did not assess differences in preintervention management of suicidal patients across sites. The method used to link individual subject baseline and follow-up surveys, by subject-supplied unique identification

Table 3. Chi-square test comparison of follow-up provider surveys, exposed versus not exposed.*

Item		Exposed, No. (%)	Not exposed, No. (%)	Chi square	
				Statistic	*P* value
If I suspect emotional distress in my patients, I always ask them about risk factors for suicide.	Agree	54 (58.1)	57 (41.3)	6.252	0.012
If I suspect emotional distress in my patients, I always ask them directly if they are having suicidal thoughts.	Agree	68 (73.1)	82 (59.4)	4.578	0.032
In the past MONTH, I have used an assessment guide to help determine LEVEL OF SUICIDE RISK.	Yes	25 (27.2)	13 (9.2)	13.326	<0.001
In the past month, I have used a guide to help in the MANAGEMENT of suicidal patients.	Yes	26 (28.3)	21 (14.8)	6.313	0.012
I feel CONFIDENT in my abilities to detect underlying or concealed suicidal ideation in my patients.	Agree	57 (61.3)	65 (46.4)	4.948	0.026
When I suspect that my patient may have ATTEMPTED suicide, if available, I usually approach FAMILY/close FRIENDS, to ask about mental health and signs of suicidal behavior.	Agree	63 (68.5)	71 (51.1)	6.880	0.009

* Significant results shown. Two hundred fifty follow-up surveys: 93 individuals reported seeing poster or receiving guide, 157 did not see/receive poster or guide. Totals do not always equal 93 or 157 due to missing data.

Table 4. Chi-square test comparison of comparator site and intervention sites, follow-up survey responses.*

Item	Control, No. (%) n = 22	Intervention, No. (%) n = 228	Chi square	
			Test statistic	*P* value†
1. In the past month, I have suspected underlying or concealed suicidal ideation in a patient who presented without a mental health–related chief complaint.				
Yes	4 (18.2)	117 (51.8)	9.052	0.003
2. The emergency department where I work has a very good protocol for managing suicidal patients when they are identified.				
Agree	10 (52.6)	166 (74.1)	4.045	0.044
3. If I suspect emotional distress in my patients, I always ask them directly if they are having suicidal thoughts.				
Agree	9 (45.0)	144 (64.8)	3.769	0.052

* Significant results shown. Totals do not always equal 228 and 22 for groups due to missing data. Agree/not agree is reduced from 5-point scale: 1, 2, 3 = not agree; 4, 5 = agree.
† Significance (2-sided).

codes, was variably effective by site. Many subjects entered different codes for the baseline and follow-up surveys, thus linking the surveys was not possible. Without linked data for preintervention and postintervention and without specific provider information in the follow-up survey, measurement of response bias also is limited. Furthermore, many providers at intervention sites did not report being exposed to the interventions. Providers that were originally more inclined to integrate suicide and mental health–related inquires in their ED assessments may have been more inclined to review the educational materials and subsequently report exposure to them. Other limitations include the fact that the study and results reflect the perceptions of ED providers regarding care for suicidal patients and did not measure patient outcomes and the fact that only 1 comparator site was used. Moreover, the ability to maintain the effects demonstrated in this study over time is unclear without a longer period of assessment.

CONCLUSION

Providers that individually received educational materials and providers at ED sites where the materials were available both indicated increased awareness of potential suicidality in ED patients. Overall, the intervention increased or improved provider perception of their knowledge and skills regarding identification and treatment of suicidality for approximately half of the providers receiving the guide or seeing the poster. ED providers generally feel that the ED is an important setting for identifying concealed suicidality in patients, that they can be a significant participant in this process, and that additional training in how to recognize patient suicidality is warranted.

Table 5. Gaining knowledge and skill questions (subjects exposed to intervention).*

Item	Not agree, No. (%)	Agree, No. (%)
As a result of the poster and/or guide, I have an increased KNOWLEDGE of signs of acute suicide risk.	30 (48.4)	32 (51.6)
As a result of the poster/guide, my SKILLS for ASKING about underlying or concealed suicidal ideation have improved.	33 (54.1)	28 (45.9)
As a result of the poster/guide, my SKILLS in MANAGING suicidal patients have improved.	36 (59.0)	25 (41.0)

* Subjects included answered yes to either "saw poster" or "received guide" (n = 93). Totals are not equal to 62 due to missing data.

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Address for Correspondence: Glenn W. Currier, MD, MPH, University of Rochester Medical Center, Department of Psychiatry, 300 Crittenden Blvd, Rochester, NY 14642. E-mail: Glenn_Currier@urmc.rochester.edu.

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Impact of the Mental Healthcare Delivery System on California Emergency Departments

Ashley Stone, MPH*‡
Debby Rogers, RN, MS*
Sheree Kruckenberg, MPA*
Alexis Lieser, MD†

* California Hospital Association, Sacramento, California
† California American College of Emergency Physicians, Sacramento, California
‡ University of California, Davis Public Health Sciences, Davis, California

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Introduction: This is an observational study of emergency departments (ED) in California to identify factors related to the magnitude of ED utilization by patients with mental health needs.

Methods: In 2010, an online survey was administered to ED directors in California querying them about factors related to the evaluation, timeliness to appropriate psychiatric treatment, and disposition of patients presenting to EDs with psychiatric complaints.

Results: One hundred twenty-three ED directors from 42 of California's 58 counties responded to the survey. The mean number of hours it took for psychiatric evaluations to be completed in the ED, from the time referral was placed to completed evaluation, was 5.97 hours (95% confidence interval [CI], 4.82–7.13). The average wait time for adult patients with a primary psychiatric diagnosis in the ED, once the decision to admit was made until placement into an inpatient psychiatric bed or transfer to an appropriate level of care, was 10.05 hours (95% CI, 8.69–11.52). The average wait time for pediatric patients with a primary psychiatric diagnosis was 12.97 hours (95% CI, 11.16–14.77). The most common reason reported for extended ED stays for this patient population was lack of inpatient psychiatric beds.

Conclusion: The extraordinary wait times for patients with mental illness in the ED, as well as the lack of resources available to EDs for effectively treating and appropriately placing these patients, indicate the existence of a mental health system in California that prevents patients in acute need of psychiatric treatment from getting it at the right time, in the right place. [West J Emerg Med. 2012;13(1):51–56.]

INTRODUCTION

California's mental healthcare delivery system—decentralized, underresourced, and disorganized—has recklessly collided with emergency medicine. Decades of cuts to local and state-funded mental health programs have led to an increased dependence on hospital emergency departments (ED) without corresponding resources.[1] The ED has become the only safety net provider for many patients with unmet mental health care needs in California.[2]

In the United States, about 1 in 4 adults suffers from a diagnosable mental disorder, and between 5% and 7% of adults suffer from a *severe* mental illness (SMI).[3] The California Department of Mental Health estimated in 2007 that there were nearly 2 million people in the state of California in need of mental health services for an SMI.[4] Mental illness, a leading cause of disability and suicide, carries huge social, economic, and personal costs.[5,6] Despite the awareness that mental illness poses a formidable burden for individuals, families, government payers, policy makers, and healthcare providers, the public health impact of mental illness remains severely underrecognized and underfunded.

In the 1950s and 1960s, the process of deinstitutionalization—the movement that shifted patients with mental illness from state hospitals to community-based care—

transformed California's mental healthcare delivery system. Bachrach[7] describes deinstitutionalization as a process involving 2 primary elements: "(1) the eschewal of traditional institutional settings—primarily State hospitals—for the care of the mentally ill; and (2) the concurrent expansion of community-based services for the treatment of these individuals." The process was aided by the passage of the Lanterman-Petris-Short (LPS) Act, signed into law by Governor Ronald Reagan in 1967, which significantly reduced involuntary commitment of individuals with mental illness to state hospitals.[8] To be involuntarily committed or treated under the LPS Act, patients had to meet imminent dangerousness criteria that effectively ended inpatient care for individuals with mental illness who met less rigid "need-for-hospitalization" criteria.[9]

The LPS Act accomplished what it set out to do: within 2 years of implementation, the number of state hospital patients decreased from 18,831 to 12,671, and by 1973, there were 7,000 patients in just 5 state institutions.[10] There was also a corresponding drop in the number of inpatient psychiatric beds in private hospitals. Between 1995 and 2009, there was a 30% loss of psychiatric beds (Figure 1). Currently, 30 of California's 58 counties lack inpatient psychiatric beds.[11] Many patients discharged from the state institutions, faced with inadequate care in their communities, became homeless or were put into "boarding houses" that offered little by way of psychiatric treatment.[2,10,12] Many discharged patients also found themselves incarcerated in the criminal justice system.[13,14] California in particular treats more individuals with mental illness in prison than outside of it; the Los Angeles county jail system has been called the largest mental health institution in the entire country.[15,16]

The promise of adequate and sustainable community-based care was unrealized, leading to a "revolving door" of homelessness, hospitalization, and incarceration for many individuals faced with debilitating mental illnesses in a fragmented system that does not provide appropriate levels of care when they are needed.[17] The Bronzan-McCorquodale Act of 1991, or *program realignment*, decentralized California's mental health system by shifting authority for mental health service delivery from the state to the counties. One of the intentions of realignment was to provide secure funding for community-based mental health services.[12] However, the contribution to counties from the state general fund has been determined more by history and politics than by the needs of counties for mental health funding. Program realignment legislation led to identification of recommended mental health services, but it was a guideline rather than a mandate with associated sanctions for not implementing community-based services.[18] Realignment funds have also not kept pace with population growth or inflation and have been negatively impacted by the economic downturn.

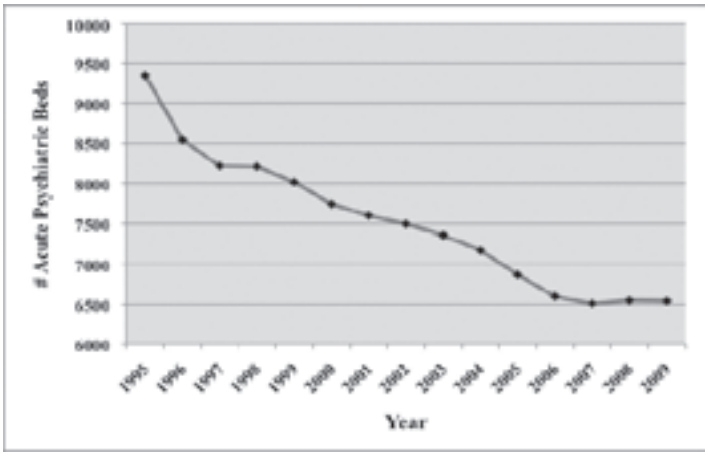

Figure 1. Total inpatient psychiatric beds in California, 1995–2009.

When mental health services and supports are unavailable or poorly coordinated, patients with unmet mental health needs turn to the ED for care.[2,19] In the current healthcare delivery system, EDs are the only institutional providers required by Federal law to evaluate anyone seeking care.[20] The Emergency Medical Treatment and Active Labor Act requires that all hospital EDs medically screen all patients seeking care in the ED—including evaluation and stabilization of patients suffering from mental illness.[21] In 2007, there were 10.1 million ED visits in California. More than 324,000 of these visits—3.2%—were by patients with a psychiatric diagnosis.[22] Research has shown a disproportionate increase in mental health–related ED visits, in comparison to ED visits in general. Between 1992 and 2001, the number of documented mental health–related ED visits increased by 38%, compared to an 8% increase in overall ED usage.[23]

This system of delivering nonemergent mental healthcare in the ED leads to inappropriate and inadequate patient care, issues with patient and staff safety, and overall decreased ED capacity.[1,2] There is a great need to reduce this reliance on EDs and identify more appropriate treatment options. *Healthy People 2020* identified the overarching goal for mental health and mental disorders as follows: "Improve mental health through prevention and by ensuring access to appropriate, quality mental health services."[24] Improving mental healthcare necessitates an understanding of how history, policy, institutions (including EDs), providers, and patients currently interact in the mental healthcare delivery system. This study evaluated a small subset of these interactions in California EDs, focusing on the patients they serve who present with psychiatric issues.

METHODS

Survey Development

The objective of the survey was to identify and quantify variables related to the magnitude of emergency and nonemergent ED utilization by patients with mental health needs by surveying hospital ED directors. The survey addressed the variables leading to prolonged ED stays, the wait

times to obtain a psychiatric evaluation and placement wait times, the concerns of staff that treat this patient group, and the external resources available to support the EDs when caring for these patients. This survey updated a 2006 survey, *Impact of Psychiatric Patients on Emergency Departments*,[25] which found that the reliance on EDs to provide care for patients with mental illness who have nonemergent physical or mental health needs creates undue strain on hospitals and their staff; moreover, it delays needed treatment for these individuals, since it takes significant amounts of time to appropriately evaluate and place patients in need of inpatient psychiatric care.

Survey Administration

To maximize response rates, the survey was administered through an online survey tool, which allowed embedded logic redirecting respondents, based on their responses. Using a member database of hospitals in California, a link to the survey was sent to all 259 ED directors at member hospitals with emergency rooms. There were an additional 68 member EDs without valid contact information for the ED directors; for each of these hospitals, a request was sent to the chief executive officer to forward to the current ED director. Of California's 58 counties, 55 have hospitals that are California Hospital Association members and have an ED.

Survey Analysis

Mean wait times were calculated from survey questions pertaining to length of wait times for evaluation, treatment, and disposition of patients in the ED. To check for statistically significant differences in median wait times, we conducted a Kruskal-Wallis test of the equality of medians for 2 or more populations. The Kruskal-Wallis test does not require that the data be normal, but instead uses the rank of the data values rather than the actual data values for the analysis.[26] Since the study data exhibit nonnormality, Kruskal-Wallis test is an appropriate choice.

RESULTS

In total, there were 123 respondents (response rate of 37.6%). The responses came from hospitals in 42 counties—76% of California counties with EDs. About a quarter of respondents (n = 33) indicated their hospitals have inpatient psychiatric beds, with 87.9% of these hospitals (n = 29) having inpatient beds designated for *involuntary* treatment. The mean wait time for psychiatric evaluation and placement determination in the ED, from the time the referral for evaluation (eg, psychiatric consult) is placed until completed evaluation, was 5.97 hours (95% confidence interval [CI], 4.82–7.13). The average wait time for adult patients with a primary psychiatric diagnosis in the ED, once the decision to admit has been made until placement into an inpatient psychiatric bed or transfer to an appropriate level of care, was 10.05 hours (95% CI, 8.6–11.52). The average wait time for pediatric patients with a primary psychiatric diagnosis was 12.97 hours (95% CI, 11.16–14.77). These average wait times exceeded those for nonpsychiatric patients in the ED, which was 7.10 hours (95% CI, 5.55–8.65) (Figure 2). Although data were not collected on total length of stay in the ED, these data suggest a total length of stay for psychiatric patients—from request for psychiatric evaluation to admission or transfer—of more than 16 hours for adults and 19 hours for children and adolescents. For several time points, hospitals with inpatient psychiatric beds had statistically significantly lower median wait times than those without inpatient psychiatric beds (Table 1).

About one third of ED directors indicated that their hospital operates a psychiatric evaluation team; 81% of the hospital psychiatric evaluation teams are available 24 hours a day, 7 days a week. The mean response time for hospital psychiatric emergency teams to evaluate patients in the ED was 1.61 hours (95% CI, l.29–1.93). More than 60% of ED directors indicated that their county operates a psychiatric evaluation team, with 71% of the county teams available 24 hours a day, 7 days a week. The mean response time for county psychiatric evaluation teams was 4.82 hours (95% CI, 4.04–5.59). Twenty percent of ED directors indicated that a private company operates a psychiatric evaluation team, with 86% of the private teams available 24 hours a day, 7 days a week. The mean response time for private teams to evaluate patients in the ED was 4.36 hours (95% CI, 3.09–5.64). Greater than 30% of hospitals reported *not* having access to a psychiatric evaluation team 24 hours a day, 7 days a week. Less than half of ED directors reported using or having access to community and county mental health resources to assist patients with mental health issues. On average, ED directors reported that 42% of patients presenting in their EDs with a behavioral health issue could have been adequately cared for at a nonemergency level of care (95% CI, 38%–47%).

"Lack of beds" was overwhelmingly the most common reason for extended ED stays in this patient population. Specifically, 78.3% of ED directors (n = 90) cited lack of pediatric/adolescent psychiatric inpatient beds as the most

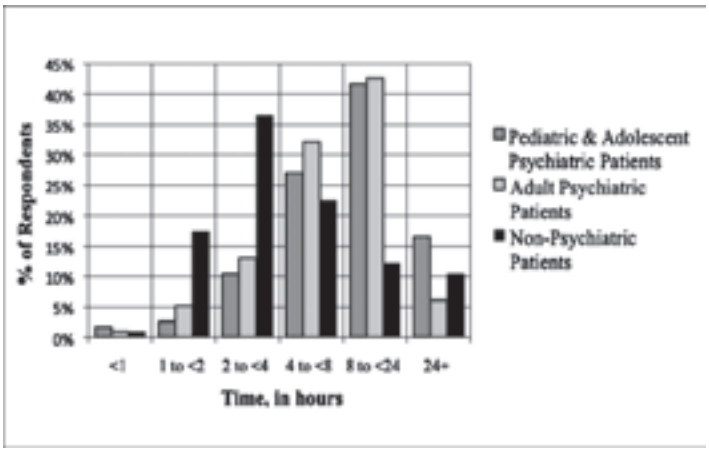

Figure 2. Average wait times, from decision to admit until admission.

Table 1. Median wait times (MWT) for psychiatric evaluation in emergency departments (ED).

	EDs in hospitals *with* inpatient psychiatric beds	EDs in hospitals *without* inpatient psychiatric beds
MWT* for psychiatric evaluation to be completed in the ED, from the time the referral is placed, hours	3	6
Adult patients with a primary psychiatric diagnosis: MWT from decision to admit until placement/appropriate transfer, hours	6	16

* Kruskal-Wallis test used to check for differences in MWT, $P < 0.001$.

common reason, followed closely by adult psychiatric inpatient beds (77.4%; n = 89). The 5 most common reported reasons for extended stays, as well as the percentage and proportion of respondents for each category, are presented in Table 2.

Open-ended questions were asked to allow respondents to express concerns not captured in the other survey questions. Comments included the following:

- Limited psychiatric evaluation team availability and resources after hours
- Problems with bed availability and disposition after psychiatric evaluation
- Nondesignated facilities cannot hold patients involuntarily after 24 hours
- Psychiatric evaluation teams will not come to evaluate a patient unless there is a bed available for the patient
- Shortage of medical-psychiatric beds for patients who require both mental health treatment and ongoing medical treatment
- Staffing/funding cut significantly in the last few years, leading to longer wait times for evaluation and placement
- Difficulty placing geriatric psychiatric patients
- Difficulty placing pregnant psychiatric patients
- Physical problem of getting an evaluation team to the ED because of geographic location
- Often evaluators will try and release patients who are a danger to themselves by commenting that "it is not against the law to be insane"
- County has to pay for anyone it hospitalizes; therefore, to make its funding stretch, it tries to not hospitalize anyone
- Closest facility that will take patients is an 8-hour drive away
- It is a fight to get our psychiatric patients the care they need

DISCUSSION

Mental illness poses a significant public health burden in California as well as nationally. In market economies such as the United States, the burden of disability associated with mental illness is at the same level as that of heart disease and cancer. Mental disorders lead to suicide, decreased quality of life for those who suffer from them, and enormous costs for the public health system.[21] Yet, mental health services and programs continue to be reduced as more patients need them. In 2010, former Governor Schwarzenegger announced a 60% cut in funding for community mental health programs, which will further ensure that the supply of mental health services does not meet the demand.[27]

The results of the survey indicate a mental healthcare delivery system in crisis—one with a high demand and decreasing supply of inpatient psychiatric beds. In one large county, ED directors reported that psychiatric evaluation teams would not come to evaluate patients in the ED if there are no inpatient psychiatric beds available to place patients, further delaying definitive treatment. Because patients have trouble accessing services in the community—including medication management and therapy—they use the ED for basic and intermediate care.[1] Our current mental health system still suffers from, and is largely a reflection of, the poor transition from state institutions to community-based treatment and the lack of local funding.

While perhaps well intentioned, the LPS Act has fostered a mental health system that requires seriously ill individuals to deteriorate to dangerousness or grave disability before they can receive needed treatment. The LPS Act gives authority to detain and transport to law enforcement, attending staff, or other persons designated by the county. Those designated may, "upon probable cause, take, or cause to be taken, the person into custody and place him or her in a facility designated by the county and approved by the State Department of Mental Health as a facility for 72-hour treatment and evaluation."[28] "LPS-designated facility" is not defined in statute, and while only such facilities can detain a person under 5150 statute, hospital EDs in nondesignated facilities still provide care for patients who may meet the criteria for an involuntary hold—some for more than 24 hours. Nondesignated EDs are thus often forced to choose between releasing a potentially dangerous patient and violating patient rights by involuntarily detaining patients beyond what is legally allowed by law.

Many ED directors reported that a significant portion of the psychiatric patients presenting in the ED could have been best cared in the outpatient setting. ED usage for needs such as an adjustment in psychiatric medication is symptomatic of both a suffering mental health system and a broader healthcare

Table 2. Most common reported reasons for extended emergency department stays related to evaluation and disposition of patients with psychiatric issues.

Reported reason	Respondents (%)	Proportion of respondents (115 responses to question)
1. Lack of beds to place or transfer patients		
Lack of pediatric/adolescent beds	78.3	90
Lack of adult beds	77.4	89
Lack of psychiatric medical-surgical beds	51.3	59
Lack of geriatric beds	43.5	50
2. Inability to manage comorbid medical and psychiatric conditions	37.4	43
3. Lack of resources to conduct psychiatric evaluation	37.4	43
4. Lack of appropriate lower levels of outpatient care for discharge	32.3	37
5. Requirement for additional medical screening before psychiatric inpatient placement	29.6	34

system in which access to care is not guaranteed.[1,2] According to the National Hospital Ambulatory Medical Care Survey, only 12.9% of all ED visits in the United States in 2006 were classified as emergent.[29] When the ED is not used for true acute care services and emergencies because patients do not have access to outpatient services to manage their disease process, there can be serious consequences, such as patients' needs not always fully met, patients enduring long wait times, and staff burnout.[30] Despite these recognized threats to patients and staff, the mental health delivery system has deteriorated to a point where the only choice of care for patients with mental illness is very often the ED. The Council on Medical Service described the influx of patients seeking psychiatric care in the ED as a "symptom of a larger systemic problem. . . . The crumbling infrastructure of the mental health system is an example of what could happen in other areas of medicine if not properly financed according to the needs of the population."[1]

Frank Lanterman, an author of the LPS Act, said in the early 1980s, "I wanted the LPS Act to help the mentally ill. I never meant for it to prevent those who need care from receiving it. The law has to be changed."[31] The LPS Act, signed in 1967, remains unchanged, and the community-based services promised by deinstitutionalization never materialized. Consequently, the ED has become a way station for patients stuck in a mental health system in desperate need of transformation.

LIMITATIONS

This study has some notable limitations. First, we cannot verify that the information obtained from ED directors was completely based on actual data. Rather, the 123 survey responses from ED directors represent both data-based and anecdotal accounts of the experiences of individual hospital EDs in treating patients suffering with psychiatric disorders. Secondly, many of the questions forced respondents to select answers representing ranges of values (eg, "1 to less than 4 hours"), thus sacrificing precision in responses and subsequent analysis. Despite these limitations, the study's broad representation of most California counties renders the results an important addition to the literature on EDs and psychiatric services in California.

CONCLUSION

The current mental health system—fostered in large part by the LPS Act and the decades-long prioritization of deinstitutionalization—provides no room for prevention and, as indicated by the results of this study, leads to long ED visits for patients suffering from mental illness. This population experiences wait times far exceeding those of patients presenting in the ED for physical health problems. This system is failing both patients, who suffer from debilitating mental illnesses, and healthcare providers, who are ill prepared and underresourced to meet the increasing demand of patients with unmet mental health care needs. Individuals suffering from mental illnesses deserve treatment in the right place, at the right time.

Address for Correspondence: Debby Rogers, RN, MS, California Hospital Association, 1215 K St, Ste 800, Sacramento, CA 95814. E-mail: drogers@calhospital.org.

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Life in the Inner City

Stephen D. Docherty, DO Keck School of Medicine of the University of Southern California, Department of Emergency Medicine, Los Angeles, California

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A 56-year-old homeless male presented to the emergency department with a bloody nose. When asked if he took medications, he replied in the affirmative. When asked which medications he took on a regular basis he produced the following photograph (Figure).

Address for Correspondence: Stephen D. Docherty, DO, Keck School of Medicine of the University of Southern California, Department of Emergency Medicine, LAC+USC Medical Center, 1200 N State St, Rm 1011, Los Angeles, CA 90033. E-mail: sdochert@usc.edu.

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Figure. Photo of medications presented.

Bath Salts: The Ivory Wave of Trouble

Travis D. Olives, MD, MPH, MEd* * Hennepin County Medical Center, Minneapolis, Minnesota
Benjamin S. Orozco, MD† † Regions Hospital Toxicology Education and Clinical Service, St Paul, Minnesota
Samuel J. Stellpflug, MD†

Supervising Section Editor: Jeffrey R. Suchard, MD
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INTRODUCTION

Mephedrone and MDPV are both β-ketophenethylamine derivatives of cathinone, a compound isolated from the East African plant *Catha edulis* (khat, qat). Mephedrone is commonly referred to as plant food, MCAT, 4-MMC, meow meow, meph, and drone; MDPV is commonly called MTV, MDPK, Magic, and Super Coke. Both are structurally similar to amphetamines, with mephedrone sharing close similarities with methamphetamine and MDPV with ecstasy (3,4-methylenedioxymethamphetamine; MDMA). Bath salts pose an increasing public health risk in the United States, with reports of toxicity and mortality increasing along with calls to poison centers throughout the United States. Packages labeled with innocuous monikers such as *White Ice*, *Ivory Wave*, *Ocean Snow*, *Lunar Wave*, and *Vanilla Sky* intentionally belie the dangerous substances within, which are by no means intended to replace legitimate bath products. The white or tan crystalline powder commonly is administered by nasal insufflation or oral ingestion; however, rectal suppository and less commonly, intramuscular or intravenous injection, are also reported.[1,2]

A movement to ban these substances is growing in the United States, following similar actions in Europe.[3] Although successfully outlawed in some locales, this movement has not eliminated the public health hazards posed by mephedrone or MDPV. Emergency physicians (EP) should thus be knowledgeable in the epidemiology of bath salt abuse, the clinical toxidrome with which bath salt toxicity presents, and appropriate treatment strategies to reduce morbidity and mortality in patients presenting with bath salt toxicity.

CLINICAL EFFECTS

Based on studies of similar compounds, mephedrone and MDPV may possess intrinsic stimulant properties owing to their effects on plasma membrane dopamine, norepinephrine, and serotonin transporters, resulting in both reuptake inhibition and direct agonist activity.[1,4,5] The exact action of mephedrone and MDPV remains somewhat theoretical, as previously tested compounds have demonstrated varying effects despite their structural similarities. Norepinephrine and dopamine reuptake inhibition are likely prominent in MDPV and mephedrone, resulting in a sympathomimetic toxidrome similar to that of more familiar illicit substances with which most providers are more familiar, including cocaine, methamphetamine, and ecstasy.[6]

User reports describe a euphoric high lasting between 2 to 4 hours with prominent letdown effects lasting several hours afterward. Reported doses range from 5 to 10 mg for the more lipophilic MDPV (although 1 patient reported taking 2 g over an unclear time course) and 100 to 500 mg for mephedrone.[7–10] Redosing of both is common. Euphoria, empathic mood, sexual stimulation, subjectively greater mental focus, and increased energy are reported in the highs of both substances.[1,7,8] In a recent survey study of past mephedrone users, the 1,506 participants revealed that ecstacy compared most similarly. Significant complications have also been documented, including seizure activity, severe agitation, myocarditis, and chest pain, as well as compulsive dosing to sustain effect.[8,11–13] Case fatalities resulting from bath salt consumption, as well as consumption of mephedrone and MDPV from other sources, have also been published in the literature.[11,14–16]

Some of the reported cases can shed light on the specific clinical manifestations.

A 36-year-old male in the Netherlands became acutely agitated and enraged after ingesting mephedrone along with cocaine, and subsequently lost consciousness and died despite resuscitation efforts.[16] A 29-year-old male found unresponsive at a nightclub died of cerebral edema and brainstem herniation. Qualitative toxicologic blood screening revealed mephedrone, and no other substance, in his blood.[11] Serum sodium was noted to be 125 mmol/L, later suggested by laboratory data to have resulted from water intoxication. The first synthetic cathinone-related death in the United States, described in the scientific literature, involved a 22-year-old male who was found unresponsive and subsequently died at the receiving hospital.

Blood and urine tested positive for mephedrone, heroin metabolites, codeine, and doxylamine.[14] One case of mephedrone-related myocarditis has also been reported in the literature.[13] In this instance, a 19-year-old male presented with crushing chest pain after ingesting mephedrone sold as "not-for-human-consumption" plant food. Electrocardiographic changes with greater than 3 mm ST-segment elevation in the anterolateral leads, and high T2 signal at the lateral left ventricle on cardiac magnetic resonance imaging, confirmed the diagnosis of myocarditis. One additional case of documented 2-mm ST depression in a patient exposed to MDPV is reported, although this case did not result in death and resolved with only sublingual nitrates.[10] MDPV was implicated by history, though never analytically confirmed, to be the cause of the death of a 24-year-old man reported by the lay press.[17] A recent series of cases of analytically confirmed mephedrone toxicity verified the sympathomimetic toxidrome that accompanies its use and provided insight into the spectrum of care and disposition undertaken for acute mephedrone toxicity. Among these cases were 4 emergency-department (ED) and short-stay discharges, 2 ward admissions, and 1 intensive care unit admission/subsequent death, reported in 7 cases.[11]

SCOPE OF THE PROBLEM

Rising use patterns and cases of self-mutilation and suicide have garnered increasing media attention.[18] A recent survey of young adult "clubbers" in England revealed that 33.6% of respondents had used mephedrone in the past month, nearing the last-month use of cocaine, MDMA, and ketamine. More than 40% of respondents reported ever having used mephedrone.[19] While reports of deaths due to mephedrone and MDPV consumption litter popular media outlets, several have also been reported in the scientific literature.[20–22] In a case series of 4 fatalities in Scotland in which mephedrone was detected in postmortem femoral blood samples, 2 of the deaths were officially attributed to mephedrone toxicity, 1 was attributed to an abdominal stab wound, and another was not described. In 2 of the 4 cases, β-keto amphetamines were not suspected but found on gas chromatography-mass spectrometry; multiple coingestants were identified in 3 of the cases.[23] As with other sympathomimetic drugs of abuse, the morbidity and mortality associated with bath salts stem both from direct physiologic toxicity and from behaviors spurred by the drugs' effects.

Incident usage of cathinone-derived sympathomimetic bath salts continues to increase in the United States. Data are limited, largely because unique surveillance coding of bath salt calls to poison centers was initiated only in mid 2010 (Bailey, personal communication, April 25, 2011). Nationally, 302 calls regarding bath salts were made to poison centers in 2010; by October 3, a total of 5,226 calls to poison centers had already been made in 2011 (Bailey, personal communication, October 3, 2011).[24,25] These data represent all calls made to US poison centers during the described time periods, and certainly represent a small slice of all bath salt use and morbidity in the United States. By contrast, poison center calls referencing synthetic cannabinoids have not seen the same increase in volume. This is a comparison that may prove instructive for EPs: the designer synthetic marijuana movement is of similar scope, likely familiar to most EPs, and it seems to be on the decline, while bath salt use seems to be on the rise (see Figure).[26]

On September 7, 2011 the US Drug Enforcement Agency (DEA) announced that it will exercise its emergency scheduling authority and temporarily control MDPV and mephedrone (along with methylone, a similar compound), as schedule I substances for a minimum of 12 months.[27] Senator Charles Schumer (D, New York), who had introduced legislation in February 2011 that urged defining both as schedule I controlled substances, now reports that he will pursue permanent scheduling.[28–30] The DEA considers both MDPV and mephedrone analogs of methcathinone, a schedule I substance, and thus covered by the Federal Analogue Act, but only if intended for human consumption. At the time of the DEA's announcement, at least 33 states had independently taken measures to control the substances specifically in bath salts.[27–35]

Some have suggested that scheduling these new "legal highs" has little effect on actual control of the substance, and that the shift from "legal" to illegal may result in increased risks for users owing to the possibility of adulterated manufacturing and reliance on street dealers.[36,37] The results of previous bans on mephedrone suggest a dim end to the battle to reduce its use through legal actions. One instructive example of this is the ban on mephedrone enacted in the United Kingdom on April 16, 2010.[38] A study of national poison center data in the year leading up to the ban revealed roughly 1,800 contacts with mephedrone-poisoned patients, incrementally increasing month by month.[39] A survey completed 3 months after banning the substance, including 150 previous mephedrone users, demonstrated that two thirds of users continued using the drug despite its illegality.[36]

SUGGESTED EVALUATION AND MANAGEMENT

Specific recommendations for ED evaluation and management of isolated bath salts exposures, specifically for mephedrone or MDPV, would be difficult in most cases presenting to the ED, and interventions typically undertaken for more common sympathomimetic toxicities remain the first line of therapy. A specific antidote does not exist, and few laboratories have the capacity to screen serum or urine for specific bath salts; to our knowledge, none on a timeline useful in the acute care setting. Features typical of the bath salt toxidrome include, but are not limited to, altered mentation and sensorium, agitation, tachycardia, hypertension, and hyperthermia, with other symptoms possible as well. Such a presentation is not only common but also consistent with a wide range of disease states, both toxicologic and nontoxicologic. Even among patients with unequivocally toxicologic etiologies

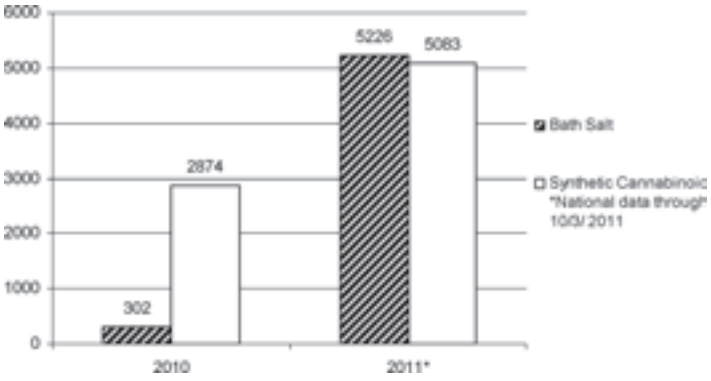

Figure. US poison center calls for bath salts and synthetic cannabinoids, 2010–2011 (Bailey, personal communication, October 3, 2011).

to explain these symptoms, bath salts are but one in a multitude of potential intoxicants and diagnoses, including serotonin syndrome, neuroleptic malignant syndrome, anticholinergic and sympathomimetic toxidromes, drug withdrawal syndrome, and exposure to older hallucinogens (ie, lysergic acid diethylamine, phencyclidine) or to a newer tryptamine or phenethylamine hallucinogen. At the moment, it remains advisable to keep designer β-ketophenethylamine cathinone derivatives such as mephedrone and MDPV on the differential diagnosis for patients presenting with the symptoms listed above or with toxidromes largely consistent with sympathomimetic toxicity.

Clinical experience in the treatment of bath salt toxicity is limited, and stratifying risk for certain outcomes that draw concern with other sympathomimetics or with new independent concerning outcomes is difficult. The risk for acute coronary syndrome, rhabdomyolysis, and serotonin syndrome, for example, remains unclear at this time, as does the subsequent need for laboratory testing and appropriate monitoring parameters. The case reports discussed above document important outcomes including death, myocarditis, agitated delirium, and hyponatremia, all of which merit considerable care in the management of documented or suspected bath salt toxicity. It thus seems prudent to include in the ED workup several key monitoring and therapeutic interventions. Peripheral intravenous access and cardiac monitoring are essential starting points, as is obtaining full vital signs at the outset of the visit *including* temperature, and repeating those vital signs during the ED stay. We recommend, at minimum, vital checks every 30 minutes until stable. Electrocardiograms and chest radiographs should be obtained for all patients presenting with tachycardia, chest pain, or shortness of breath. The specific role of cardiac markers in the evaluation of these patients has not been elucidated; however, given previously documented mephedrone cardiac toxicity[13] and well-known propensity of amphetamines to cause direct cardiac damage by way of vasospasm and ischemia, it is prudent to approach the treatment of patients with acute bath salt toxicity and chest pain with at least the same level of caution as nonintoxicated patients with cardiac chest pain. A basic metabolic panel should be drawn for all patients to seek out hyponatremia and metabolic acidosis. The risk of rhabdomyolysis is uncertain, but in the setting of persistent agitation, obtaining a baseline creatine kinase would be reasonable. A complete blood count is unlikely to aid in the workup of bath salt toxicity, although in the setting of altered mental status, agitation, and hyperthermia of unclear etiology, it may be improper to ignore.

Agitation can be controlled with benzodiazepines as first-line therapy. Other supportive measures, including fluid management and temperature control, may play significant roles in individual cases. More advanced fluid management techniques may be required for cases complicated by hyponatremia or rhabdomyolysis. Seizures, should they occur, would be expected to respond to medications acting through γ-aminobutyric acid promotion, such as benzodiazepines, barbiturates, and propofol. Airway compromise, extreme sedation needs, seizures evolving to status epilepticus, and uncontrolled agitation all suggest the need for advanced airway management.

A controversial portion of the workup for any patient whose primary problem is exposure to a drug or poison is the "tox screen." It should be noted that, in general, for emergency-department decision making for the toxicologic patient, this screening test is unhelpful.[40] This notwithstanding, there is increasing precedent for detection of both mephedrone and MDPV. The first isolated mephedrone-related case report stated that the substance was not identified by routine toxicologic analysis, but subsequent reports had success with more advanced testing.[2] Identification of mephedrone from samples seized in police raids has been recently described.[41] Both mephedrone and MDPV have recently been identified in urine samples by gas chromatography-mass spectrometry, and mephedrone can be identified by liquid chromatography-mass spectrometry.[42–45] MDPV has also been identified by nuclear magnetic resonance spectroscopic analysis.[46] At least 1 commercial laboratory currently offers diagnostic testing on urine for both via liquid chromatography-tandem mass spectrometry with a reporting limit of 1.0 ng/mL.[47] Despite these advances, a urine or blood toxicologic assessment negative for amphetamines in the face of convincing history and physical examination should not dissuade the astute EP from undertaking care appropriate for a bath salt ingestion. As with many emerging substances of abuse, little data are available to describe the sensitivity of currently used immunoassays in broad terms, and at this time it is likely that the clinical assessment of the patient who has ingested bath salts will be more sensitive for the diagnosis than a toxicologic screen.

CONCLUSION

There is a limited but building body of literature consisting largely of case reports, case series, surveys, media releases, and poison center data regarding mephedrone and MDPV toxicity.

Chemical structure, case reports, popular media, user forums, and the sparse provider data that exist all support the notion that both compounds act to create a sympathomimetic toxidrome akin to that of cocaine and certain amphetamines. Proposed evaluation and management arise from experience and case reports only, but are likely congruous with standard proposed management of more well-known drugs such as cocaine, methamphetamine, and MDMA. Epidemiologic data are suggestive of a growing disease burden stemming from markedly increasing popularity of these so-called legal highs. Even in areas in which they have been banned, the problem of their acute toxicity persists and should be recognized by the well-prepared EP.

Address for Correspondence: Travis D. Olives, MD, MPH, MEd, Hennepin County Medical Center, 701 Park Ave, Mailcode 825, Minneapolis, MN 55415. E-mail: travis.olives@gmail.com.

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Dose-Dependent Response to Cyclodextrin Infusion in a Rat Model of Verapamil Toxicity

Allan R. Mottram, MD*
Sean M. Bryant, MD†
Steven E. Aks, DO†

* University of Wisconsin School of Medicine and Public Health, Department of Medicine, Madison, Wisconsin
† Cook County-Stroger Hospital, Department of Emergency Medicine, Chicago, Illinois

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Introduction: Sulfobutylether-β-cyclodextrin (SBE-CD) is a pharmaceutical excipient known to bind verapamil. Following intravenous administration, clearance of SBE-CD approximates glomerular filtration rate. We hypothesized that infusion of SBE-CD would increase time to asystole in a rat model of verapamil toxicity in a dose-dependent manner. The objective was to demonstrate the effect of a range of SBE-CD concentrations in a rat model of verapamil toxicity.

Methods: Twenty-five Wistar rats were allocated to control or 1 of 4 intervention groups. All received ketamine and diazepam anesthesia followed by verapamil infusion 32 mg/kg/h. The verapamil infusion for the intervention groups was premixed with SBE-CD in a 1:1, 1:2, 1:4, or 1:8 molar ratio (verapamil to SBE-CD). The control group infusion did not contain SBE-CD. Additional saline or water was added to the infusion so that the total volume infused was the same across groups, and the osmolality was maintained as close to physiologic as possible. Heart rate, respiratory rate, and temperature were monitored. The primary endpoint was time to asystole.

Results: Verapamil coinfused with SBE-CD in a molar ratio of 1:4 resulted in prolonged time to asystole compared to control (21.2 minutes vs 17.6 minutes, $P < 0.05$). There were no differences in time to asystole between control and any other intervention group. There was no significant difference in time to apnea between control and any intervention group. We assessed the effect of a range of SBE-CD concentrations and identified 1 concentration that prolonged time to asystole. Mechanisms that may explain this effect include optimal volume expansion with a hyperosmolar cyclodextrin containing solution, complexation of verapamil within the hydrophobic cyclodextrin pore, and/or complexation within micelle-like aggregates of cyclodextrin. However, mechanistic explanations for the observed findings are speculative at this point.

Conclusion: The 1:4 verapamil to SBE-CD concentration was modestly effective with SBE-CD concentrations above and below this range demonstrating nonstatistically significant improvements in time to asystole. [West J Emerg Med. 2012;13(1):63–67.]

INTRODUCTION

Cyclodextrins are hydrophilic circular oligosaccharides of various sizes containing a hydrophobic core. Lipophilic molecules fit into this hydrophobic core via nonionic bonds. Altering the number of substituent groups to the outer ring of the molecule significantly changes its characteristics, including its affinity for complexing with drugs and its osmolarity. They are widely used as pharmaceutical excipients to modify drug solubility and stability.[1]

Rather than improving drug delivery, we are interested in the ability of cyclodextrins to enhance elimination of drug from the body. This concept has proven feasible by the success of

sugammadex, a gamma-cyclodextrin that was modified to function as an intravenous reversal agent for rocuronium-induced neuromuscular blockade following anesthesia.[2,3] Cyclodextrin molecules bind target drugs as a function of a complexation constant.[4] Modification of hydroxyl groups at the outer ring of the hydrophobic core enhances this property. We hypothesize that a favorable complexation constant, in addition to an equilibrium inequality driving the formula towards complexation, would allow binding and subsequent renal elimination of the target drug.

Additional mechanisms may contribute to reversal of toxicity. Micelle-like aggregates of cyclodextrin and drug-cyclodextrin complexes may solublize lipophilic drugs.[5] By creating an intravascular sink, the drug is prevented from reaching the target organ. This is analogous to one proposed mechanism for how intralipid infusion works in the setting of bupivicaine, chlomipramine, and verapamil overdose.[6–8]

The study of cyclodextrins as antidotal therapy does appear in the literature. However, in contrast to the massive body of work regarding pharmaceutical applications of cyclodextrins, those regarding toxicologic applications are minimal. In-vitro inactivation of sarin and soman, treatment of organophosphate poisoning, and treatment of tunicaminyluracil toxicity is reported.[9–12] The utility of cycodextrins as antidotes for the most common cardiovascular and neurologic toxins has not been evaluated.

The mechanism of drug reversal with intravenous cyclodextrin infusion is sound and has proven efficacy (ie, sugammadex). As such, we sought to apply this concept to models of drug toxicity which have shown significant morbidity and mortality and for which there are currently limited therapeutic options. Potential drug candidates that fit these criteria included tricyclic antidepressants, propoxyphene, calcium-channel antagonists, methamphetamine, and cocaine among others. Selecting the ideal drug was aided by the pharmaceutical chemistry literature. Verapamil is known to complex well with sulfobutylether-β-cyclodextrin (SBE-CD) as evidenced by bench work of verapamil enantiomeric separation.[13,14]

We have previously undertaken a study assessing the utility of SBE-CD as antidotal therapy for verapamil-induced cardiotoxicity in rats.[15] This was a rescue model that used the same verapamil infusion as in the current study but a much higher concentration of SBE-CD (1:16 molar ratio of verapamil to cyclodextrin; 2.25 g/kg SBE-CD, 32 mg/kg/h verapamil). This was administered as a bolus after the onset of cardiotoxicity. Survival times were poorer in the cyclodextrin group, which we proposed was primarily related to the hyperosmolar load that accompanied the SBE-CD infusion (1,025 mOsm/kg) in combination with verapamil-induced cardiogenic shock. Additional confounding factors included use of a rescue model that induced severe, refractory cardiogenic shock prior to administration of our study drug, and the occurrence of isoflurane induced apnea. As such, with the current study, we used isoflurane for induction only, maintained anesthesia with ketamine and diazepam, studied a range of SBE-CD doses, and designed the study as a coinfusion model rather than a rescue treatment model. The aim of this study was to demonstrate the effect of a range of SBE-CD concentrations in a rat model of verapamil toxicity.

METHODS

The study design was an unblinded controlled trial to assess the effect of SBE-CD infusion in a rat model of verapamil toxicity. The protocol was approved by the Animal Care Committee at the University of Illinois Chicago. Male Wistar rats (strain 003) weighing 224 to 301 g with indwelling central venous femoral lines were purchased from Charles River Laboratories and housed in single cages with free access to food and water. Verapamil (2.5 mg/mL) was purchased from Hospira Inc (Lake Forest, Illinois). A 30% w/v solution of SBE-CD was prepared by dissolving 3 g dry SBE-CD in 100 mL of sterile water.

Preceding the experimental protocol, rats were allocated to either the control group (n = 5) or 1 of 4 intervention groups (n = 20). All rats underwent induction with 3% isoflurane via enclosed box. They were then weighed, and anesthesia was maintained with weight-based doses of ketamine (90 mg/kg initial; 30 mg/kg supplemental) and diazepam (6 mg/kg initial; 2 mg/kg supplemental). Following this, they were placed on a warming pad, and central venous lines were accessed for administration of verapamil and SBE-CD via infusion pump (Outlook 100 B.Braun, Bethlehem, Pennsylvania). Cardiac monitoring electrodes were attached using alligator clips for continuous monitoring (Escort II, Medical Data Electronics, Arleta, California). The study protocol was initiated with 5 minutes of observation under anesthesia for all rats to ensure stable respiratory rate and heart rate while maintaining adequate depth of anesthesia. Following this, the verapamil and SBE-CD infusion was initiated via central line. Verapamil (32 mg/kg/h) and SBE-CD (dose specified later) were compounded into the same infusion bag. The drugs were diluted in normal saline with additional saline and/or water added to the final concentrations as needed such that each subject would receive the same volume infusion (total volume 18.6 ml/kg/h across all groups) and the maximum osmolality would be limited. The verapamil dose was selected as it is the established LD50. It was efficacious in our prior work and is only slightly less than the dose established by Tebbutt et al (37.5 mg/kg/h) in a similar rat model of verapamil toxicity.[15,7] The control group (group 1, n = 5) received no SBE-CD. The 4 intervention groups received verapamil and SBE-CD with the SBE-CD dose being 141 mg/kg (group 2, n = 5, 1:1 molar ratio of verapamil to SBE-CD), 282 mg/kg (group 3, n = 5, 1:2 molar ratio of verapamil to SBE-CD), 564 mg/kg (group 4, n = 5, 1:4 molar ratio of verapamil to SBE-CD), and 1,227 mg/kg (group 5, n = 5, 1:8 molar ratio of verapamil to SBE-CD). Infusate osmolality

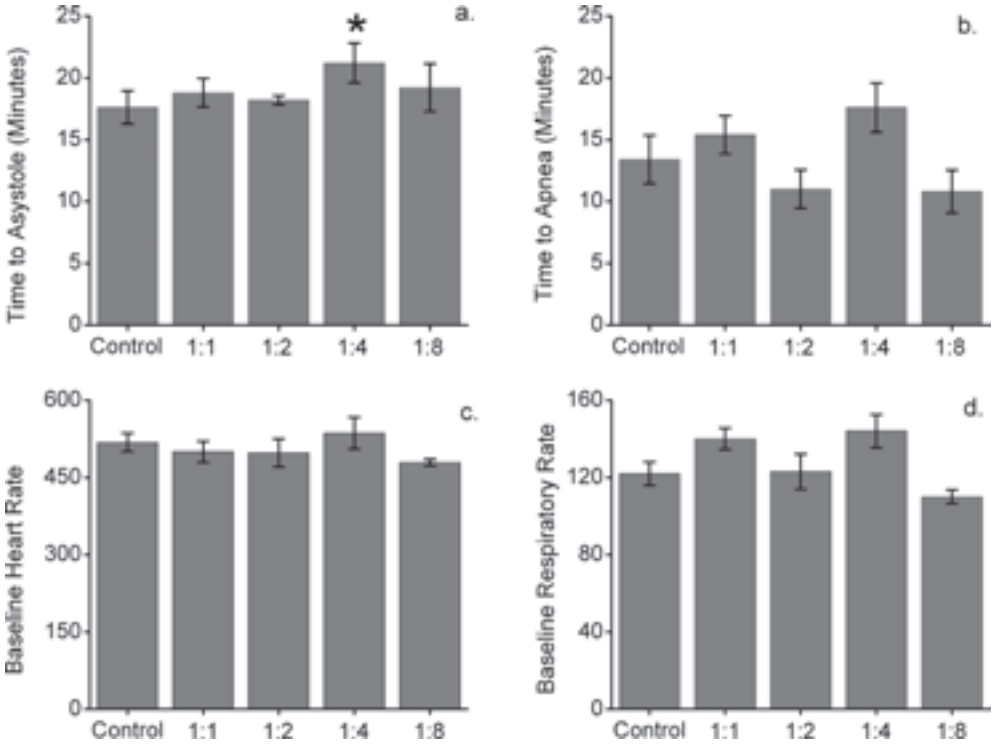

Figure 1. a, Time to asytole represented as absolute time ± standard error (SE). The 1:4 group time to asystole (*) was significantly prolonged compared to control (21.2 vs 17.6 minutes, $P < 0.05$). b, Time to apnea represented as absolute time ± SE. There was no significant difference between control and intervention groups. c, Baseline heart rate with 95% confidence interval (CI). There was no significant difference between control and intervention groups. d, Baseline respiratory rate with 95% CI. There was significant variability between control and intervention groups 2 (mean difference 18.2, 95% CI 4.7–31.7, $P < 0.05$) and 4 (mean difference 21.5, 95% CI 7.9–35, $P < 0.05$).

varied between 300 mOsm/L (1:1 infusion) and 352 mOsm/L (1:8 infusion). Of note, SBE-CD is specified to have no effect when administered to rats intravenously up to 2,000 mg/kg as documented in commonly available materials safety data sheet documents. Heart rate, respiratory rate, and temperature were monitored from onset of anesthesia to asystole or 1 h. The primary endpoint was time to asystole. Statistical analysis was performed using SPSS Statistics 18.0 (SPSS Inc, Chicago, Illinois). Data were analyzed for descriptive characteristics to ensure it was normally distributed. Further analysis was via paired t tests (discrete data) or one-way analysis of variance with Bonferroni post-hoc t tests (continuous data) as appropriate. Significance was set at $P < 0.05$. Figures were generated via Origin 7.5 (OriginLab Corporation, Northampton, Massachusetts).

RESULTS

The baseline heart rate did not vary between control and intervention groups over the first 5 minutes of anesthesia, prior to initiation of verapamil and SBE-CD infusion. The baseline respiratory rate did vary between control and intervention groups 2 (mean difference 18.2, 95% confidence interval [CI] 4.7–31.7, $P < 0.05$) and 4 (mean difference 21.5, 95% CI 7.9–35, $P < 0.05$) during this time period (Figure 1). There were no significant differences between control and intervention group heart rate or respiratory rate during the drug infusion period (Figure 1). Time to asystole was significantly prolonged in group 4 compared to control (21.2 vs 17.6 minutes, $P < 0.05$; Figure 2). There were no other differences in time to asystole between control and intervention groups (Figure 1). There was no significant difference in time to apnea between control and any intervention group (Figure 1).

DISCUSSION

This study sought to identify a treatment benefit defined as a prolonged time to asystole in verapamil cardiotoxic rats coinfused with 141 mg/kg, 282 mg/kg, 564 mg/kg, or 1,227 mg/kg SBE-CD. These doses corresponded to a 1:1, 1:2, 1:4, and 1:8 molar ratio of verapamil to cyclodextrin respectively. The group infused with verapamil and SBE-CD in a 1:4 ratio demonstrated prolonged time to asystole. It is possible that this resulted from complexation of SBE-CD with verapamil, either in the infusion bag or intravascularly, followed by increased renal clearance, decreased tissue concentration, and reduced toxicity. Complexation and sequestration of verapamil within the hydrophobic cyclodextrin pore or within micelle-like

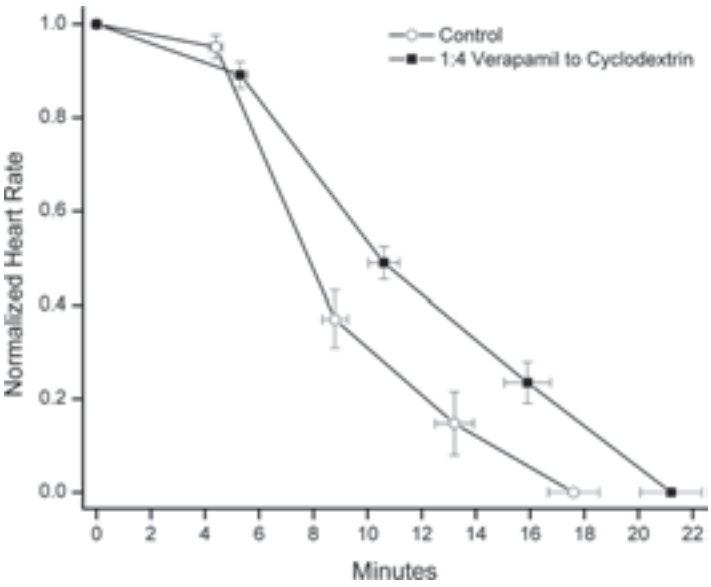

Figure 2. Normalized heart rate versus time to asystole for control and group 4 (1:4 verapamil to sulfobutylether-β-cyclodextrin). Values are indicated as mean normalized heart rate ± standard error (vertical error bars). Measurements are plotted from the beginning of drug infusion and at 25% increments of time to asystole. The horizontal error bars denote the variability in time to asystole between groups. Mean heart rate did not differ between groups. Time to asystole was significantly shorter in the control group (17.6 vs 21.2 minutes, $P < 0.05$).

aggregates of cyclodextrin is an intriguing mechanism, though speculative at this point. An alternative explanation is that 18 ml/kg/h of hypertonic solution (315 mOsm/kg) provided an ideal intravascular volume expansion, thus prolonging time to asystole. Arguing against this alternative is the fact that the solution administered to the 1:2 group was of the same approximate osmolality (318 mOsm/kg) and did not show any improvement in time to asystole.

LIMITATIONS

This project has several significant limitations. Rats in all groups experienced apnea very early in the course of the protocol, despite the modification of our protocol (ie, the use of diazepam and ketamine). Ideally, they would have been artificially ventilated to remove apnea as a confounding variable. We were unable to perform invasive hemodynamic monitoring. Such a capability would have allowed us to better characterize the hemodynamic effects of SBE-CD. Lastly, a small n, utilizing a small animal model, and lack of clear mechanism of action limits the applicability of our findings.

CONCLUSION

In conclusion, this study explores the application of a novel molecule to a challenging poisoning scenario. It demonstrated prolonged time to asystole in rats infused with both verapamil and SBE-CD compared to those infused with verapamil only. While acknowledging its limitations, the study provides support for further investigation of the use of cyclodextrins as antidotes to drug toxicity.

Address for Correspondence: Allan R. Mottram, MD, University of Wisconsin School of Medicine and Public Health, Department of Emergency Medicine, F2/204 Clinical Science Center MC3280, 600 Highland Ave, Madison, WI 53792. E-mail: allan.mottram@gmail.com

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The Role for Coagulation Markers in Mild Snakebite Envenomations

Risa S. Moriarity, MD
Sylvia Dryer, MPH
William Replogle, PhD
Richard L. Summers, MD

University of Mississippi Medical Center, Department of Emergency Medicine, Jackson, Mississippi

Supervising Section Editor: Brandon K. Wills, DO, MS
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Introduction: The majority of patients seeking medical treatment for snakebites do not suffer from severe envenomation. However, no guidelines exist for ordering coagulation markers in patients with minimal or moderate envenomation, nor in those who do not receive antivenom. In this study, we sought to determine whether it was possible to limit the practice of ordering coagulation studies to those patients suffering severe envenomation, rattlesnake envenomation, or both.

Methods: A retrospective chart review was performed on all cases of crotalid snakebite presenting to an adult emergency department (ED) from April 1998 to June 2006. Each chart was abstracted for patient's age, gender, type of snake (if known), severity of envenomation at initial presentation, coagulation test results, whether antivenom was administered, and whether the patient was admitted.

Results: Over an approximately 8-year period, 131 snakebite cases presented that met the inclusion criteria, of which 35 (26.7%) had some type of coagulation marker abnormality. Limiting coagulation testing to patients suffering severe envenomation or rattlesnake envenomation would have resulted in failure to identify 89% or 77%, respectively, of the 35 patients who were found to have at least 1 abnormal coagulation marker.

Conclusion: Our study failed to identify a subset of patients that could be defined as low risk or for whom coagulation marker testing could be foregone. This study suggests that coagulation tests should be routinely performed on all patients presenting to the ED with complaints of envenomation by copperheads, moccasins, or rattlesnakes. Further clarification of when coagulation markers are indicated may require a prospective study that standardizes snake identification and the timing of coagulation marker testing. [West J Emerg Med. 2012;13(1):68–74.]

INTRODUCTION

More than 2,800 venomous snakebites were reported to the American Association of Poison Control Centers in 2008.[1] Venomous snakes in the Southeastern United States include rattlesnakes, copperheads, and water moccasins of the crotalid family, as well as coral snakes of the elapid family. A small number of bites by these poisonous snakes are termed dry, when little or no venom is actually injected and symptoms of envenomation do not develop. Envenomation is generally defined as occurrence of a snakebite plus evidence of tissue damage and can result in a spectrum of clinical symptoms and laboratory abnormalities from mild, local tissue injury to systemic illness, including hypotension, neuromuscular dysfunction, and coagulopathy.[2] For a known envenomation, standard management includes advanced life support, if indicated, immobilization of the affected limb, local wound care, tetanus immunization booster, and analgesia. Patients are usually observed in the emergency department (ED) setting for 6 to 8 hours. Antivenom (CroFab by Protherics Inc, Brentwood, Tennessee) is typically given for progressive injury, with

progression being defined as a worsening of local tissue injury, systemic manifestations, or coagulation abnormalities by laboratory testing.[2]

No clear guidelines exist for ordering coagulation markers in patients with minimal or moderate envenomation, nor in those who do not receive antivenom. Many ED physicians routinely order coagulation markers on all patients with snakebites, regardless of type of snake or severity of envenomation. The costs of platelet counts, prothrombin times (PT), activated partial thromboplastin times (aPTT), and fibrinogen concentrations are significant and contribute to the expense of the management of these patients. Further costs may also be incurred simply by keeping the patient in the ED longer than necessary. In this study, we sought to determine whether coagulation markers are indicated for all snakebite patients in our region or whether we could limit the practice to ordering these tests on only those patients suffering severe envenomation, rattlesnake envenomation, or both.

METHODS

A retrospective chart review was conducted for all cases of snakebite presenting to a university medical center adult ED from April 1998 to June 2006. Prior to chart review, 1 abstractor was trained by the principal investigator on the data collection process. The abstractor was not blinded to the study's hypothesis. Inclusion criteria were age greater than 15 years, documented historical and clinical evidence of snakebite, and any of 4 coagulation markers recorded. Exclusion criteria were a known preexisting coagulopathy or hypercoagulable state, ED presentation delayed more than 6 hours, charts with insufficient data to determine the severity of envenomation, and charts with no coagulation markers recorded. Data was collected from an electronic medical record system. Data not included in the electronic record was reviewed in paper charts to gather remaining data variables. Case information used in our study included the ED physicians' notes, nurses' drug administration notes, and laboratory values.

Each chart was abstracted using a standardized data collection form for age, gender, type of snake, if known, severity of envenomation at initial presentation, coagulation test results (platelet count, PT, aPTT, and fibrinogen concentration), whether antivenom was administered, and whether the patient was admitted. In cases where the snake was not identified, it was recorded as unknown. Severity of envenomation at the time of presentation was taken directly from the physicians' notes, if documented. If not explicitly recorded by the ED physician, physical examination and laboratory data were used to classify the envenomation as minimal, moderate, or severe using the severity scoring guidelines published by Gold et al in 2002.[2] The severity scoring guidelines are detailed in Table 1. When a patient received antivenom at another hospital prior to transfer to the ED, these vials were included in the total number recorded. The hospital laboratory's standard ranges were used to determine whether coagulation markers were normal or abnormal. In cases where a patient had serial coagulation markers documented, the most abnormal measurement for each coagulation marker was used.

Table 1. Guidelines for assessing the severity of North American pit viper envenomations.*

Type of signs or symptoms	Severity of envenomation		
	Minimal	Moderate	Severe
Local	Swelling, erythema, or ecchymosis confined to the site of the bite	Progression of swelling, erythema, or ecchymosis beyond the site of the bite	Rapid swelling, erythema, or ecchymosis involving the entire body part
Systemic	No systemic signs or symptoms	Non-life threatening signs and symptoms (nausea, vomiting, perioral paresthesias, myokymia, and mild hypotension)	Markedly severe signs and symptoms (hypotension [systolic blood pressure <80 mm Hg], altered sensorium, tachycardia, tachypnea, and respiratory distress)
Coagulation	No coagulation abnormalities or other important laboratory abnormalities	Mildly abnormal coagulation profile without clinically significant bleeding; mild abnormalities on other laboratory tests	Markedly abnormal coagulation profile with evidence of bleeding or threat of spontaneous hemorrhage (unmeasurable INR, APTT, and fibrinogen; severe thrombocytopenia with platelet count <20,000 per mm^3); results of other laboratory tests may be severely abnormal

INR, international normalized ratio; *APTT*, activated partial-thromboplastin time.
* The ultimate grade of severity of any envenomation is determined on the basis of the most severe sign, symptom, or laboratory abnormality (eg systolic blood pressure <70 mm Hg in the absence of local swelling should be graded as a severe envenomation).

Data were analyzed using SPSS version 18.0 (Chicago, Illinois). Chi-square tests were used to test for associations between nominal variables. Mann-Whitney U and Kruskal-Wallis H tests were used to test for differences in mean rank of the dependent variables when there were 2 and more than 2 levels, respectively, of the independent variable. Logistic regression was also used to test the relationship between various risk factors and the presence or absence of an abnormal coagulation marker. The criterion for statistical significance was $P < 0.05$.

RESULTS

Over the approximately 8-year period, 132 snakebite cases presenting to the ED met the inclusion criteria. There was only 1 patient bitten by a coral snake, and this patient was excluded from all subsequent analyses. The study sample of 131 included 87 (66.4%) men and 44 (33.6%) women. The mean age was 43.3 years (range 16–90). Forty-nine patients (37.4%) were bitten by copperheads, 29 (22.1%) by moccasins, 17 (13.0%) by rattlesnakes, and 36 patients (27.5%) could not identify the snake. There were 37 (28.2%) minimal, 86 (65.6%) moderate, and 8 (6.1%) severe envenomations. Seventy-two patients (55.0%) received antivenom, 57 (43.5%) did not receive antivenom, and we were unable to determine if 2 (1.5%) patients received antivenom. Among those patients administered antivenom, a median of 10 vials was used in the course of their treatment. Thirty-four patients (26.0%) were admitted to the hospital and the remainder discharged following ED observation and treatment. During routine laboratory testing, some type of coagulation marker abnormality was identified in 35 (26.7%) of the 131 snakebite patients. Seventeen (13.8%) had an abnormal PT, 17 (13.9%) had an abnormal aPTT, 8 (6.2%) had thrombocytopenia, and 5 (13.2%) had abnormal fibrinogen concentrations. The range of abnormalities is shown in Table 2. In the group of 35 patients with a coagulation marker abnormality, 89% were classified as having a mild or moderate envenomation, and 77% did not have a rattlesnake envenomation. Only 1 of the 131 patients in our study had documented bleeding in the ED. This patient suffered a severe rattlesnake envenomation and was noted to have hematemesis while in the ED.

There were statistically significant associations between the identified type of snake and PT ($P = 0.015$), aPTT ($P = 0.043$), and fibrinogen ($P = 0.028$) abnormalities. Rattlesnake envenomation was associated with the greatest rate of coagulation abnormalities for each marker. Among patients envenomated by a rattlesnake, approximately 35%, 35%, and 40% had abnormal PT, aPTT, and fibrinogen concentrations, respectively. These percentages were more than double of those found for other snake types. There was also a significant association between the type of snake identified and the systemic symptoms experienced by the patient ($P = 0.035$). The percentage of rattlesnake envenomated patients with systemic symptomatology (35%) was twice the combined percentages experienced by patients envenomated by other snake types. There was no significant association found between the snake type and the frequency of observed thrombocytopenia (Table 3).

There was a significant association between the graded severity of envenomation and a laboratory finding of a PT abnormality ($P = 0.021$) for patients with moderate and severe envenomations being more likely to have a PT abnormality. There was also an association between the patient's systemic symptomatology and abnormalities found on some of the coagulation makers (PT [$P = 0.063$), aPTT [$P = 0.066$], platelet [$P = 0.005$], and fibrinogen [$P = 0.001$]; Table 4). Sensitivity, specificity, and positive and negative values of each risk factor are shown in Table 5. The association between administration of antivenom and a finding of a PT abnormality was significant ($P = 0.016$), However, the association between administration of antivenom and a finding of a fibrinogen abnormality was not significant ($P = 0.096$; Table 4). Patients with abnormal fibrinogen concentrations tended to receive more vials of antivenom as compared to patients with normal concentrations, though this difference was not found to be statistically significant ($P = 0.057$; Table 6).

There was a significant association between the type of snake identified and a decision to administer antivenom ($P = 0.018$). A total of 76% of patients with a rattlesnake envenomation and 72% of patients with a moccasin envenomation received antivenom treatment. Only 49% of patients bitten by a copperhead and 41% of patients envenomated by an unknown snake type received antivenom (Table 3). Among patients who received antivenom, there was a significant difference in the number of vials administrated during the course of treatment when considering the specific snake types identified ($P = 0.002$) with rattlesnake victims receiving twice the number of vials as compared to patients bitten by other snake types. Patients with abnormal PTT tests also received more vials of antivenom ($P = 0.015$) than patients with other laboratory findings. We failed to find a significant difference in the number of vials administered and observed PT or platelet abnormalities (Table 6).

We performed an analysis using at least 1 abnormal coagulation marker as the outcome variable. Rattlesnake bite (+/−) was significantly associated with an abnormality ($P = 0.04$), and systemic symptoms were marginally associated with an abnormality ($P = 0.068$). Forty-seven percent of rattlesnake patients had a coagulation marker abnormality versus 23.7% of nonrattlesnake patients, relative risk was 1.99 (95% confidence interval [CI], 1.09–3.63). Severity and individual snakes (copperhead, moccasin, or unknown) failed to reach statistical significance. We then performed a binary logistic regression using at least 1 coagulation marker as the response variable. Severity (severe versus not severe), systemic symptoms (+/−), and rattlesnake bite (+/−) were used as predictors. The P value associated with severity was 0.974, and severity was dropped

Table 2. Range of laboratory abnormalities.

Coagulation markers	Normal range	Abnormal values of snakebites in patients	Abnormal labs (%)
PLT	130–400	90.0	8/128 (6.30)
		96.0	
		108.0	
		113.0	
		114.0	
		450.0	
		453.0	
		548.0	
PT	9.3–12.5 s	12.6	17/122 (13.90)
		13.6	
		13.9	
		14.0	
		14.0	
		14.1	
		14.3	
		14.5	
		14.8	
		15.9	
		17.1	
		17.8	
		21.3	
		60.0	
		60.0	
		100.0	
		100.0	
aPTT	23.9–33.1 s	34.6	17/122 (13.90)
		34.7	
		34.9	
		35.0	
		35.5	
		35.6	
		35.6	
		36.2	
		36.4	
		37.0	
		37.7	
		39.1	
		39.8	
		43.0	
		57.9	
		100.0	
		100.0	
Fibrinogen	150–400	37.0	5/38 (13.00)
		50.0	
		50.0	
		135.0	
		494.0	

PLT, platelet; *PT*, prothrombin time; *aPTT*, activated partial thromboplastin times.

from the model. In the subsequent model, we used systemic symptoms and rattlesnake plus an interaction term as predictors. The interaction was found to be nonsignificant ($P = 0.093$). Main effects for both systemic symptoms and rattlesnake bites were significant ($P = 0.044$ and $P = 0.035$, respectively). Patients with systemic symptoms had a 13-fold (odds ratio [OR]) increase in the odds of an abnormal marker (OR = 13.33; 95% CI, 1.069–166.37). A rattlesnake bite was also associated with 13-fold increase in odds of an abnormal marker, (95% CI, 1.207–156.64). Finally, we performed a logistic regression with a dichotomous predictor representing those positive for both systemic symptoms and rattlesnake bite and those not positive for both. This dichotomy was a significant predictor ($P = 0.013$), and being positive for both systemic symptoms and rattlesnake bite was associated with increased odds of an abnormal marker of 15.8 (95% CI, 1.779–140.89). Finally, in this patient population, the positive and negative predictive values for this dichotomy were 83.3% and 76.0%, respectively (Table 5).

DISCUSSION

While it is common practice to order coagulation studies on patients with severe snakebite envenomations, the role of these tests for patients with mild or moderate envenomation is less certain. In this study, we attempted to determine whether coagulation markers are critical in the evaluation of all snakebite patients in our region or whether we could limit the practice to those patients suffering severe envenomation, rattlesnake envenomation, or both. The results of this study indicate that limiting such laboratory studies in this way could result in a failure to identify a large proportion of patients with abnormal coagulation markers. Restricting coagulation marker testing to patients suffering severe envenomation or rattlesnake envenomation would have resulted in our missing coagulation marker abnormalities in 89% or 77% patients, respectively. Restricting coagulation testing to patients with both a rattlesnake bite and systemic symptoms would have resulted in missing 86% of patients with coagulation marker abnormalities.

Table 3. Association between snake type and laboratory abnormalities and antivenom use.

	Copperhead	Moccasin	Rattlesnake	Unknown	n	*P* value
PT abnormality (%)	4.4	18.5	35.3	11.8	123	0.015
aPTT abnormality (%)	13.6	7.4	35.3	8.8	122	0.043
Platelet abnormality (%)	6.3	3.4	6.3	8.3	129	0.833
Fibrinogen abnormality (%)	0.0	10.0	40.0	0.0	38	0.028
Systemic symptoms (%)	6.1	17.2	35.3	19.4	131	0.035
Antivenom use (%)	49.0	72.4	76.5	41.2	129	0.018

PT, prothrombin time; *aPTT*, activated partial thromboplastin time.

Table 4. Association between coagulation markers and envenomation severity, systemic symptoms, and antivenom use.*

	PT+ (%)	PTT+ (%)	Platelets+ (%)	Fibrinogen+ (%)
Envenomation severity				
Minimal	1/35 (2.9)	4/35 (11.4)	2/36 (5.5)	0/6 (0.0)
Moderate	13/80 (16.2)	10/79 (12.6)	5/86 (5.8)	3/26 (11.5)
Severe	3/8 (37.5)	3/8 (37.5)	1/7 (14.3)	2/6 (33.3)
P	0.021	0.136	0.659	0.212
Systemic symptoms				
No	12/105 (11.4)	12/104 (11.5)	4/109 (3.7)	1/30 (3.3)
Yes	5/18 (27.7)	5/18 (27.7)	4/20 (20.0)	4/8 (50.0)
P	0.063	0.066	0.005	0.001
Antivenom use				
No	3/54 (5.5)	7/54 (12.9)	2/56 (3.5)	0/12 (0.0)
Yes	14/67 (20.9)	9/66 (13.6)	6/71 (8.4)	5/25 (20.0)
P	0.016	0.914	0.261	0.096

PT, prothrombin time; *PTT*, partial thromboplastin time.
* All denominators represent the row total for the respective coagulation marker, eg of the 35 patients with minimal envenomation severity and a PT test, 1 had a positive PT test.

Table 5. Sensitivity, specificity, and positive and negative predictive values of risk factors for predicting at least 1 coagulation abnormality. Values are %, with 95% confidence interval in parentheses.

Risk factor	Sensitivity	Specificity	Predictive value +	Predictive value −
Copperhead	31.4 (17.7–47.7)	60.4 (50.5–69.8)	22.4 (12.4–35.4)	70.7 (60.3–79.8)
Moccasin	17.1 (7.2–31.7)	76.0 (66.9–83.8)	20.7 (8.8–37.5)	71.6 (62.4–79.7)
Rattlesnake	22.9 (11.2–38.4)	90.6 (83.7–95.4)	47.1 (25.0–69.9)	76.3 (68.0–83.5)
Unknown snake type	28.6 (15.5–44.7)	72.9 (63.5–81.1)	27.8 (15.0–43.6)	73.7 (64.3–81.8)
Envenomation severe versus not severe	11.4 (3.7–24.6)	95.8 (90.6–98.7)	50.0 (19.1–80.9)	74.8 (66.7–81.9)
Systemic symptoms yes versus no	25.7 (12.5–43.3)	87.5 (79.2–93.4)	42.9 (21.8–65.9)	76.4 (67.3–83.9)
Systemic symptoms and rattlesnake	14.3 (4.8–30.2)	99.0 (94.3–99.9)	83.3 (35.8–99.5)	76.0 (67.5–83.1)

It is not surprising that patients bitten by rattlesnakes were more likely to have abnormal coagulation studies and greater systemic symptomatology as compared to patients bitten by other snakes. Also, it was not unexpected that patients with severe envenomations, and in which a clinical decision to use antivenom was made, were more likely to have coagulation marker abnormalities. Most clinicians who routinely treat snakebites are aware of the importance of performing these studies in these subsets of snakebite victims. However, in our experience, this practice has become a routine part of the evaluation and management of virtually all snakebite cases with limited objective evidence of necessity or benefit. A

Table 6. Median number of antivenom vials administered.

Snake type		*P* value 0.002
Copperhead	11 (n = 24)	
Moccasin	10 (n = 21)	
Rattlesnake	17.5 (n = 13)	
Unknown	6 (n = 13)	
Coagulation marker		
PT + versus –	10 (n = 14) versus 10 (n = 52)	0.726
PTT + versus –	15 (n = 9) versus 10 (n = 56)	0.015
Platelet + versus –	11 (n = 6) versus 10 (n = 64)	0.220
Fibrinogen + versus –	15 (n = 5) versus 10 (n = 19)	0.057

PT, prothrombin time; *PTT*, partial thromboplastin time.

differentiation of a low-acuity patient subset in which this testing would not be required could potentially result in better resource utilization. The most interesting result of this study was the discovery that coagulation abnormalities were not confined to patients with severe envenomation, nor to patients with rattlesnake bites. Coagulation abnormalities were detected in 24% of patients bitten by copperheads and 15% of patients bitten by moccasins. Abnormalities were also detected in nearly one third of patients with moderate envenomation. Furthermore, 19% of patients who could not identify the snakes had some laboratory abnormality. These findings suggest that routine coagulation marker testing in nearly all ED patients with snakebites may be an important part of their clinical monitoring and management.

LIMITATIONS

There are several reasons that could explain our failure to identify a subset of patients that do not require coagulation marker testing. In general, however, the limitations of our study probably resulted in an underestimation of the prevalence of coagulation marker abnormalities in the study population. Difficulties involved in accurately identifying a snake type at the time of a bite event are self evident. Unless documented as unknown, we assumed that patients correctly identified the snakes that bit them. Ninety-five out of 131 patients in our study reported being bitten by venomous snakes. This may be partially accounted for by selection bias; many people bitten by nonvenomous snakes presumably identify them correctly and do not seek medical attention. However, the majority of snakes in our region are nonvenomous, and it is possible that some patients in our study incorrectly identified the snakes that bit them as venomous.

In order to stratify our patients by severity of envenomation, we used the severity scoring guidelines published by Gold et al in 2002 (Table 1). These guidelines rely in part on subjective measurements, such as degree of edema, erythema, and ecchymosis. In addition, they are designed only for patients with known envenomation.[2] Nevertheless, we chose to use this system, as no completely objective tool for clinical stratification currently exists.

Envenomation is also a dynamic process. It is often difficult to ascertain whether a bite represents true envenomation on initial ED presentation, and estimations of dry bites vary widely.[2,3] The amount of venom injected varies by age, condition, and species of the snake, size of the victim, and many other factors. The clinical management of snakebites typically requires a longitudinal monitoring of the patient's response to the envenomation, which many also vary greatly according to the location of the bite and the inherent physiology of the affected individual. As snake venom is a complex mixture of enzymes, the clinical responses to envenomation are myriad.[4] The venom of many snake species contains several components that can induce hemorrhage, including fibrinolytics, platelet aggregation inhibitors, and hemorrhagins. All crotalid snake venoms are theoretically capable of causing some degree of coagulopathy.[5] Copperhead venom contains a protein C activator, water moccasins carry beta fibrinogenases, and timber rattlesnakes carry serine proteases.[4] Case reports even exist of presumed nonvenomous snakes causing coagulopathy.[6] This biologic complexity and the evolving nature of snakebite signs and symptoms may make it difficult to classify an evenomation as severe early in the course of clinical management. When considering all these factors, it is perhaps not surprising to find such a prevalence of laboratory abnormalities among the patients in the current study.

Additionally, not all patients in our study had all 4 coagulation markers (PT, aPTT, platelets, and fibrinogen concentration) drawn. Timing of patient blood draws also varied among our patients. In most cases, our patients had their coagulation markers drawn immediately after being placed in an ED examination room. However, the delay from the time of the bite to presentation in the ED varied. Asymptomatic coagulopathy may, therefore, have occurred in some patients following their initial blood draw and would not have been detected in our study. Several case reports and studies exist that document patients with delayed coagulopathy following envenomation.[7,8,9]

Patient charts were not abstracted for hemoglobin or hematocrit, so we may have failed to detect occult bleeding during ED observation. Only 1 patient in 131 had documented bleeding in the ED. Our study also did not address whether knowledge of abnormal coagulation markers changed management or impacted patient outcomes.

CONCLUSION

In this study, we sought to determine whether all patients presenting to an ED in our region should have coagulation markers routinely drawn as part of their management. We hypothesized that this practice could be limited to ordering the tests only for patients suffering severe envenomation,

rattlesnake envenomation, or both. However, our study failed to identify a group of patients that could be defined as low risk or for whom coagulation marker testing could be foregone. Instead, this study suggests that coagulation tests should be routinely performed on all patients presenting to the ED with complaints of envenomation by copperheads, moccasins, or rattlesnakes. The study had a number of limitations, including snake identification, severity of envenomation stratification, and collection of coagulation markers. Further guidance for ED physicians might be provided by a prospective, multicenter trial, enrolling only patients for whom the snake could be positively identified and in which the same coagulation markers were drawn at a standardized time.

Address for Correspondence: Richard L. Summers, MD, University of Mississippi Medical Center, Department of Emergency Medicine, 2500 N State St, Jackson, MI 39216. E-mail: rsummers@umc.edu.

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Coagulopathy: The Most Important Thing We Still Don't Know About Snakebite

Eric J. Lavonas, MD Rocky Mountain Poison and Drug Center–Denver Health, Denver, Colorado

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In this issue of *Western Journal of Emergency Medicine*, Moriarty et al present a thoughtfully analyzed case series of patients with crotaline snake envenomation.[1] They seek to answer a common and important question: Can we define a group of pit viper victims who are at low risk for hematologic venom effects (fibrinogen degradation and/or platelet destruction) and spare these patients the time and expense of serial laboratory testing? Because their institution in Mississippi sees a large number of rattlesnake and *Agkistrodon* victims, Moriarty and colleagues are well positioned to address this question.

The answer, notwithstanding the limitations of this paper, is no.

We know that bites inflicted by *Agkistrodon* snakes, and particularly copperheads, are generally less severe than rattlesnake bites. Previous studies suggest that only 10% to 20% of copperhead victims develop coagulopathy or thrombocytopenia at any time in their clinical course, compared with 50% or more of rattlesnake victims.[2,3] Physicians treating copperhead victims often order relatively few blood tests; in many cases, this can be considered standard care.[4] Similarly, it has long been observed that early hematologic venom effects strongly predict the late hematologic effects.[5] As a result, it seems like patients who have completely normal early labs are at low risk for developing coagulopathy and thrombocytopenia later. More recent studies have refuted this observation, at least in rattlesnake envenomation.[3] It is not clear who can forgo testing.

These are not academic arguments. Snakebite is a blue-collar disease. Medical bills are the leading cause of personal bankruptcy; despite the recent housing crisis, this proportion is actually increasing.[6] The costs of prolonged hospitalization and serial laboratory testing, transportation, and missed work are a real concern for our patients. In low-risk situations, practicing medicine with no regard for cost is disrespectful and harmful.

In their very nicely conducted multivariate analysis, Moriarity et al seem to have disproven the notion that any patient is safe from the risk of hematologic venom effects. Neither copperhead victim status nor low initial severity nor any other combination of initial factors they evaluated proved sufficiently sensitive to define a group of patients at low risk of hematologic venom effects. This is particularly striking because their observations ended at the time of hospital discharge. Had results of postdischarge lab testing been available for their study, Moriarity et al's results would have been even more convincing.

A crucial piece of this puzzle remains missing. Nearly 13 years after the first focused evaluation of late hematologic venom effects in the Fab-antivenom era, our understanding of the relationship between abnormal laboratory values and the risk of bleeding is weak and driven by anecdote.[5] To date, 4 case reports of late-onset, medically significant bleeding have been published.[7–10] At least 1 additional case has been litigated to conclusion.[11] Though very worrisome, these cases must be considered in context. Data from the National Electronic Injury Surveillance System–All Injuries Program show that more than 8,000 patients are treated in US emergency departments for crotaline snakebite each year.[12] In a multicenter case series of crotaline victims, only 1 of 209 antivenom-treated patients was reported to have delayed onset of medically significant bleeding.[10] None of the 42 patients in the Fab antivenom premarketing trials had major bleeding.[13,14] Two large cohort studies from Phoenix found no cases of delayed bleeding among 94 patients, most of whom had coagulopathy, thrombocytopenia, or both.[3,15] None of Moriarity et al's 131 patients experienced bleeding, and most of the laboratory abnormalities they evaluated posed only a minimal risk of bleeding.[1]

How many snakebite patients must we test or treat with additional antivenom to prevent 1 case of serious bleeding? Fortunately (for our patients), these cases are so infrequent that the issue is nearly impossible to study. As a result, there is currently no evidence-supported standard of care for surveillance laboratory testing. Substantial controversy exists

about what we should do with clinically occult hematologic venom effects once we find them. Moriarity et al tried to help us out of this morass, at least for putative low-risk patients, and found that even in this group there are no easy answers.

Address for Correspondence: Eric J. Lavonas, MD, Rocky Mountain Poison and Drug Center–Denver Health, 777 Bannock St, Denver, CO 80204. E-mail: eric.lavonas@rmpdc.org.

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Massive Atropine Eye Drop Ingestion Treated with High-Dose Physostigmine to Avoid Intubation

Samuel J. Stellpflug, MD*†
Jon B. Cole, MD*†
Brian A. Isaacson, MD‡
Christian P. Lintner, DBAT†
Elisabeth F. Bilden, MD†

* Regions Hospital, Department of Emergency Medicine, Toxicology Education and Clinical Service, St Paul, Minnesota
† Hennepin Regional Poison Center, Minneapolis, Minnesota
‡ Fairview Ridges Medical Center, Department of Emergency Medicine, Burnsville, Minnesota

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Case: A 34-year-old male presented after ingesting 150 mg of atropine. He had altered mental status, sinus tachycardia, dry mucosa, flushed skin, and hyperthermia. Sequential doses of physostigmine, totaling 14 mg, were successful in reversing antimuscarinic toxicity and prevented the need to perform airway control with endotracheal intubation. At completion of treatment, heart rate and mental status had improved, and intubation was never performed.

Discussion: Atropine causes anticholinergic toxicity; physostigmine reverses this by inhibiting acetylcholinesterase. Atropine eye drop ingestions are rare. The 14 mg of physostigmine administered is much higher than typical dosing. It is likely the physostigmine prevented intubation. Atropine eye drops can be dangerous, and physostigmine should be considered in treatment. [West J Emerg Med. 2012;13(1):77–79.]

INTRODUCTION

Atropine, or hyoscyamine, is an alkaloid used commonly for its antimuscarinic properties.[1] It acts as a competitive antagonist of acetylcholine at muscarinic receptors. It can be administered by various routes, including the eye drop formulation of atropine sulfate, used to induce cycloplegia and mydriasis.[2] In overdose, atropine can cause tachycardia, agitation, delirium, dilated pupils, dry mucous membranes, dry skin, and hypoactive bowel sounds. These phenomena have been described even with attempted therapeutic ophthalmic use.[1,3] Ingestion of as little as a few drops of atropine in eye drop formulation can cause anticholinergic, or more specifically antimuscarinic, toxicity.[4] The antimuscarinic toxidrome results from blockade of the neurotransmitter acetylcholine at central and peripheral muscarinic receptors.[5]

Physostigmine is a carbamate that acts by reversibly inhibiting acetylcholinesterase. Unlike quaternary amine acetylcholinesterase inhibitors (such as neostigmine) that treat peripheral manifestations of the antimuscarinic toxidrome, physostigmine is a tertiary amine, and thus is able to cross the blood-brain barrier to treat both central (eg, agitation and delirium) and peripheral (eg, tachycardia) antimuscarinic manifestations.[5] The use of physostigmine began as early as the 19th century for its ability to reverse the signs and symptoms of anticholinergic poisonings. Its popularity grew in the 1960s and 1970s as a general antidote and diagnostic tool for altered mental status.[6] A case series published in 1980 illustrated 2 cases of patients who developed asystolic cardiac arrests in the context of tricyclic antidepressant overdose where treatment included physostigmine.[7] The frequency of use of this antidote declined after that report. However, recent literature has tempered some of the concern about the deleterious effects of physostigmine, and its use has again become more frequent.[6,8] The usual dose of physostigmine is 0.5 to 2 mg administered by slow intravenous (IV) push, with repeat doses administered every 15 to 40 minutes as necessary.[9] It is unusual for doses in the emergency department to exceed 2 to 4 mg.

We describe an adult male with a massive ingestion of atropine eye drops treated successfully with 11 mg IV physostigmine in the emergency department. Successful

treatment in this case is defined as improvement of altered mental status and avoidance of need for intubation.

CASE REPORT

A 34-year-old male presented to an urgent care center, where he collapsed on arrival in the triage area, per providers in that department. He stated that he had emptied a full bottle of atropine eye drop solution into a glass of water and ingested it in an attempt to harm himself. The atropine concentration was 10 mg/mL, making for a total ingestion of 150 mg. On initial presentation, he had altered mental status with waxing and waning coherence, and when awake, he was very combative. He was also tachycardic with a heart rate (HR) of 125 beats per minute. A fingerstick glucose was normal. He was given 2 mg IV lorazepam, 4 mg IV ondansetron, 50 gm oral activated charcoal, and quickly transferred to a larger local hospital for further care.

In the emergency department at the accepting facility, the patient continued to have altered mental status, varying between severe sedation and uncontrolled agitation. His HR was 150 beats per minute, blood pressure (BP) 150/90 mmHg, respiratory rate 24 breaths per minute, and oxygen saturation 95% on room air. He had flushed skin, dry oral mucosa, nonreactive mydriasis, and a rectal temperature of 100.2°F. He showed no signs of trauma and had a nonfocal neurologic examination other than the gross altered mental status. The remainder of his physical exam was unremarkable. Electrocardiogram revealed sinus tachycardia and no interval or segment abnormalities.

Due to the intermittent somnolence and uncontrolled agitation, the emergency physicians at the accepting facility were concerned for the patient's ability to protect his airway enough to maintain oxygenation and ventilation. This, in combination with the recently administered charcoal and the possibility of emesis with subsequent aspiration, was enough cause for them to move towards rapid sequence induction (RSI) and endotracheal intubation. While preparing for the intubation, a medical toxicologist was consulted by the treating physicians. In an attempt to avoid the morbidity of the RSI, intubation, sedation, and mechanical ventilation, the decision was made to administer physostigmine to reverse the anticholinergic effects of the atropine.

Over the subsequent 75 minutes, physostigmine was titrated in 1 mg increments, each given over 3 to 5 minutes to a total dose of 11 mg. There was minimal change with the initial few doses, but after the fifth dose had been administered each 1 mg dose improved the patient's mental status along with normalizing the heart rate from the tachycardia listed above. Each of these signs would slowly worsen again over the ensuing 5 to 15 minutes, necessitating another dose. At the completion of treatment in the emergency department, the patient's HR had declined into the 90s beats per minute, and BP to 125/80 mmHg. His mental status was normalizing to the point where he was relatively calm and could provide a little bit of subjective history.

Laboratory evaluation at the second hospital included a negative serum ethanol, negative acetaminophen, normal basic metabolic panel, normal complete blood count, and negative urine drugs of abuse immunoassay for amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine, opiates, and phencyclidine. A serum atropine level later returned at 240 ng/mL (this was performed using the initial blood draw at the accepting facility).

He was admitted to the intensive care unit (ICU) for further observation and treatment. During the night in the ICU, he required 3 additional 1 mg doses of physostigmine for agitation that recurred. Endotracheal intubation was never performed. The remainder of the medical portion of his hospital stay was uneventful.

DISCUSSION

Atropine ingestions are common. In 2008, poison centers received 1,040 calls regarding plants containing anticholinergic toxins and 435 calls regarding atropine or diphenoxylate containing antidiarrheal medications.[10] Ophthalmic preparation exposures are also a common poison center call with 3,481 calls occurring in 2008.[10] Despite this, a paucity of atropine eye drop ingestion cases exist in the literature.

Atropine toxicity and lethality are not predictable by dose. Fatalities have been reported with exposures of less than 100 mg, and survival has been described with doses greater than 1 g orally.[1] The amount of atropine ingested by the patient in the case presented above falls into this potentially lethal range. To put the serum level of 240 ng/mL into perspective, an adult patient with a reported 1-g ingestion of atropine had a serum level of 129 ng/mL.[11] A study of 248 cases of accidental injections with personal autoinjectors demonstrated levels of 7.5 to 69 ng/mL.[12]

Making this case more remarkable was the liberal use of physostigmine. Standard dosing of physostigmine is 0.5 to 2 mg (0.02 mg/kg in children) given intravenously over at least 5 minutes with repeat dosing as needed 15 to 40 minutes later.[9] When repeat dosing is needed, 4 mg total is usually sufficient.[9] Our case is also unique in that physostigmine was administered in more rapid succession, and a total dose of 14 mg was required to control agitation and reverse delirium. The large amount of physostigmine required to treat this patient was likely due to the massive amount of atropine ingested, confirmed by the serum level of 240 ng/mL.

This case illustrates one of the most beneficial aspects of physostigmine use: the ability to control agitation and reverse delirium, thereby reducing the need for invasive interventions. Physostigmine administration, in this case, prevented the need for intubation, thus preventing patient exposure to the potential complications of this invasive procedure. This may be understated at first glance, but the morbidity of RSI, intubation, and mechanical ventilation are serious and well established.[13,14]

It should be noted that along with potentially eliminating the need for intubation and potentially lowering the level of observation needed for a patient in the hospital, there are some concerns about the safety of the antidote itself. As mentioned earlier, much of this concern is likely unfounded and is based on a case series with questionable causality between the physostigmine and the negative outcomes.[7] The most recent look in the literature on this topic is a retrospective case series including hundreds of patients that received the antidote. There was a seizure rate of less than 1% (all were self-limited) and there were no cardiac arrhythmias.[15] The above benefits and risks of both physostigmine and intubation need to be weighed when deciding whether or not to use this treatment in this clinical scenario.

CONCLUSION

An oral overdose of atropine sulfate eye drops with severe altered mental status reversed with physostigmine is unique to the literature. Additionally, the dose of physostigmine administered was much higher than what is usually recommended or necessary. It is highly likely that the physostigmine administration prevented the potential morbidity of intubation and mechanical ventilation for the patient. Eye drops should be considered a potentially dangerous means of atropine exposure, and physostigmine should be considered in cases similar to this one with the goal of improving patient care and use of hospital resources. While the authors are not advocating a blanket use of high dose physostigmine for known antimuscarinic overdoses such as the one in this case, this treatment method may be considered if the likely benefit appears to outweigh a potential morbidity-inducing alternative.

Address for Correspondence: Samuel J. Stellpflug, MD, Regions Hospital, Department of Emergency Medicine, Toxicology Education and Clinical Service, 640 Jackson St, St Paul, MN 55101. E-mail: samuel.j.stellpflug@healthpartners.com.

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A Pain in the Wrist: Stingray Envenomation

Adele E. Tse, MD
David P. Evans, MD
Francis L. Counselman, MD, CPE
Eastern Virginia Medical School, Department of Emergency Medicine, Norfolk, Virginia

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A 43-year-old man presented to the emergency department after he was swimming in the ocean and felt a sudden sting followed by a burning pain and muscle spasms in his right hand. Physical exam was remarkable for tachycardia and the foreign body (Figure 1). Radiograph of his right wrist is also shown (Figure 2).

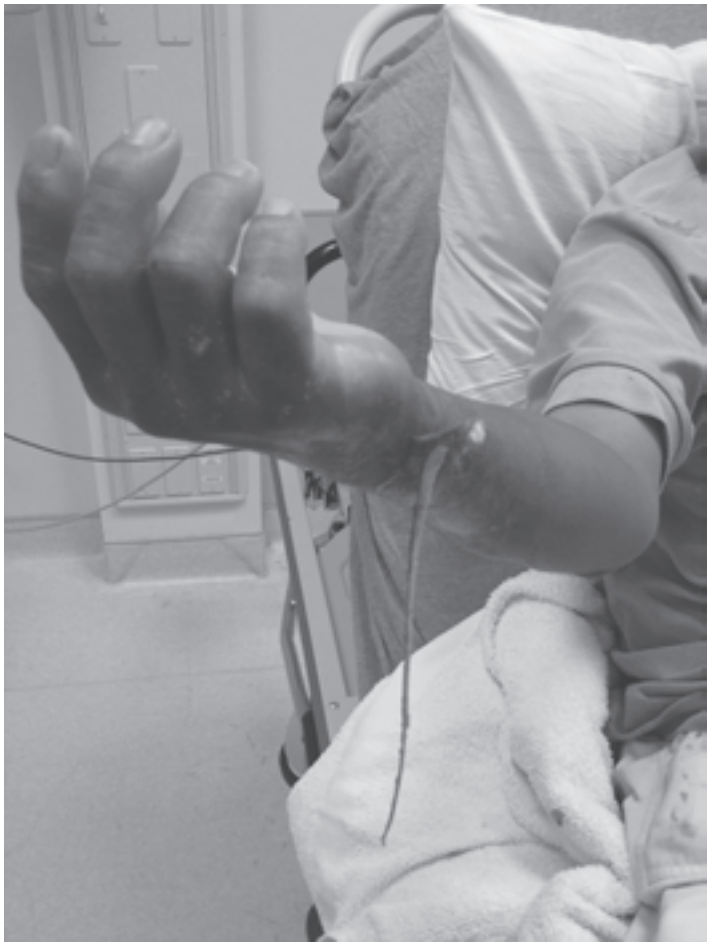

Figure 1. Patient in emergency department.

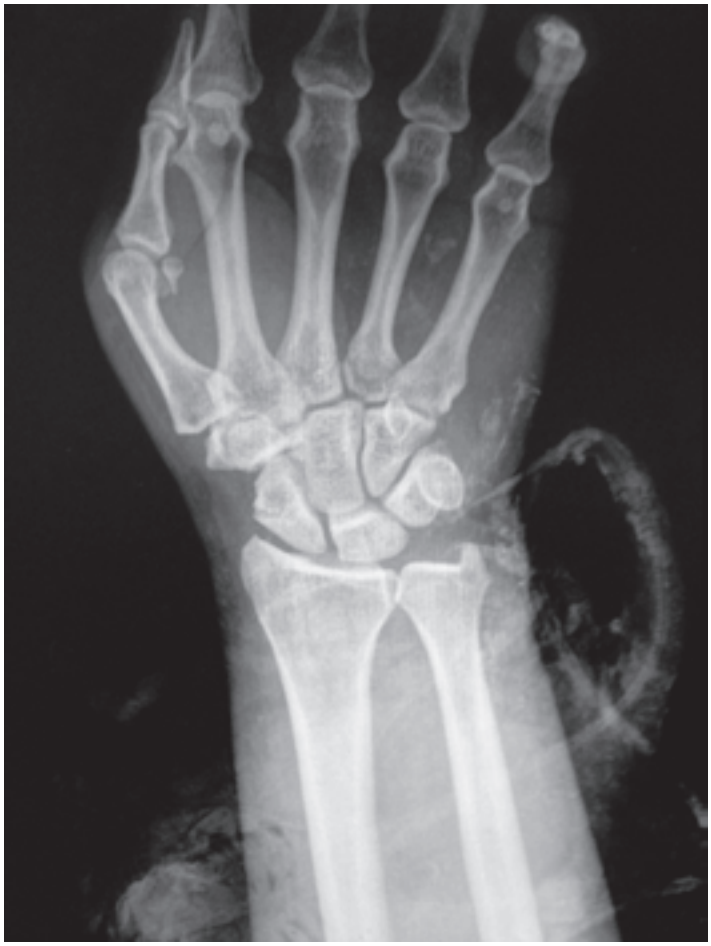

Figure 2. Radiograph of hand.

DIAGNOSIS

Stingray envenomations: Stingrays are a cartilaginous bottom-dwelling fish with a hard tail and 2 or more hard barbs, each containing a venom sac. Commonly, envenomation occurs when the victim unintentionally steps on a buried stingray and reflexes cause the ray to lash out with its tail. The venom has vasoconstrictive properties, causing possible necrosis and poor wound healing. Common presentations include local pain, muscle cramps, vomiting, and diarrhea, and rare complications include artery laceration and compartment syndrome. Since the venom is heat sensitive, immediate management includes irrigation and soaking the wound in hot water (110 to 115°F) for 30 to 90 minutes to inactivate the protein.[1] A radiograph should be obtained of the area to evaluate for embedded spines. One must be careful when removing the serrated spine. The best strategy is to open the wound with a scalpel along the nonjagged edges of the spine without disturbing the barbs. These wounds are to be closed by delayed primary closure. According to 1 study, due to the rate of infection, these patients are to be started on a quinolone and updated on tetanus.[2]

Address for Correspondence: David P. Evans, MD, Eastern Virginia Medical School, Department of Emergency Medicine, Rm 304, Raleigh Building, 600 Gresham Dr, Norfolk, VA 23507. E-mail: evansdp@me.com.

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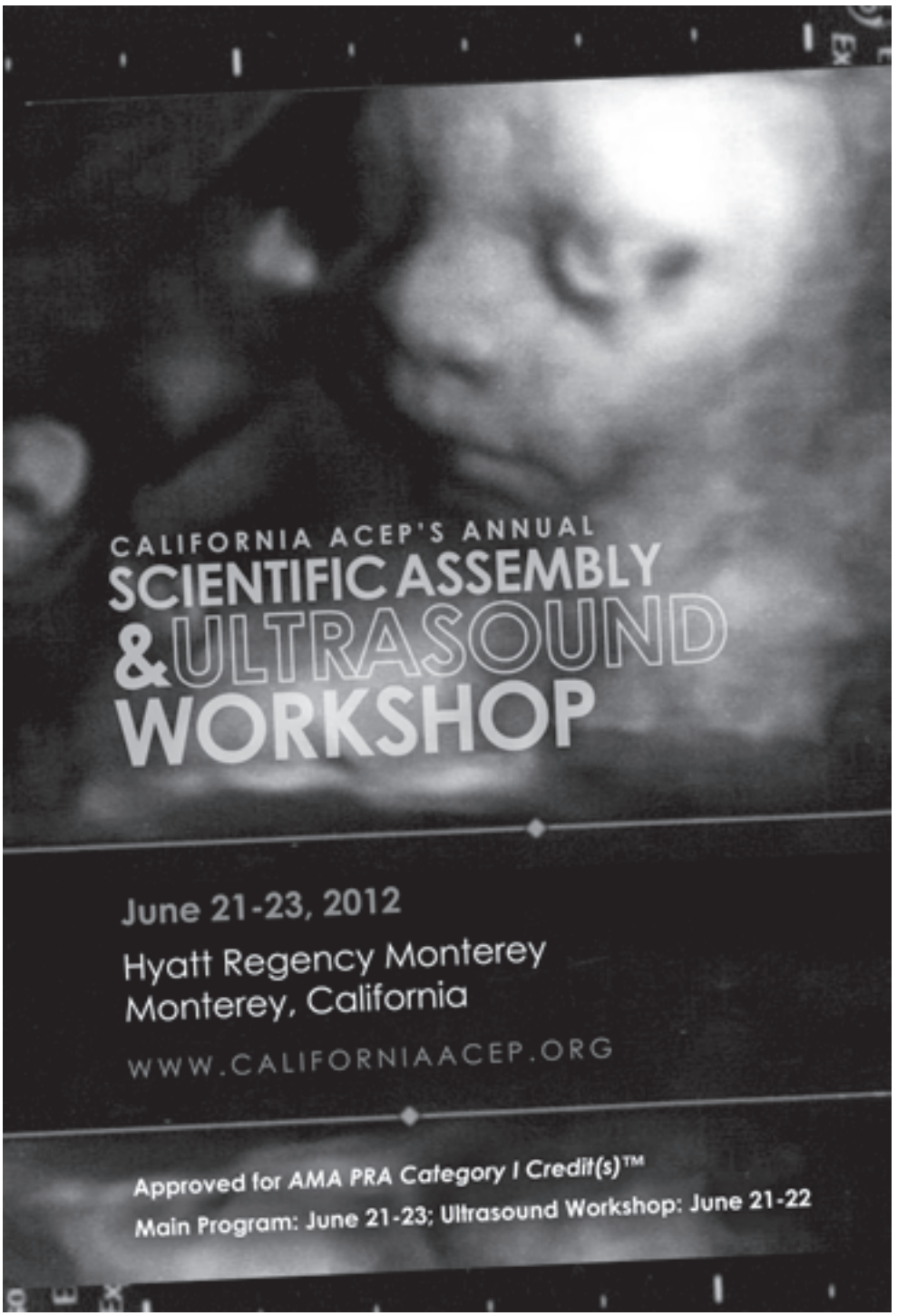
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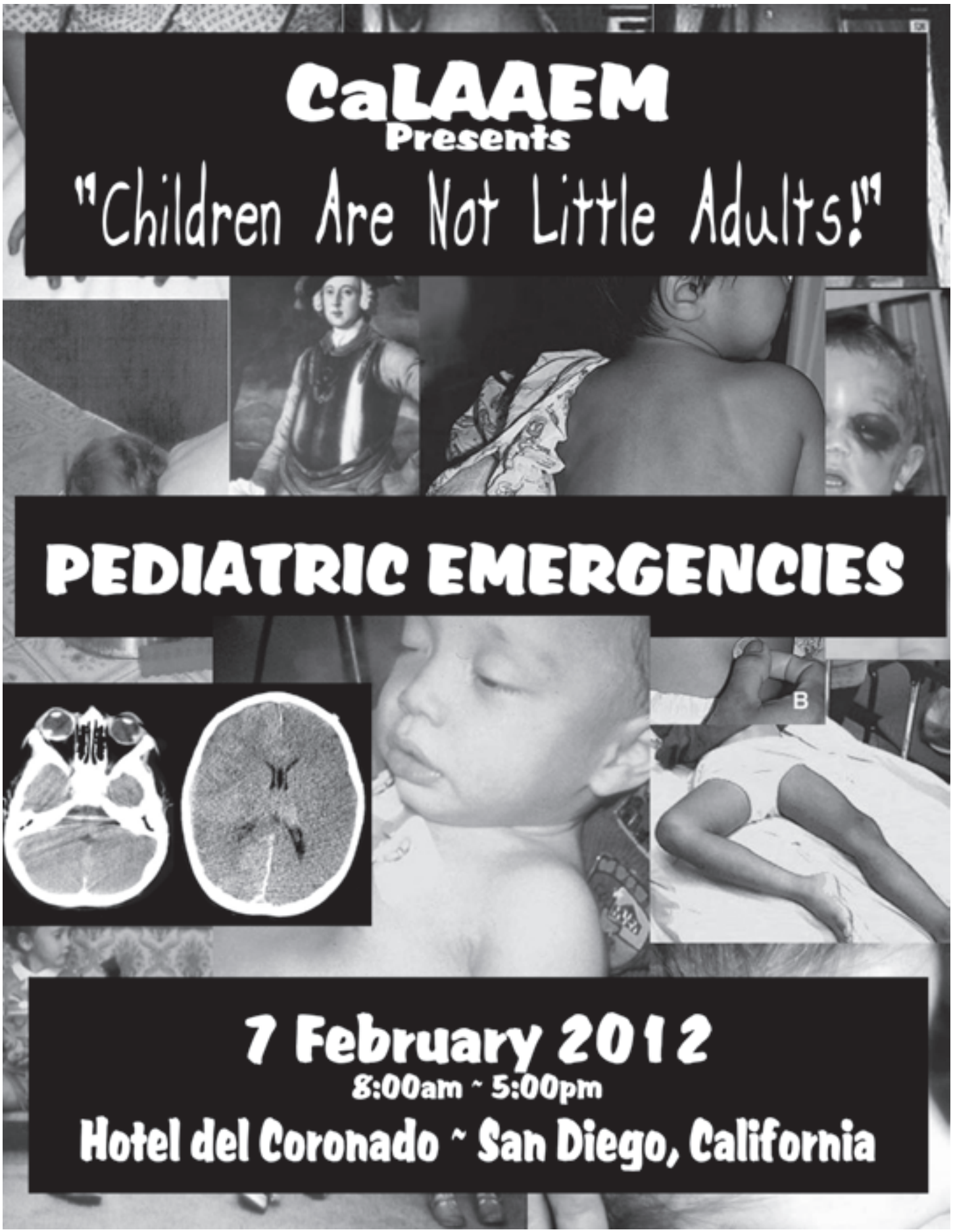
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The *Western Journal of Emergency Medicine* has received a detailed critique by Dr Christopher Greeley of the article, "Challenging the Pathophysiologic Connection between Subdural Hematoma, Retinal Hemorrhage, and Shaken Baby Syndrome" by Dr Steven Gabaeff, published in May 2011, Volume XII, Issue 2. The author's response is even more detailed. *The Journal* recognizes that these 2 authorities are diametrically opposed in their opinions, and in the interest of fair academic discourse, we are publishing both the letter to the editor and response to the editor in electronic form for those interested in this highly contentious debate.

We leave it to the reader to judge the original article, its critique, and rebuttal, on their own merits.

The Editor

DOI: 10.581/westjem.2011.9.6891

Challenging the Pathophysiologic Connection between Subdural Hematoma, Retinal Hemorrhage, and Shaken Baby Syndrome

Gabaeff SC. Challenging the Pathophysiologic Connection between Subdural Hematoma, Retinal Hemorrhage, and Shaken Baby Syndrome. *West J Emerg Med.* 2011;12(2):144–158.

To the Editor:

As having board certification in both general pediatrics and child abuse pediatrics, and having experience and training in clinical research and medical literature appraisal, I read with great interest the "Special Contribution" by Dr Steven Gabaeff.[1] I appreciate the special relationship that the author has with the *Western Journal of Emergency Medicine* as having been instrumental in the rebranding from *The California Journal of Emergency Medicine*, past president of the California chapter of the American Academy of Emergency Medicine, and a current editorial board member. Given the complex and contentious nature of the subject matter, I am impressed that it took less than 4 weeks for a meaningful peer review to occur, for recommending revisions for the author, and for receiving those revisions.

I recognize that there are a number of medical professionals who disagree with some of the accepted clinical features of abusive head trauma (AHT) (formerly referred to as "shaken baby syndrome") and I believe that critical scrutiny and lively debate of much of clinical medicine is a healthy and necessary endeavor. As a result, there exists a small cadre of professionals who have become denialists to many of the central tenets of AHT[2] and use various rhetorical techniques[3,4] to further an ideology, and not to meaningfully contribute to the field. Unfortunately, I fear the piece by Dr Gabaeff does not contribute to a substantive deconstruction of some of the basic tenets of child abuse pediatrics or further the discussion. I would like to point out some of the methodologic flaws the author makes so as to afford your readership a more accurate appreciation of this complex and often contentious field. Owing to space constraints, I cannot present a counterfactual argument for each of the presented hypotheses. I will limit my comments to highlighting certain rhetorical sleights that may mislead the reader, and provide some examples from Dr Gabaeff's text.

Throughout the article, the author uses a common technique of preceding and/or following controversial and unsupported statements with cited comments or phrases. This technique gives the appearance of cited literature support for an unsupported opinion. The first example of this is when the author discusses the work of Dr Ommaya in whiplash forces on the brain and cervical spine of monkeys. The author writes, "With current technology, these neck findings following whiplash injury would be evident as soft tissue swelling from hematoma or edema on magnetic resonance image (MRI) and computed tomography (CT) of the neck." This is placed before and after well-cited work by Dr Ommaya but is itself uncited, and in the pediatric population has been shown to be untrue.[5,6] It is this sentence that is meaningful to clinicians, but it is this sentence that is unsupported. This "citation sandwich" is a common way in which unsupported opinions are given the veil of legitimacy by their proximity to cited and supported concepts. Another example of this is when the author describes the hypothesis that shaking an infant is dangerous. The author writes, "based on analysis of the force required to cause intracranial injury and the impact of shaking on the neck, without some findings of neck injury on imaging, intracranial pathology resulting from human shaking of a previously healthy child should be seriously called into question." While this statement is uncited, it is preceded by a cited discussion of the G forces required to cause injury and followed by a cited

discussion of helmet forces, which occur during football collisions. Of note, the discussion of the forces generated in football collisions is an example of "irrelevant conclusion" (*ignoratio elenchi*). This technique is used to divert attention away from an underlying argument by introducing a tangential and irrelevant argument theme. The forces generated by the collisions of adults playing football are physiologically and biomechanically unrelated to the theory that shaking of an infant can result in retinal hemorrhages.

Another methodologic flaw the author uses is "denying the antecedent." This is a technique in which conclusions are made that are not supported by the presented evidence. The author writes, "On this basis, the consideration of intentional impact must be carefully evaluated to diagnose abuse, as it is clear that short falls in household situations are sufficient to cause not only ICT, but even death." The citation for this is a review of 75,000 falls involving playground equipment reported to the US Consumer Protection Agency, of which 18 were fatal.[7] In reading the "Methods" section of this citation, it is readily apparent that none of these were household falls and none involved children younger than 12 months. While this is an important article as support for consideration of falls as a cause of death in young children, to imply that it supports that a short household fall can kill an infant is misleading. Another example of denying the antecedent is when the author discusses the differential diagnosis of retinal hemorrhaging in infants. The author writes, "Lantz found from autopsy work on 425 eyes of the recently deceased that 17% exhibited RHs associated with a variety of diseases and conditions." The citation for this is a single case report of a 14-month old child who had a crush injury to his head. His evaluation revealed "bilateral dot and blot intraretinal haemorrhages, preretinal haemorrhages, and perimacular retinal folds." This is another important article but in no way supports the contention offered by the author. (Apparently, the author was intending to refer to Dr Lantz's 2006 American Academy of Forensic Sciences presentation[8] in which he described his experience with 111 people (16% of his total sample) with retinal hemorrhages, only 30 of whom were children. Of these 30, only 19 were younger than 1 year. Dr Lantz reported that 15 of these infants had retinal hemorrhages, which were from nonabusive causes.[9] These data have not been published in peer-reviewed literature.

Another example of denying the antecedent in this piece is when the author discusses apparent life-threatening events (ALTE). The author hypothesizes that the symptoms associated with an ALTE ("seizures, decreased muscle tone [limpness], vomiting, failure to thrive, hydrocephalus, altered level of consciousness [LOC], color changes from hypoxic episodes, conventional or dysphagic choking, abnormal breathing patterns, and apnea") could be the manifestations of a chronic subdural hematoma. Ironically, to support this contention, the author cites a 1968 cohort (pre–computed tomography [CT] technology) of 116 infants with "subdural effusions or hematomas" described by Till.[10] Of these 116 infants, nearly half had retinal hemorrhages, a number that "would have been undoubtedly higher if more time had been spent examining the fundi of these babies."[10] Till reports for the subdural collections "no satisfactory explanation in many cases, although trauma is an important factor in the majority."[10] It appears that the citation used to support Dr Gabaeff's contention that the ALTE-like symptoms of a chronic subdural hematoma (SDH) can be spontaneous is that of a cohort of children many of whom likely had been abused.

Another subtle rhetorical technique used is the "straw man" argument. This is the most widely known rhetorical technique and involves constructing an opposing point of view in a manner that makes it seem unbelievable, and thus easily discountable. The author performs this when he refers to the large number of accidental falls that occur each day, and that "it is illogical to reflexively assume a different, sinister act has occurred in patients who are found to have SDH after an accidental fall. Rather, we should recognize that a very small subset of all accidental falls can and do result in serious brain injury. With a large denominator of accidental falls, the serious brain injuries can and do result from innocent, accidental mechanisms, and each of these cases most likely prompts a medical encounter." This description makes the "pediatric child abuse specialist" seem irrational and thus unbelievable. In using this rhetorical sleight, one does not have to discuss the data that fatal falls from any height in children are exceedingly rare (55 per year in children younger than 5 years[11]) nor outline the detailed protocols that hospitals and professional organizations[12,13] have regarding the meticulous evaluation of suspect abuse. The straw man argument technique is intended to simply make the opposite position seem unfounded.

Lastly, the author also uses "converse fallacy of hasty generalization." This is a technique in which a very specific premise is constructed and the conclusions are (mis)applied by generalization. This is a very common technique of rhetorical argument in which a single case report or instance is used to dispel an entire theory. The author uses this technique when he discusses the article by Rooks et al.[14] This is a study of neuroimaging of newborn infants. Of the 101 infants undergoing neuroimaging, 1 (1%) had "a new frontal SDH on the 2-week MR imaging follow-up examination." Rooks et al note that this neonate "had bilateral occipital and posterior fossa SDH on initial imaging at birth, confirmed on the 7-day follow-up MR imaging. He was also noted to have extra-axial collections of infancy. At 26-days postnatal age, the MR imaging demonstrated left frontal subdural collections that did not conform to CSF signal intensity." This single case, that may have had something unique about it, is used to support a recommendation for a screening magnetic resonance imaging on all infants with "subtle behavioral abnormalities to prevent later accusations of abuse if complications arise." (Of note, this infant was not described by Rooks et al as having hydrocephalus as Dr Gabaeff contends.)

A subtle variant of the converse fallacy of hasty generalization is to simply not provide literature support for a broad generalization. An example of this is when the author discusses the presence of retinal hemorrhages. He writes, "The American Academy of Ophthalmology has endorsed and taught the current corps of ophthalmologists that RH, schisis, retinal folds and vitreous hemorrhage are identified with intentional abuse when in fact these findings are more likely the consequence of metabolic catastrophe within the eye itself and unrelated to shaking forces as discussed above." This sentence is uncited and nowhere in the article does the author refer to data on metabolic diseases and retinal findings. While case reports are quite rare of infants or children with Menke disease, von Willebrand disease, leukemia, and glutaria aciduria (to name a few) who have been noted to have retinal hemorrhages, the author's sweeping generalization is simply unsupported by clinical practice or medical literature.

In closely appraising the "Special Contribution" by Dr Gabaeff, we see a number of concerning logical fallacies and rhetorical sleights of hand. While this piece is not a systematic review and simply represents the opinion of the author, much of what is written is intended to be used in legal proceedings, and to be cited as being from a peer-reviewed publication. The distinction between a methodologically rigorous systematic review and an opinion piece will be lost on many readers (and juries). The peer-review process is seen by many uninitiated readers as "validating as true." As a sophisticated end-user of the medical literature, I am continually reminded it is ultimately up to me to critically scrutinize everything that I read and to assess the quality of methodology and data presented. Given the adversarial nature of some of the scholarship of AHT, I am very conscientious of many of the logical and rhetorical landmines readers can encounter. While it is I who ultimately assigns meaning and value to what I read, it is beholden to journals to maintain very high standard of quality and to not create artificial confusion where none exists. I fear the piece by Dr Gabaeff contributes little to the discussion and merely obfuscates the truth.

Christopher S. Greeley, MD
Associate Professor of Pediatrics
Center for Clinical Research and Evidence-Based Medicine
University of Texas Health Science Center at Houston

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In reply:

I welcome the opportunity to respond to Dr Greeley's letter to the *Western Journal of Emergency Medicine*, criticizing the journal, the editorial staff, myself, and the content of what I have written.

The legal consequences of the misdiagnosis of accidents and medical problems as abuse are dreadful. The nonevidence-based "certainty" that retinal hemorrhage (RH) and subdural hematoma (SDH) are sufficient to diagnose abuse is expressed often, early, and with conviction by virtually all board-certified child abuse pediatricians, many radiologists, and most ophthalmologists. The reliance on these nonspecific findings as pathognomonic of abuse is the rule, not the exception. All other facts and circumstances in any specific case are subservient to the 2 nonspecific finding that were challenged in my article. Using these findings to accuse caregivers of abuse is backward thinking. The findings themselves, long established as inexact on their own and in combination, have been used to speculate

about intent, mechanism, and as the basis of abuse allegations. Clinging to dogma long since exposed as unreliable and scientifically invalid, and attacking the messengers exposing the flaws in that dogma, have been the modus operandi of the child abuse establishment, in this case represented by Dr Greeley's letter.

Dr Greeley recently presented a talk entitled "A Wolf in Evidence Clothing [sic]: Denialism in Child Abuse Pediatrics"[1] and gave a presentation in 2011 at the conference on abusive head trauma (AHT) in Hershey, Pennsylvania, that was titled "Deconstructing Donohoe: The Evidence Behind the 'Lack of Evidence.'" In each case, those who disagree with the child abuse establishment are referred to as "denialists" and their integrity and professionalism is attacked to blunt the impact of their analyses. Donohoe, who I cite, and whom Dr Greely criticized, was singled out by him at a meeting of key members of that establishment precisely because Donohoe's criticism of the child abuse literature is so impactful to the current state of child abuse pediatrics.

Donohoe[2] was cited in my article, and by many others, for his valid criticism of the child abuse literature. As the readers of my article might recall, Donohoe evaluated the child abuse literature from 1966 to 1998 and found significant weaknesses, concluding that there was inadequate scientific evidence to come to "a firm conclusion on most matters pertaining to SBS." He graded all of the child abuse literature at the lowest end of an accepted methodology quality scale. Appropriately, Donohoe called for controlled, prospective trials into shaken baby syndrome (SBS) and opined: "Without published and replicated studies of that type, the commonly held opinion that the findings of subdural hematoma and RH in an infant was strong evidence of SBS was unsustainable, at least from the medical literature."

Greeley attacked the scholarship of Donohoe in his "Denialism in Child Abuse Pediatrics" presentation and he stated that "Those who cite Donohoe as 'evidence based' are either inexperienced in medical literature appraisal or are being disingenuous; there is no third option."

Regarding the issues themselves, 6 questions remain critical to this debate. They sit at the core of the controversies in child abuse pediatrics and are the primary questions that must be answered to evaluate medical histories in potential abuse cases both for plausibility and probability. One could pose the questions central to an objective analysis and explore the literature, both old and new, to see if support for an alternative narrative, not abuse related, exists. Is the existing literature sufficient to create medical uncertainty or legal reasonable doubt regarding the allegations of abuse when these questions are asked? Does the literature in fact support the scientific invalidity of some of the core assumptions in child abuse pediatrics and their unreliability when used to prosecute alleged child abusers? Are innocent people being incarcerated with nonevidence-based assertions in medical records and in court?

The critical questions are as follows:

Can short falls cause serious injury?

Is chronic SDH likely to rebleed with relatively minor trauma?

Does increased intracranial pressure, from SDH, cerebral edema, infectious disease, hypoxic ischemic encephalopathy and other causes, without any evidence of shaking, cause retinal hemorrhage?

Can medical problems generate findings that can be misdiagnosed as abuse?

Is shaking biomechanically insufficient to cause brain hemorrhage?

Will extreme abusive shaking result in obvious neck damage?

As the number of studies supporting the affirmative response to these questions increases, the primary constructs of child abuse pediatrics are shown to be false. Even a cursory review of the literature reveals many studies that indicate the answer to these questions is a resounding "yes."

Plunkett[3] in 2001 proved short falls cause serious injury. The 2009 article by Vezina[4] showed that chronic SDH rebleeds occur with relatively minor trauma or no trauma. Aoki and Masuzawa's 1984 study[5] shows that 100% of 26 children with SDH, not resulting from shaking, have retinal hemorrhage. Sirotnak and Frazier devote 2 chapters to "Medical Disorders that Mimic Abusive Head Trauma" in the text *Abusive Head Trauma in Infants and Children*,[6,7] published in 2006. They discuss numerous infectious, hematologic, metabolic, accidental, and other disease entities that can mimic abuse. Prange et al[8] in 2003 showed that human shaking is insufficient to cause brain damage. The study by Bandak[9] in 2005 proved that any shaking sufficient to cause brain damage will cause severe and obvious neck damage.

Given these, and numerous other studies, showing the same things, how valuable is the highly restricted certification in child abuse medicine? Does the certification advance science or justice when those seeking certification are taught that they must answer "no" to these questions to be certified? Is there any latitude to disagree with the established dogma? If you do, do you risk being labeled an "outlier" or a "denialist" too?

Dr Greeley's criticism of my article starts with innuendo that my efforts as president of the California chapter of the American Academy of Emergency Medicine in 2006, during which I initiated the effort to create a new top-tier, open-access journal of emergency medicine, created an "inside deal" that led to the publication of my article. This is unsupported and untrue. I chose *The Journal* because it offered open access that other professionals would have easy access to the material. Dr Greeley states that it took only 4 weeks to go through the peer-review process. In reality the article was submitted on December 16, 2009, some 1.5 years earlier, and went through

24 distinct drafts in response to peer review. The final version of the submission was turned around by *The Journal* in 8 weeks. The effort was coordinated by the editor and section editor to construct the message in a nuanced way, fully embracing and remaining sensitive to the controversy that the article would generate. The intent was to try to open the mind to possibilities beyond the dogma that sits at the core of child abuse pediatrics.

I am not alone in recognizing the dogmatic aspects of the positions held by Dr Greely. A recent presentation by Dr Evan Matshes at the American Academy of Forensic Medicine in 2010 was introduced with this statement:

> *"For many years, the dogma of pediatric forensic pathology was 'retinal and optic nerve sheath hemorrhages are pathognomonic of abusive head injury,' including especially, the shaken baby syndrome (SBS)."*[10]

And he ends with the following:

> *"Retinal hemorrhage and optic nerve sheath hemorrhage are not limited to children who die of inflicted head injuries; instead, they may be seen in a wide variety of situations, and may be linked to cerebral edema and sequelae of advanced cardiac life support."*

Dr Greeley prefaces his critique by claiming a "small cadre of . . . denialists" are furthering an "ideology," using a variety of "rhetorical sleights" for which he provides examples.

First, he states that I have used the common technique of "preceding and/or following controversial and unsupported statements with cited comments or phrases," the "citation sandwich." The study of cognitive errors and logical fallacies, analyzed in depth by Croskerry,[11] lists numerous types of cognitive errors, and this is not among them.

The sandwich's pieces of bread in this arcane metaphor, he argues, start with Ommaya's 1968 study,[12] the entire basis for the theory of SBS. This study measured the whiplash forces that cause loss of consciousness in monkeys and then looked at autopsy findings in those that were rendered unconscious. Massive neck injury occurred whenever brain injury was present. The other piece of bread in Dr Greeley's sandwich was the follow-up study by Ommaya and Gennarelli[13] that demonstrated abnormal neurophysiology of the cervical spine after severe whiplash. This study followed 6 years later.

His criticism is that I have "sandwiched" between Ommaya's 2 studies the idea that there would have been evidence of neck injury on computed tomography (CT) or magnetic resonance imaging (MRI) after a 600-g whiplash. Dr Greeley characterized this idea as "unsupported." That is a false statement. Barnes,[14] Bandak,[9] and others,[15–20] have stated the same thing for many years. I do cite these studies in my article, something that he seems to have overlooked with the use of this culinary metaphor.

It is known how much force is needed to cause SDH and it is known how much force it takes for the neck to fail. The ratio is greater than 10 to 1. The neck, according to all biomechanical analyses, will fail well before the forces that can cause SDH in the head can form. I wanted the reader to consider that any baby allegedly shaken to unconsciousness, and with an SDH, would likely have neck findings on CT or MRI. It was written to suggest that the absence of neck findings may provide a basis to question the shaking component of SBS and consider other medical or accidental etiologies for the brain pathology.

Next, he cites what he says is an "irrelevant conclusion." He declares that 26,000 measured helmet impacts during college football games are "unrelated to the theory that shaking of an infant can result in retinal hemorrhage." He seems to miss the point I was making, which is that impacts above 85 g do not cause SDH (or retinal hemorrhage) and human shaking can only generate a force of 10 g to 14 g. This is about one tenth of the known thresholds for injury, established by the National Highway Transportation Safety Administration at 100 g, making shaking even more unlikely as mechanism for brain or eye injury. The football study *is* relevant to a discussion of force and I believe it is relevant to retinal hemorrhage too, since none of the athletes had retinal hemorrhages at forces greater than 100 g and since humans can only generate a fraction of that force.

The next methodical criticism is "denying the antecedent." He defines this as "conclusions made that are not supported by the presented evidence." Referring to the seminal study by Plunkett[3] showing that accidental short falls from playground equipment can cause death, he himself cites a study that showed 18 of 75,000 falls (about 0.024%) resulted in death. That's about 2 out of 10,000, a rate of serious injury more frequent than the commonly quoted "1 in a million" falls that will result in serious injury, promoted by Chadwick and his colleagues[21] in 2008. The children in the study cited by Greeley were older than 1 year, with harder, more structurally solid skulls. They were less vulnerable to brain injury than infants. Children falling 5 feet or less from playground equipment can fall from similar heights at home, yet his "point" is that these household falls should be regarded as different. Biomechanically, a 5-foot fall on the playground and a 5-foot fall at home, are the same. Evidence of a 5-foot fall on the playground causing death to me, and others, is evidence that infants falling 5 feet at home can be killed as well. He states that "to imply that it [Plunkett's article] supports a short household fall can kill an infant is misleading." Really?

Furthermore, he fails to mention that serious injury from short falls, a much more common clinical event, well established by Greenes and Schutzman,[22,23] occurs as frequently as 1 in every 6 frightening short falls that present in an emergency department (ED).

Another of his examples of "denying the antecedent," reaching a false conclusion from evidence presented, is based on my selecting the wrong citation (not the wrong information) from a long list of articles by Dr Patrick Lantz, which I have in my computer files. Dr Lantz is a pediatric ophthalmologic forensic pathologist at Wake Forest University (Winston-Salem, North Carolina). Dr Greeley is right, I did intend to use Dr Lantz's 2006 American Academy of Forensic Sciences presentation[24] in which he described his experience with 111 people (16% of his total sample) with retinal hemorrhages, of whom only 30 were children who had RH at autopsy from causes other than shaking abuse. The point being made, however, remains the same: a large percentage of all deaths from any cause, have RH at autopsy.

Dr Greeley then criticizes my use of Till as a reference. I had cited Till to validate the common symptoms of apparent life-threatening events (ALTE), I was describing the presentations and nothing more. This was something I was asked to do by the editors during our 1.5-year process.

The statement I made was as follows:

> *"When these infants present after an ALTE, they may have seizures, decreased muscle tone (limpness), vomiting, failure to thrive, hydrocephalus, altered level of consciousness (LOC), color changes from hypoxic episodes, conventional or dysphagic choking, abnormal breathing patterns, and apnea.[60]"*

Reference 60 was that of Till. Dr Greeley speculates that I intended to use this study to say that ALTEs can occur with a rebleed of chronic subdural hematoma. That is true, as Vezina[4] showed, but I wasn't using Till to make that point. And he cites the following quote from Till, which I had no intention of using, since I was focused on only the symptoms associated with an ALTE.

> *"Of these 116 infants [with subdural effusions-hygroma or hematoma-SDH] nearly half had retinal hemorrhages a number which "would have been undoubtedly higher if more time had been spent examining the fundi of these babies." Till reports that the subdural collections have "no satisfactory explanation in many cases, although trauma is an important factor in the majority."*

It is my feeling that this supports my opinion (and Vezina's) about the role of minor trauma in chronic SDH causing rebleeds. Dr Greeley then states that it

> *"appears that the citation used to support Dr. Gabaeff's contention that the ALTE like symptoms of a chronic SDH can be spontaneous is that of a cohort of children many of whom likely had been abused."*

Dr Greeley's comment, "whom likely had been abused," inappropriately expands Till's causality statement beyond trauma to "abuse," when "no satisfactory explanation" is given.

Next, he raises the "straw man" argument. He writes, "This is the most widely known rhetorical technique and involved constructing an opposing point of view in a manner which makes it seem unbelievable, and thus easily discountable."

He raises the straw man argument in reference to the following statement about accidental falls that I made.

> *"[I]t is illogical to reflexively assume a different, sinister act [occult shaking] has occurred in patients who are found to have SDH after an accidental fall. Rather, we should recognize that a very small subset of all accidental falls can and do result in serious brain injury. With a large denominator of accidental falls, the serious brain injuries can and do result from innocent, accidental mechanisms, and each of these cases most likely prompts a medical encounter."*

He himself acknowledges that 0.024% of all falls cause death. Many more cause serious injury. I said simply that "a very small subset of all accidental falls can and do result in serious brain injury." I don't see the straw man. I see 2 people saying the same thing: a tiny percentage of all short falls cause serious injury. He says that this idea "makes the 'pediatric child abuse specialist' seem irrational and thus unbelievable."

Last, he invokes the "converse fallacy of hasty generalization" 3 times. This he defines as an "argument in which a single case report or instance is used to dispel an entire theory." Well, if a single short fall kills a baby, I think any statement to the effect that short falls can't cause serious injury becomes a deception. Even if it is "exceedingly rare," as Dr Greeley suggests, it still occurs, and only those with serious injury present to the ED. If only the serious, frightening falls present, and each is incorrectly diagnosed as abuse on the basis of the "exceedingly rare" argument (a logical fallacy itself), then 100% of short fall accidents that have caused serious injury will be misdiagnosed as abuse.

He references my use of Rooks as another converse fallacy of hasty generalization, for reasons that are tangential as well. I cited Rooks to show that 46% of children are born with SDH. He seems to be implying I was citing Rooks to justify that the "single case" that she characterized as a "complication" is not a justification for screening neonates for perinatal SDH.

My point regarding screening, not based on Rooks, was that abnormal behaviors in the perinatal period, followed by

enlarging heads and vague neurologic symptoms, might indicate perinatal SDH and its complications and be a reason to screen symptomatic neonates.

That point was not based on a "single case" from Rooks but from a study by Zahl and Wester[25] in Norway that demonstrated that the number of children with complications is considerably higher. By looking for complications, Zahl and Wester showed that the equivalent of approximately 2,400 babies in the United States each year will develop hydrocephalus and hygroma, diagnostic signs of chronic SDH. My suggestion was that if the condition of these babies were identified early, or widespread screening of symptomatic neonates were done, it would (1) validate the complication rate of perinatal subdural hematoma and (2) spare innocent families the false accusations of abuse after an ALTE related to these complications.

His last example of the converse fallacy of hasty generalizations relates to this statement:

> *"The American Academy of Ophthalmology has endorsed and taught the current corps of ophthalmologists that RH, schisis, retinal folds and vitreous hemorrhage are identified with intentional abuse when in fact these findings are more likely the consequence of metabolic catastrophe within the eye itself and unrelated to shaking forces as discussed above."*

It is hard to see how this is an "argument in which a single case report or instance is used to dispel an entire theory," but I can respond to Dr Greeley's misunderstanding of the point I was trying to make.

The metabolic catastrophe I referred to is clearly hypoxic ischemic encephalopathy (HIE), the type of catastrophe that is seen daily in the EDs.

Dr Greeley's narrow list of metabolic "diseases" (Menke disease, von Willebrand disease, leukemia, and glutaric aciduria), which he feels are adequate to rule out metabolic causes of bleeding, are almost never seen, and results are often not available before child abuse allegations have been made. Testing for them may create an illusion of differential diagnosis but does not change the frequency of HIE as a cause in intracranial pathology.

CONCLUSION

It was, and remains, my hope that some of the material herein and my article itself will penetrate the minds of the child abuse specialists who remain the linchpin, energy source, and ultimately, the key witnesses in court when prosecutors try to convict innocent caregivers of child abuse.

In lieu of reaching them, I hope that district attorneys, social workers, police, and judges will take the time to read about these issues. Understanding the issues in child abuse investigation and prosecution, independent of the child abuse specialist, may be necessary to correct the injustices related to the misdiagnosis of child abuse. Recognizing misplaced "certainty" of abuse, when nonspecific findings are used to diagnose abuse, is within reach for nonmedical professionals. Any independent efforts to understand the issues related to the accurate diagnosis of abuse, I believe will lead to more objective and to just end results for all concerned.

Responses like Dr Greeley's seem to indicate an intransigence to even consider alternatives. As more literature is published that undermines the dogma of child abuse pediatrics, it is neither academically appropriate nor fair to the falsely accused caregivers, families, and children to shield the past from new analyses that expose its flaws. Yet, it still seems clear that for many recognized and influential child abuse specialists this path of resistance must be followed and defended at any cost. Isn't that true denialism?

Steven Gabaeff, MD

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Aortocaval Fistula

Preeti Dalawari, MD, MSPH*
Sushma Jonna, BA†
John C. Vandover, MD‡

* St Louis University School of Medicine, Division of Emergency Medicine, St Louis, Missouri
† St Louis University School of Medicine, St Louis, Missouri
‡ St Anthony's Medical Center, Department of Emergency Medicine, St Louis, Missouri

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A 77-year-old Caucasian male with a history of hypertension presented with sudden onset of lower back pain, nausea, and vomiting. Initial vital signs included a pulse rate of 104 beats/minute, a blood pressure of 117/72 mm Hg, and pulse oximetry of 95% on room air. Abdominal examination revealed a midline pulsatile mass and bruit. The patient had bilateral lower extremity edema, which was worse on the right side. Right-sided dorsalis pedis and posterior tibial arteries were not palpable.

Computed tomography of the abdomen revealed a large 11 × 9-cm fusiform infrarenal abdominal aortic aneurysm (AAA) extending to both external iliac arteries, with contrast opacification of the inferior vena cava (Figure 1). Reconstruction imaging identified a fistula between the right common iliac artery and vein (Figure 2). The patient emergently went to the operating room and underwent repair of the AAA and ilio-iliac fistula with placement of an aortobi-iliac graft.

The reported incidence for aortocaval fistulas subsequent to an AAA is 3% to 4%. The classic triad of back or abdominal pain, a pulsatile abdominal mass, and abdominal bruit is only present in 63% of patients.[1] Thus, aortocaval fistulas are missed preoperatively in 50% of patients.[2] Presentations vary, depending on the site of fistula formation, but include high-output heart failure due to a compensatory increased stroke

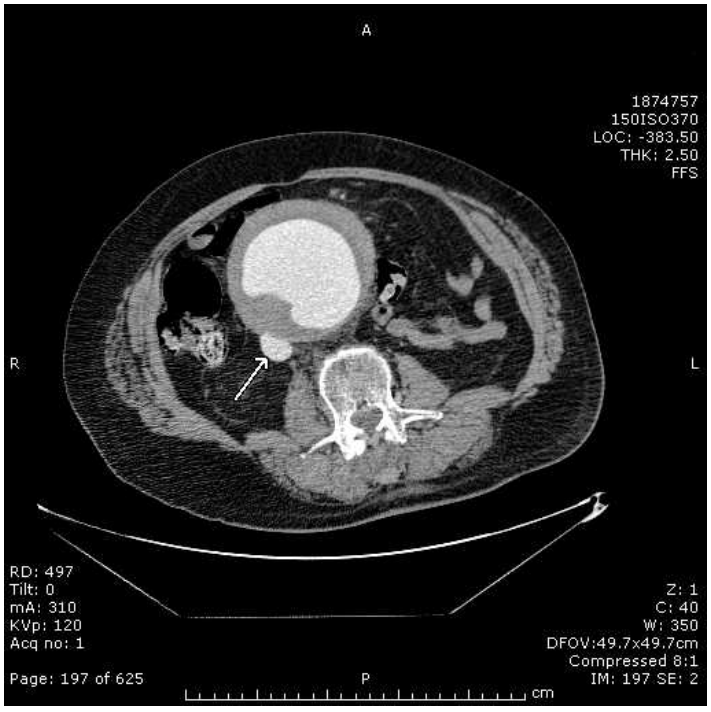

Figure 1. Computed tomography of the abdomen with contrast opacification of the inferior vena cava suggesting an aortocaval fistula.

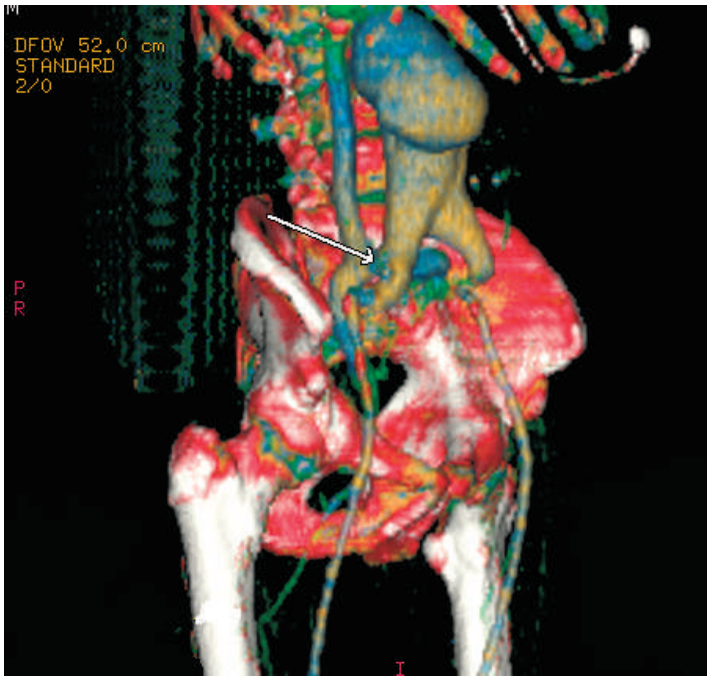

Figure 2. Reconstruction imaging showing fistula between iliac artery and vein.

volume and regional venous hypertension, such as lower extremity edema.

Address for Correspondence: Preeti Dalawari, MD, St Louis University School of Medicine, Division of Emergency Medicine, West Pavilion, Rm 315, 3635 Vista Ave at Grand Blvd, St Louis, MO 63110-0250. E-mail: pdalawar@slu.edu.

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Recognizing Infective Endocarditis in the Emergency Department

Costandinos Tsagaratos, DO
Farook W. Taha, MS, IV

University of Medicine and Dentistry at New Jersey, School of Osteopathic Medicine, Department of Emergency Medicine, Stratford, New Jersey

Supervising Section Editor: Sean Henderson, MD
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A 52-year-old Caucasian male presented to the emergency department complaining of nontraumatic painful swelling and redness of the distal left fourth finger for 2 days, associated with malaise and subjective fever. The patient denied medical history, drugs, tobacco, or alcohol use. [West J Emerg Med. 2012;13(1):92–93.]

On physical exam, temperature was 100.8°F. Heart rate was 118 with normal blood pressure and respirations. The patient had significant tenderness to pulp of finger with good capillary refill and without fluctuance (Figure 1). Cardiac exam revealed 3/6 pansystolic murmur heard loudest at the left sternal border with transmission to the apex and into the cardiac base. The patient specifically denied history of heart murmur. Upon further questioning, the patient admitted to dental work done 3 weeks prior to presentation resulting in a tooth abscess requiring oral antibiotics.

The patient had a white blood count of 16. A transthoracic echocardiogram was performed showing a 9-mm vegetation on the mitral valve consistent with infective endocarditis (IE) (Figure 2). The patient had 1 set of blood cultures drawn and was treated with appropriate intravenous antibiotics. Several sets were drawn on subsequent days, all of which showed no growth, likely secondary to partial treatment from oral antibiotics. Patient was treated empirically for a total of 6 weeks. He underwent surgical repair of leaflet due to concern of septic embolism after only 2 weeks of treatment. Patient fully recovered.

Infective endocarditis affects 10,000 to 20,000 people

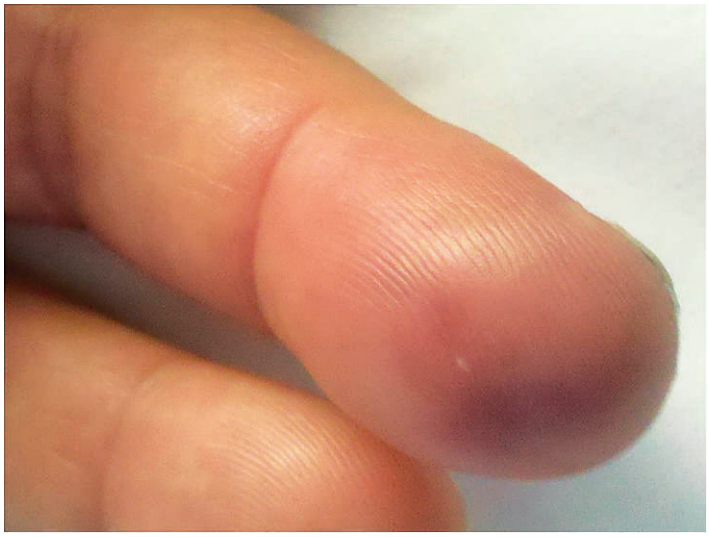

Figure 1. The Osler node on the patient's fourth digit.

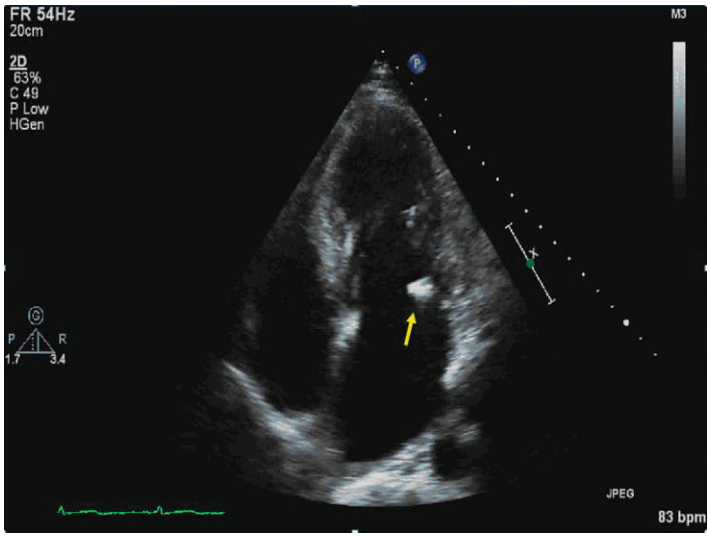

Figure 2. The vegetation on the mitral valve leaflet (arrow).

annually in the United States.[1] Patients can present with fever, fatigue, malaise, night sweats, and weight loss. On exam, a new murmur or change in existing one are clues to making a preliminary diagnosis in the emergency department.[1,2] Janeway lesions or Osler nodes can support clinical suspicion of IE.[1,3] In a prospective cohort study, the International Collaboration on Endocarditis reported 3% of 2,648 adult patients with IE had Osler node.[2] Infective endocarditis is diagnosed according to the Duke Criteria and the mainstay of treatment is Ivantibiotics.[3]

Address for Correspondence: Costandinos Tsagaratos, DO, University of Medicine and Dentistry at New Jersey, School of Osteopathic Medicine, Department of Emergency Medicine, 18 E Laurel Rd, Stratford, NJ 08084. E-mail: tsagarco@umdnj.edu.

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Acute Appendicitis from Metastatic Small Cell Lung Cancer

Radhika Sundararajan, MD, PhD
Adam B. Landman, MD
Brigham and Women's Hospital, Department of Emergency Medicine, Boston, Massachusetts

Supervising Section Editor: Sean Henderson, MD
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A 62-year-old female presented to the emergency department (ED) with right lower quadrant pain for 3 days. Past medical history was notable for small cell lung cancer (SCLC) with brain metastases. A staging positron emission tomography (PET) performed 10 days prior to presentation showed fludeoxyglucose uptake in the proximal appendix without surrounding inflammation (Figure 1).

In the ED, her physical exam was notable only for right lower quadrant tenderness to palpation, without rebound or guarding. Vital signs were within normal limits. Laboratory results were notable for a white blood count of 22 K/uL with 94% neutrophils (<5% bands). Contrast-enhanced abdominal computed tomography (CT) was performed in the ED showing a dilated appendix with enhancing wall and surrounding fat stranding suggestive of acute appendicitis (Figure 2). She was taken to the operating room emergently for a laproscopic appendectomy, which revealed a perforated appendix. The patient did well and 1 week postoperatively resumed treatment for her SCLC.

Pathology of the appendiceal specimen demonstrated oval cells and nuclear molding consistent with small cell carcinoma involving the musclaris propria.

Malignancies are a rare but known cause of appendiceal obstruction and inflammation, most commonly primary tumors, such as carcinoid or adenocarcinoma. This case of appendicitis from metastatic SCLC is exceedingly more rare, with only 7 prior case reports documented.[1,2] Interestingly, this patient also had a staging PET CT that detected the metastasis prior to appendiceal obstruction and symptom onset. Other case reports have described screening PET CT incidentally diagnosing acute appendicitis.[3,4]

While metastases are an uncommon cause of appendiceal obstruction and inflammation, they should be considered as part of the differential diagnosis when a patient with known malignancy presents with symptoms consistent with appendicitis.

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Address for Correspondence: Radhika Sundararajan, MD, PhD, Brigham and Women's Hospital, Department of Emergency Medicine, 75 Francis St, Neville House 236A, Boston, MA 02115. E-mail: rsundararajan@partners.org.

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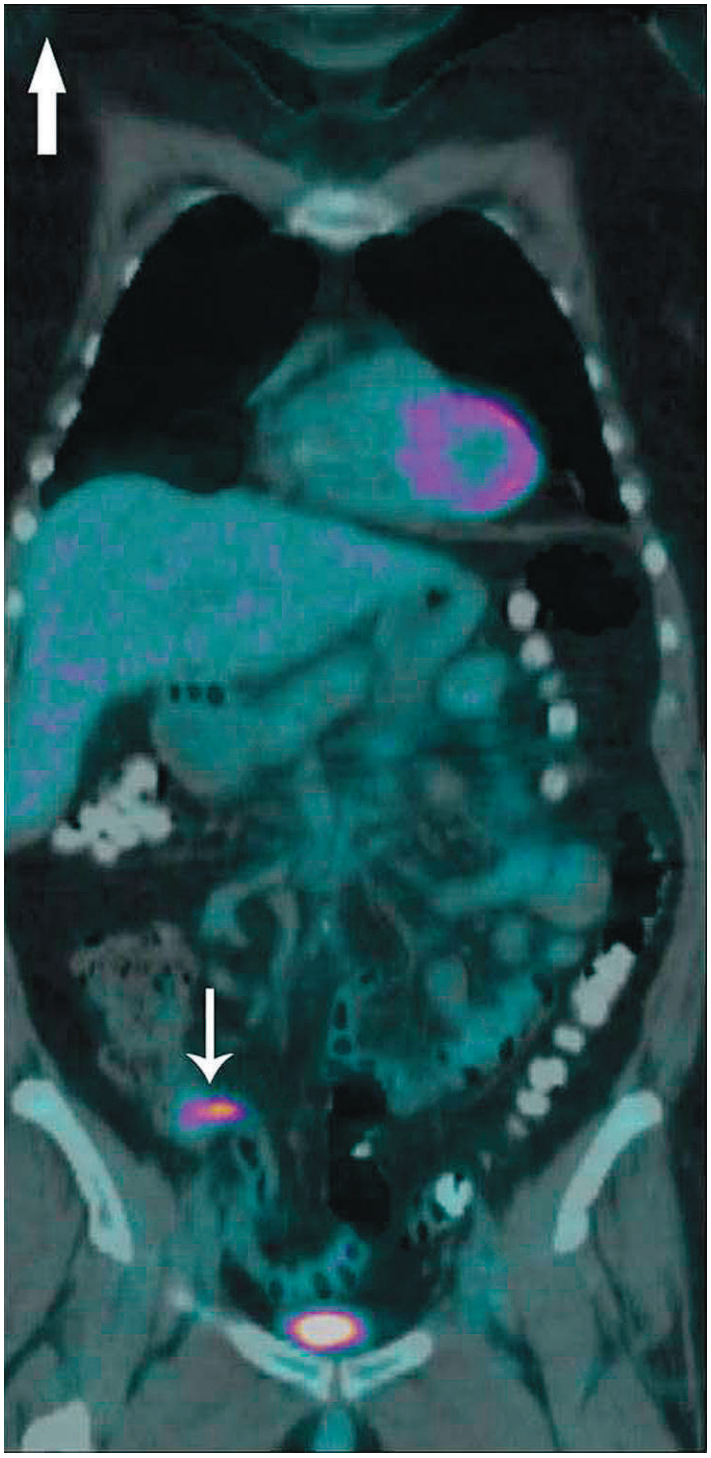

Figure 1. Positron emission tomography performed 10 days prior to presentation showing fludeoxyglucose uptake in the proximal appendix without surrounding inflammation consistent with appendiceal metastasis (arrow).

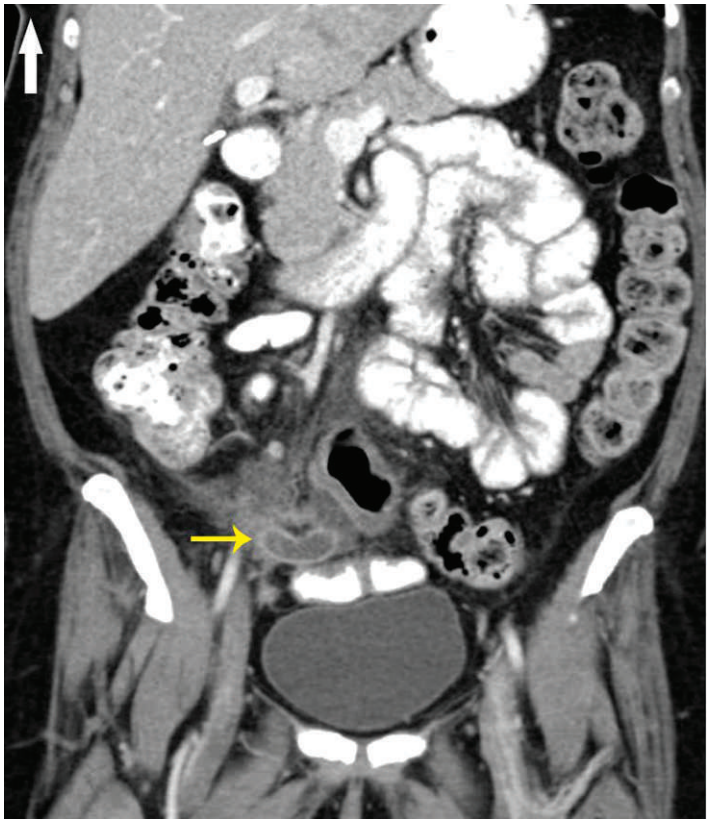

Figure 2. Contrast-enhanced abdominal computed tomography of the abdomen performed in the emergency department showing a dilated appendix with enhancing wall and surrounding fat stranding (arrow) suggestive of acute appendicitis.

Intussusception Status-Post Roux-en-Y Gastric Bypass

Steve C. Christos, DO, MS
Bridgette Svancarek, MD
Adam Glassman, MD

Resurrection Medical Center, Emergency Medicine Residency Program, Chicago, Illinois

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted April 19, 2011; Revision received April 23, 2011; Accepted April 25, 2011
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A 38-year-old female presented with acute onset epigastric abdominal pain and vomiting. Surgical history included gastric bypass surgery 8 years prior and cesarean delivery. The patient was in severe distress, afebrile, had significant epigastric tenderness with guarding, normal bowel sounds, and no distention or masses.

Results for white blood cell count, serum chemistry panel, anion gap, urinalysis, liver function tests, lipase test, and plain radiographs were all normal; computed tomography (CT) of the abdomen/pelvis showed intussusception at the jejunojejunal anastomosis (Figure). The patient underwent resection of the affected bowel segment and had an uneventful recovery.

Roux-en-Y gastric bypass (RYGB) is the most common surgical treatment of morbid obesity in the United States.[1–5] The frequency of small-bowel obstruction after laparoscopic RYGB is between 0.2% to 4.5% and can occur months to years after the procedure.[1,5] Small-bowel obstruction in these patients is usually caused by adhesions, internal hernias, and rarely, intussusception.[1–5] Intussusception must be considered because ischemia and necrosis of the affected bowel segment can occur.[5]

Clinical presentation can be acute or subacute (recurrent vague abdominal pain) and is variable (most patients do not appear ill). The most common presentation is vague abdominal pain, nausea, and vomiting.[4] Severity of pain is usually out of proportion to physical examination. Lack of obstruction symptoms does not rule out intussusception.

Findings on plain radiographs are often negative.[1,5] CT of the abdomen and pelvis (oral and intravenous contrast) is the diagnostic test of choice, with an accuracy of 80%. Pathognomonic findings include a "target sign" (Figure).[5] Patients with a history of gastric bypass surgery, persistent abdominal pain, and a negative CT finding still require surgical evaluation and possibly surgical exploration.[1,5] Blind nasogastric tube placement can lead to perforation at the gastrojejunostomy.[1] Treatment is surgical intervention, usually with resection of the affected bowel segment and reconstruction of a new jejunojejunostomy distally.[1] Recurrences can occur after surgical repair.[1]

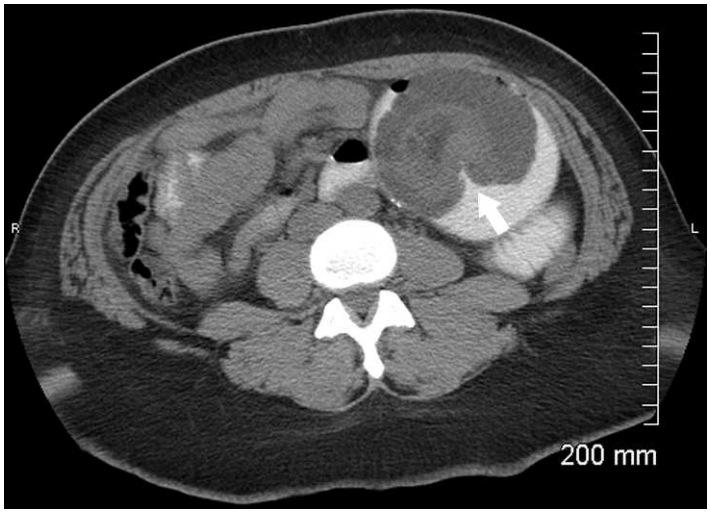

Figure. Computed tomography with oral and intravenous contrast showing the pathognomonic "target sign" (white arrow).

Address for Correspondence: Steve C. Christos, DO, MS, Resurrection Medical Center, Emergency Medicine Residency Program, 7435 W Talcott, Chicago, IL 60631. E-mail: stevesfmc@gmail.com.

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More than Just another Pulmonary Embolism

Eric R. Schmitt, MD, MPH*
Michael D. Burg, MD†

* Harbor-UCLA Medical Center, Department of Emergency Medicine, Torrance, California
† Kaweah Delta Medical Center, Department of Emergency Medicine, Visalia, California

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A 25-year-old male presented to the emergency department with 1 week of progressive right-sided chest pain. The pain was sharp, radiated to the back, and worse with deep inspiration. He denied dyspnea and leg pain or swelling. The patient's medical history was remarkable for a lower extremity deep vein thrombosis diagnosed 6 months prior. He'd self-discontinued anticoagulant therapy 2 months prior. Blood pressure was 131/66 mmHg, heart rate 76 beats per minute, respiratory rate 20 breaths per minute, temperature 36.4°C, and oxygen saturation 99% on room air. Physical examination was essentially unremarkable. Chest radiograph, urinalysis, and blood count were normal; basic chemistry was normal, except for glucose of 111 mg/dL; D-dimer was elevated at 1,485 µg/L.

A computed tomographic pulmonary angiogram (CTPA) was performed using a 64-slice machine and read by a staff radiologist as showing a pulmonary vein thrombosis (PVT) and a pulmonary artery embolism (Figures 1 and 2). A clot was also identified in the azygos vein (Figure 3). Anticoagulation was begun, and the patient was admitted. The inpatient workup for an underlying prothrombotic state was negative, including negative screen for anticardiolipin antibodies, lupus anticoagulant, and activated protein C resistance, which is found in patients with factor V Leiden; normal levels of protein S and antithrombin III. Protein C was slightly elevated at 182% (70–130%). It is unclear if the patient was tested for a prothrombin gene mutation. The patient was discharged on hospital day 3. A CTPA 7 days after hospital discharge showed no remaining arterial or venous thrombus.

PVT is an uncommon complication of lung transplantation, lobectomy, and primary and secondary tumors of the lung.[1] Case reports describe PVT following radiofrequency ablation for atrial fibrillation and repair of type B aortic dissection.[2,3] Our patient had none of these conditions, nor had he recently undergone any medical or surgical procedures. Rare idiopathic cases are described as well.[4,5] Thrombosis of the azygos vein has been described and in 1 case was associated with pulmonary embolism, but in all these cases, an underlying aneurysm of the vein was present. This was not the case in our patient.[6–8]

Presenting findings associated with PVT are nonspecific and include: fever, chest pain, dyspnea, cough, hemoptysis, hypoxia, and isovolemic opacification (an infiltrate without volume loss, ie, not atelectasis) on chest radiograph.[1,4] Evidence of embolization to the peripheral circulation—splenic infarction and bilateral femoral artery occlusion—has also been reported.[5,9]

According to published reports, PVT has been diagnosed by angiography or echocardiography.[1,4,9] PVT may also be diagnosed with a contrast-enhanced chest computed tomography and magnetic resonance imaging.[5] However, often

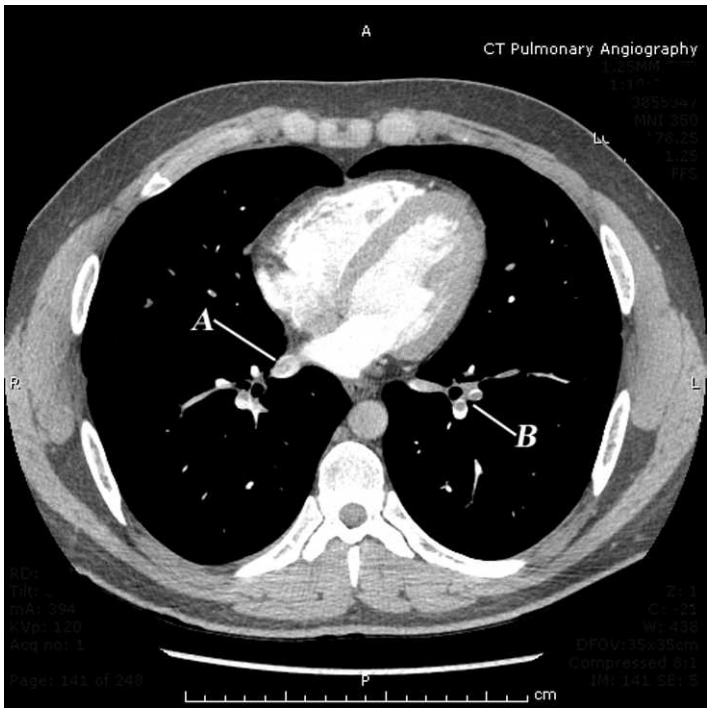

Figure 1. Axial section from the computed tomographic pulmonary angiogram demonstrating, A, filling defect in the right inferior pulmonary vein, in addition to, B, pulmonary emboli on the left.

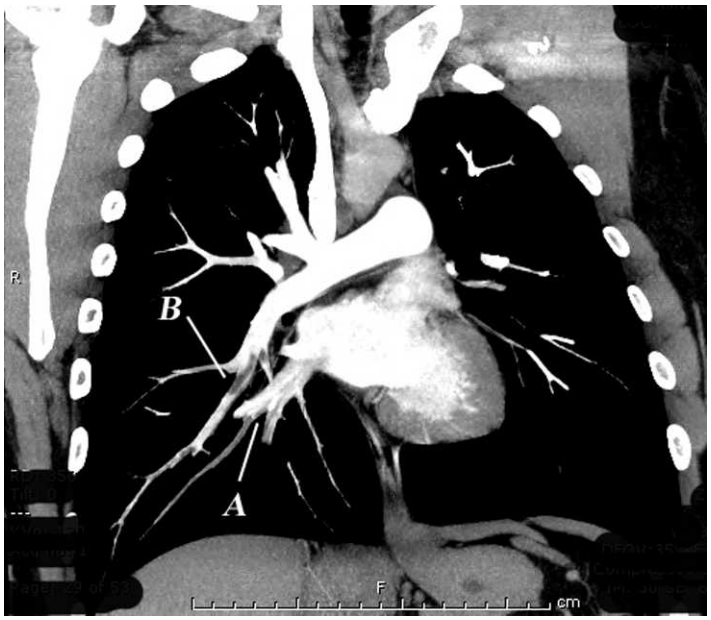

Figure 2. In this coronal reconstruction, the right pulmonary vein is seen entering into the left atrium; A, filling defects in the right inferior pulmonary vein and its branches indicate the presence of thrombus. B, Right-sided arterial emboli are also visualized.

the diagnosis is established at autopsy or during pathological evaluation of resected necrotic lung tissue.[3,4]

Conservative management hinges on anticoagulation. Earlier case reports reference the use of antibiotics, but they lack a proven role in the case of pure PVT, absent infection.[1,5] Worsening clinical status may indicate lung infarction and embolectomy, or lobe resection may be indicated.[1,4]

To our knowledge, there is only 1 prior report in the emergency medicine literature of PVT, and our case is the first reported of a PVT presenting concurrently with pulmonary artery embolism and azygos vein thrombosis.[10]

Address for Correspondence: Eric R. Schmitt, MD, MPH, Harbor-UCLA Medical Center, Department of Emergency Medicine, 1000 W Carson St, Box 21, Torrance, CA 90509. E-mail: ericrschmitt@yahoo.com.

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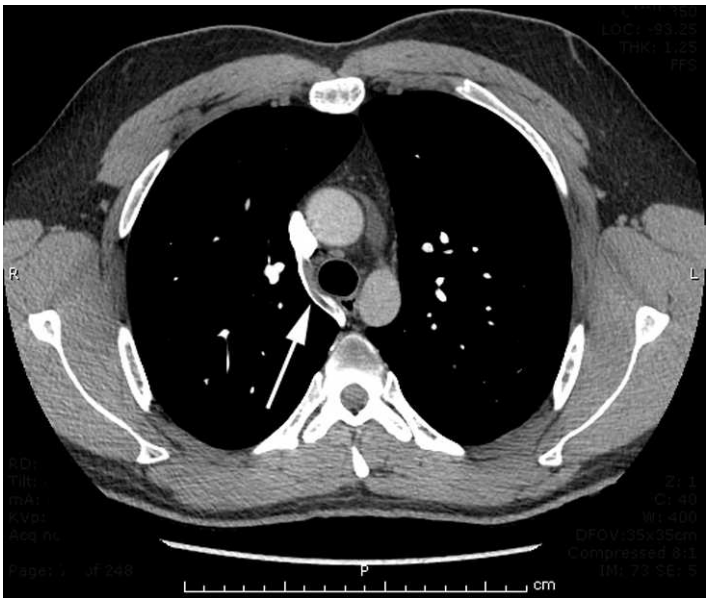

Figure 3. Axial section from the computed tomographic pulmonary angiogram showing a thrombus in the azygos vein (arrow).

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Delayed Presentation of Sigmoid Volvulus in a Young Woman

Daniel Weingrow, DO*
Andrew McCague, DO*
Ravi Shah, DO*
Fariborz Lalezarzadeh, DO†

* Arrowhead Regional Medical Center, Departments of Emergency Medicine and Surgery, Colton, California
† Private practice, San Bernardino, California

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Volvulus is an unusual condition in Western countries, generally isolated to elderly patients with multiple comorbidities. This report describes an unusual case of a very large gangrenous sigmoid volvulus in a young, otherwise healthy 25-year-old female. A review of the diagnosis and management is subsequently described. Without a consideration of the atypical demographics for sigmoid volvulus, the case illustrates the potential morbidity due to a delayed diagnosis. Early identification and management are crucial in treating sigmoid volvulus before the appearance of gangrene and necrosis, thereby avoiding further complications and associated mortality. [West J Emerg Med. 2012; 13(1):100–102.]

HISTORY

A 25-year-old female, without significant medical history, presented to our hospital emergency department with 5 days of constipation. The patient had previously been admitted to another hospital 2 days before presentation at our institution. At the previous hospital, she received magnesium citrate, sodium phosphate enema, bisacodyl suppository, 4 L of GOLYTELY (Braintree Laboratories Inc, Braintree, Massachusetts), metoclopramide, metronidazole, promethazine, and dicyclomine, along with various analgesics and intravenous fluids. The patient then left the other facility without experiencing improvement. At the time of presentation at our institution, the patient had complaints of diffuse abdominal pain, nausea, vomiting, and persistent constipation but with passage of flatus. The patient reported no fever, dysuria, melena, hematochezia, or recent travel.

On examination, the patient appeared moderately distressed. Breath sounds were clear bilaterally and the patient's heart was auscultated as a regular rate without murmurs. There was a well-healed anterolateral thoracotomy scar consistent with an unknown cardiac surgery performed shortly after birth. The abdomen was severely distended with diffuse marked tenderness without rigidity or guarding; no hernias or masses were noted and no stool was present in the rectal vault.

The patient's vital signs were unremarkable. The patient's laboratory results were notable for a leukocytosis of 18.9 mm^3 with a neutrophilic predominance and bandemia of 7%; and sodium levels of 130 mEq/L, potassium levels of 3.3 mEq/L, and bicarbonate levels of 21 mEq/L with an anion gap of 11 mEq/L. Urinalysis was notable for acetonuria and negative β-humanchorionic gonadotropin. Computed tomography (CT) images (Figure 1) were read by the staff radiologist as sigmoid volvulus with severe dilation of the colon.

The patient was taken to the operating room for exploratory laparotomy and was found to have 15 to 20 cm of dilated sigmoid colon (Figure 2) with multiple mesenteric twists of sigmoid mesentery with redundant transverse colon. The sigmoid colon was resected with a stapled anastomosis. No intraoperative complications were noted, and the patient was transferred to the surgical intensive care unit. The patient had an uneventful postoperative course and was discharged on postoperative day 7.

DISCUSSION

Volvulus occurs when colon twists on its mesenteric axis with a greater than 180-degree rotation, producing obstruction of intestinal lumen and mesenteric vessels.[1] The most common locations for volvulus to occur include the sigmoid colon, cecum, splenic flexure, and transverse colon in order of decreasing frequency.[2]

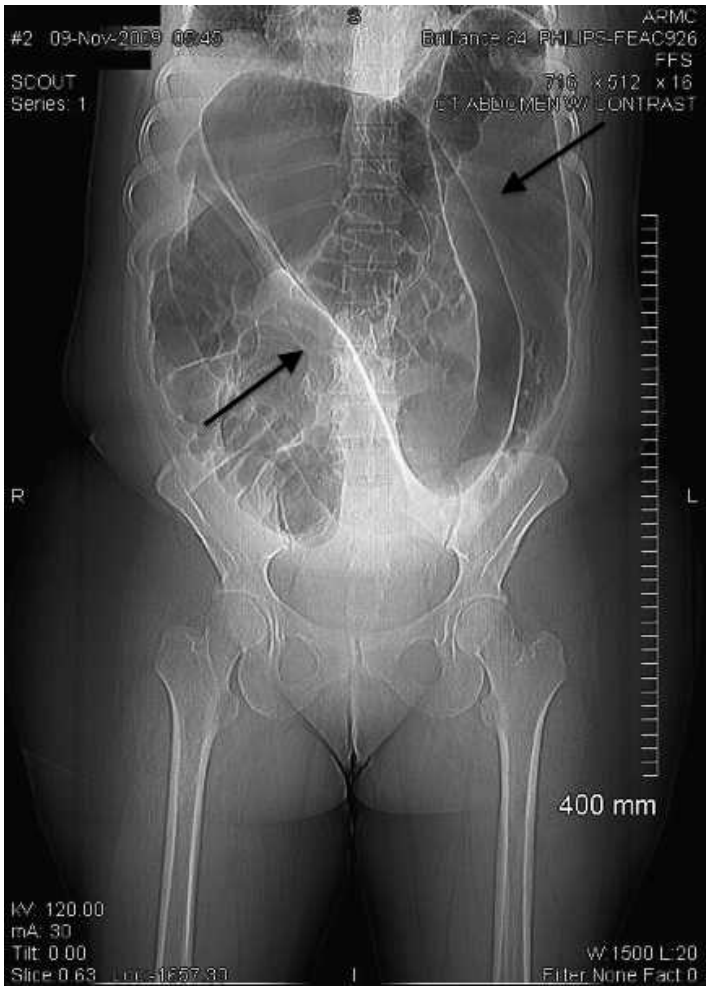

Figure 1. Computed tomography of abdomen showing "coffee bean sign."

Sigmoid volvulus has a variable geographic distribution, being extremely common in developing countries where it affects the young patient, with a lower incidence in Western countries where it predominantly affects the elderly.[3] Chronic constipation is blamed for the Western type of sigmoid volvulus, while a high amount of fiber in the diet has been deemed a major factor in the development of sigmoid volvulus in the African population.[2,4]

Sigmoid volvulus has been classically divided into 2 types, by clinical course, as described by Hinshaw and Carter.[5] Acute fulminating volvulus, caused by a complete obstruction, has a clinical presentation of sudden onset periumbilical pain with emesis and constipation. Patients frequently have peritoneal signs on examination.[5] Gangrene and perforation are commonly early complications with this type of volvulus. Conversely, with subacute progressive volvulus, patients have only partial obstruction and therefore have a more insidious onset. The subacute form is frequently seen in older patients, with a more subtle clinical picture, described as poorly characterized abdominal cramping, often worse on the left side

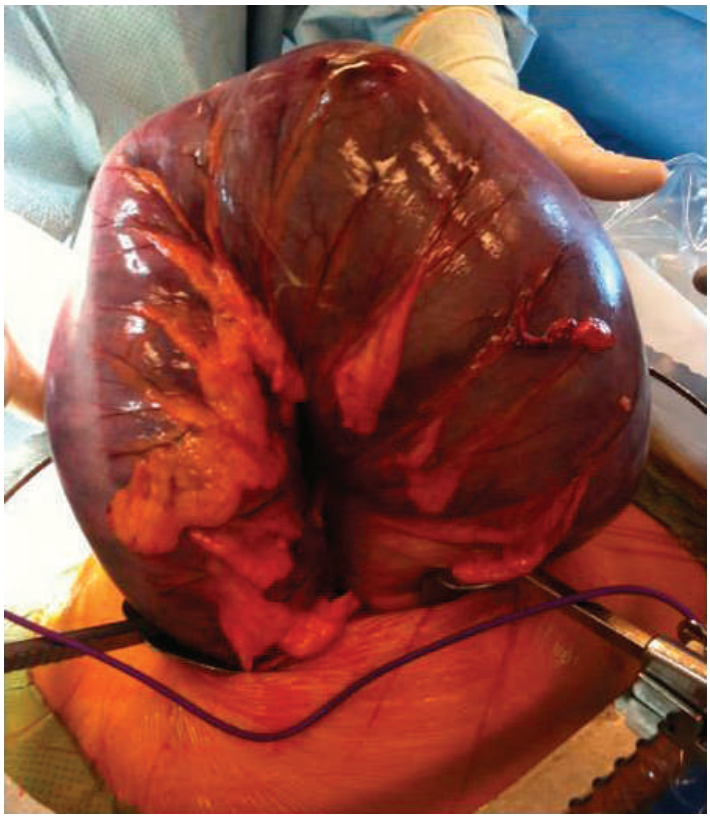

Figure 2. Intraoperative findings revealing sigmoid volvulus.

of the abdomen.[5] The understated clinical symptoms in subacute progressive volvulus often lead to delay in diagnosis. On physical examination, upper abdominal distention with associated tenderness, tympany, an empty rectum, and visible peristalsis are all associated with both forms of sigmoid volvulus.

Plain abdominal radiographs may help the diagnosis; however CT, magnetic resonance imaging, and flexible endoscopy are more accurate.[6,7] Several radiologic diagnostic signs are described, such as omega or horseshoe sign, bird's beak sign, Y sign, northern exposure sign, coffee bean sign, bent inner tube or ace of spades sign, left pelvic overlap or left flank overlap sign, liver overlap sign, the whirl sign, and empty left iliac fossa sign.[7–9]

Surgeons generally advise a 2-step approach, first an endoscopic derotation followed by a subsequent elective surgical correction by colopexy.[5,10] Sigmoidosocopy is the initial treatment for those patients without peritoneal signs. Decompression rates vary, with 70% to 90% success. Insertion of a rectal tube should follow to further decompress the viable bowel.[3,11,12] Barium enema has been described as another alternative when attempting to untwist a volvulus and is successful in about 5% of patients.[13] The disadvantage of sigmoidoscopic decompression includes risk of perforation. Expectant management is not recommended, as spontaneous

reduction is found in only 2% of patients and recurrence is high in this group.[14]

Urgent laparotomy is recommended when decompression is unsuccessful or if the patient is felt to be at high risk for gangrene or perforation. When gangrenous bowel is discovered, immediate resection is necessary.[15] After resection, colostomy and mucous fistula, or Hartmann procedure, is recommended.[16] In only 10% of cases is colon found to be gangrenous. In cases for which viable colon is encountered, the decision of whether or not to resect must be made. When resected, there is controversy regarding whether to restore intestinal continuity. Generally, if the colon is viable, evidence favors primary anastomosis when feasible.

Nonsurgical detorsion offers the flexibility of scheduling surgery at a next available date.

Some authors suggest a 4-week delay before definitive surgery. Traditional operation is resection of at least the sigmoid colon.[3] Laparoscopic resection of the sigmoid colon is growing in popularity and may have a role for high-risk patients or those who may not tolerate conventional surgery.[17,18]

This report describes sigmoid volvulus in an otherwise healthy young patient with considerable delay in definitive diagnosis. Unfortunately, the patient experienced considerable morbidity because of the unconventional patient profile, traditionally associated with sigmoid volvulus in Western countries. The case emphasizes the importance of early identification in the atypical patient before the appearance of twisted loop gangrene, in order to optimize patient management.

Address for Correspondence: Daniel Weingrow, DO, Arrowhead Regional Medical Center, Department of Emergency Medicine, 400 N Pepper Ave, Colton, CA 92324-1819. E-mail: dangrow@gmail.com.

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Right-Sided Sigmoid Diverticular Perforation

Andrew Little, MS
Andy Culver, DO
Ohio University College of Osteopathic Medicine, Affinity Medical Center Emergency Department, Massillon, Ohio

Supervising Section Editor: Rick McPheeters, DO
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Diverticulosis is a common disorder among geriatric patients, of whom 10% to 25% go on to develop diverticulitis. Known complications of diverticulitis include formation of phlegmon, fistula, bowel obstruction, bleeding, perforation, and colonic abscess. A less common complication is perforation with formation of an extra-abdominal necrotizing abscess. This case is a report of an 83-year-old female who presented to the emergency department with a necrotizing abdominal wall abscess secondary to right-sided diverticular microperforation. [West J Emerg Med. 2012;13(1):103–105.]

CASE REPORT

An 83-year-old female presented to the emergency department secondary to an accident involving her wheelchair. Earlier that day she had been seen by her family physician regarding a subcutaneous abscess located on her abdomen and was on her way to see a general surgeon when she had an accident, whereby she was flung from her wheelchair. She was fully immobilized and on a backboard. She was later taken off the backboard after passing Nexus criteria for cervical spine immobilization, with no complaints of pain other than in her left lower quadrant. After being taken off the backboard, a full physical examination was performed. The patient's vital signs were blood pressure of 124/59, heart rate of 107, respiratory rate of 18, temperature of 97.5°F, and a pulse oximetry of 95% on room air. Auscultation of lungs found them to be clear bilaterally in all fields; cardiac auscultation found normal S1 and S2 with no murmurs, gallops, or rubs. Upon examining the patient's abdomen, bowel sounds were found in all 4 quadrants and pain was elicited upon palpation of her left lower quadrant, where an 18 × 8-cm subcutaneous abscess was noted. The abscess was fluctuant with a central area of black necrotic skin measuring 8 × 3 cm. Upon direct palpation around the abscess, some oozing was noted centrally. During the physical examination we learned that the patient also suffered from Parkinson disease complicated by dementia, so her husband became our primary historian. Upon questioning him about the mass, he said he had noticed it in the previous 2 days, with the central black necrosis only being present since the previous day. He also stated that his wife had been complaining of increasing pain over the past few days, which was worsened with bowel movements. Upon review of her old medical records, it was noted that she also had a history of gastroesophageal reflux, hypertension, hypothyroidism, renal failure, pulmonary hypertension, diastolic heart failure, and was recently hospitalized for an episode of acute diverticulitis with perforation, for which she was treated medically. With the combination of her presenting symptoms and recent and past medical history, we ordered a complete blood count, basic metabolic panel (BMP), and a urinalysis. An abdominal computed tomography (CT) with intravenous (IV) contrast was ordered. A surgical consult was made while waiting for her laboratory results.

The patient's laboratory results showed a white blood cell count of 27.9 × 1,000/uL (92% neutrophils, 3.4% lymphocytes, 4.5% monocytes, 0.1% eosinophils, 0.05% basophils), hemoglobin of 9.4 g/dL, hematocrit of 27.6%, and a platelet count of 446. Her BMP showed a serum sodium of 134 mmol/L, potassium of 3.4 mmol/L, blood urea nitrogen of 23 mg/dL, and creatinine of 1.1 mg/dL. Finally, her urinalysis was positive for nitrites, with microscopy showing 1+ leukocytes and 4+ bacteria. The CT revealed an inflamed colon with a fistulous tract winding into the left lower quadrant connecting to a large subcutaneous abscess, with inflammatory changes and pockets of gas and subcutaneous air noted throughout. The patient's history and her physical examination, laboratory, and CT findings were indicative of necrotizing fasciitis possibly secondary to diverticular perforation (Figure).

A surgical consultant evaluated the patient and agreed with

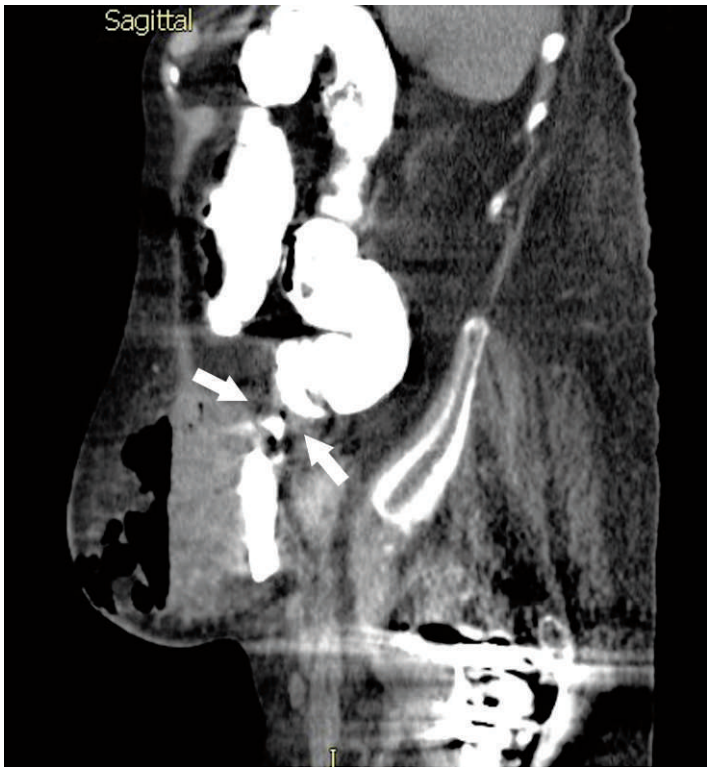

Figure. Sagittal reconstruction of abdominal computed tomography showing connection of abscess with the sigmoid colon (white arrows).

our findings of possible necrotizing infectious process, possibly due to her previous diverticular perforation. The patient was given broad-spectrum antibiotics and IV fluids. Presurgical laboratory tests were performed and she was admitted to the intensive care unit (ICU) and later underwent surgical intervention, which included debridement of large amounts of malodorous pus and fluid consistent with a mixed anaerobic infection. After successful debridement of the abscess, a complete survey of the abscess showed numerous areas of necrosis within the subcutaneous fat, and a small opening at the base of the abscess was noted that emanated pus from the abdominal cavity. This was enlarged for further examination of the patient's large bowel, where the distal right sigmoid colon was very inflamed and a tiny microperforation was found. Examination of the rest of the large bowel showed no other signs of infection, and the sigmoid microperforation was ruled the source of the patient's necrotizing infection. The patient then underwent sigmoid resection with placement of a diverting colostomy with wound vacuum placement. Her wound site was cultured during surgery and subsequently grew multiple organisms, which included *Proteus mirabilis, Escherichia coli, E faecalis, Bacillus fragilis*, and a coagulase-negative staphylococcus. The patient was transferred to the ICU on ventilatory support, where she received medical therapy for sepsis. Her hospitalization was complicated by failure to wean from ventilatory support and by multiple bouts of fever secondary to sepsis. She underwent 3 other surgeries for further abscess debridement. She was later transferred to a long-term acute care hospital for long-term care and rehabilitation.

DISCUSSION

This case is an example of a rare, emergent, important complication of acute diverticulitis.[1] Typically, patients with acute diverticulitis present with symptoms that include lower left quadrant pain, nausea, vomiting, diarrhea, constipation, flatulence and fever.[2] It can also manifest with numerous complications including phlegmon, intra-abdominal abscess, fistulas involving adjacent organs, and distant septicemia.[2–4]

Necrotizing fasciitis is a condition caused by anaerobic and gram-negative bacteria (ie, *Bacteroides*, *Proteus*, and *Enterobacter*, as in this case). It typically proliferates in areas of trauma, hypoxia, recent surgery, and medical compromise.[5] Its presentation is hallmarked by the symptoms of fever, pain out of proportion, crepitance upon palpation of the abscess, and areas of erythema. The diagnoses can be made by incising the suspected abscess; looking for visual clues of necrosis; or by performing imaging to look for free air and/or gas under the skin or in the subcutaneous tissue.[5] Care should be taken to recognize necrotizing infections as soon as possible so prompt and proper treatment can be initiated. Owing to the prevalence of diverticulitis, and its high prevalence of perforation in patients older than 60 years, it is very important to get a gastrointestinal history when dealing with anyone presenting with such abdominal complaints.[2,3,6] Since necrotizing infections are considered a surgical emergency, it is also important to find the source of infection and consult surgery services emergently so as not to delay surgical intervention.[5]

As used for this patient, abdominal CT is the best imaging study for ruling in or ruling out colonic involvement.[7] When diagnosing acute diverticulitis, CT gives clinicians the most information about location and involvement of adjacent or distant organs or structures when compared to other imaging modalities.[3,4] Another imaging modality that could be attempted in this patient would be bedside ultrasonography. Ultrasonography, to the trained user, is a quick, easy, and relatively painless imaging modality that can be used to measure abscess size and to look for channeling abscess contents. Ultrasonography can also be used to differentiate between cellulitis and an actual abscess.[8] The limitation of ultrasonography is the difficulty in determining (as in this case) whether or not colonic involvement is present or whether the abscess has penetrated the abdominal wall.[3] Magnetic resonance imaging (MRI) is a modality that can also be used when investigating intra-abdominal abscesses.[9,10] Its effectiveness compared to CT has not been investigated to date, but the utility of MRI is likely comparable to that of CT.[11] Some of the issues with using MRI in emergent situations, and other cases of necrotizing infections, are the time necessary to

complete the scan (hours instead of minutes) and the availability of scanners.

Treatment for acute diverticulitis is often purely medical, but surgical intervention is indicated in some cases.[2,3] Medical management consists of antibiotics targeted to treat common bacteria found in the colon (gram-negative rods and anaerobic bacteria), intravenous fluids, and pain medication while monitoring the patient's vital signs; management should be started in the emergency department before admission to the hospital. Medical treatment alone is typically reserved for patients who are deemed to be poor surgical candidates and for those who do not suffer from concurrent complications (ie, perforation, bleeding), with a success rate of 70% to 100%.[3] Surgical intervention would include treating the patient medically and removing involved portions of the sigmoid colon along with other involved sections of bowel. Patients are typically managed medically first until the complications mentioned above are apparent or are imminent.[2,3] The key to treatment of complicated acute diverticulitis is early recognition, early surgical consultation, and initiating treatment in a timely manner.[3]

Acute diverticulitis is a disease that, as our population ages, is sure to increase in prevalence and presentation to the emergency department. It is important for clinicians to promptly recognize patients who are having an acute episode of diverticulitis so that they can begin treatment and avoid severe complications such as those suffered by the patient in this case study.

Address for Correspondence: Andrew Little, MS, Ohio University College of Osteopathic Medicine, Affinity Medical Center Emergency Department, 875 8th St NE, Massillon, OH 44646. E-mail: al257807@ohio.edu.

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Massive Gastric Distension from Chronic Intestinal Pseudo-Obstruction

Andrew G. DeNazareth, MD* * Creighton University, Division of Internal Medicine, Omaha, Nebraska
Venkata M. Alla, MD† † Creighton University, Division of Cardiology, Omaha, Nebraska
Stephen J. Lanspa, MD‡ ‡ Creighton University, Division of Gastroenterology, Omaha, Nebraska

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A 68-year-old male with moderate mental retardation presented to the emergency department with anorexia, constipation, and abdominal distension for 4 days. Medical history was significant for partial colon resection for presumed bowel obstruction 3 years previously (no true anatomic cause for obstruction was identified at surgery). On examination, he was dehydrated and tachycardic, with blood pressure of 90/60 mmHg and a distended, tympanic, and mildly tender abdomen. Metabolic panel, amylase, and lipase test results were otherwise normal. An abdominal radiograph demonstrated marked gastric distension with multiple dilated loops of small and large bowel (Figure 1). Computed tomography of the abdomen with contrast confirmed the above findings but did not identify any mechanical cause for bowel obstruction (Figure 2). The patient had significant symptomatic improvement after intravenous

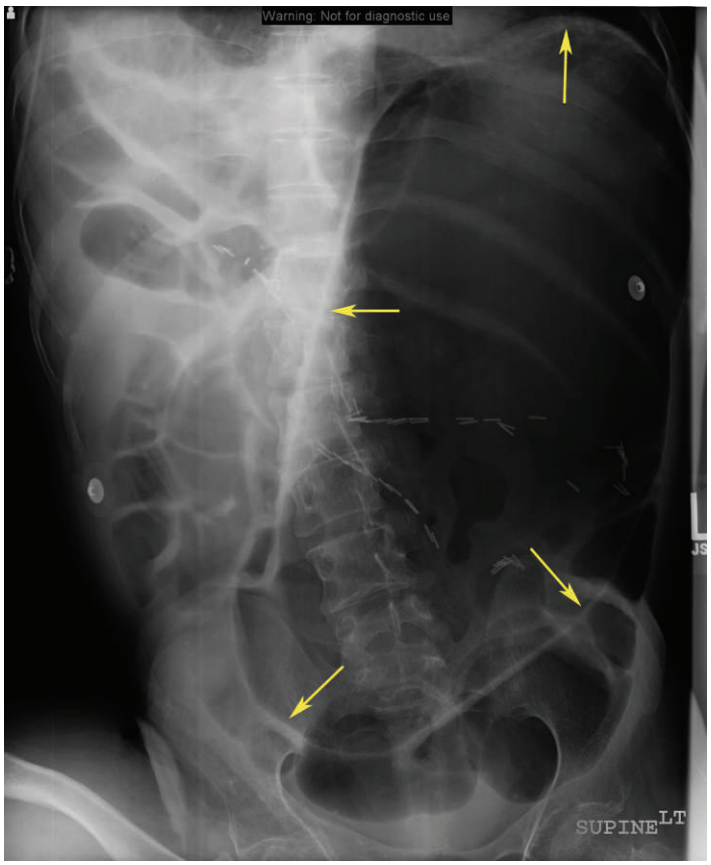

Figure 1. Abdominal radiograph showing massive gastric distension outlined by arrows.

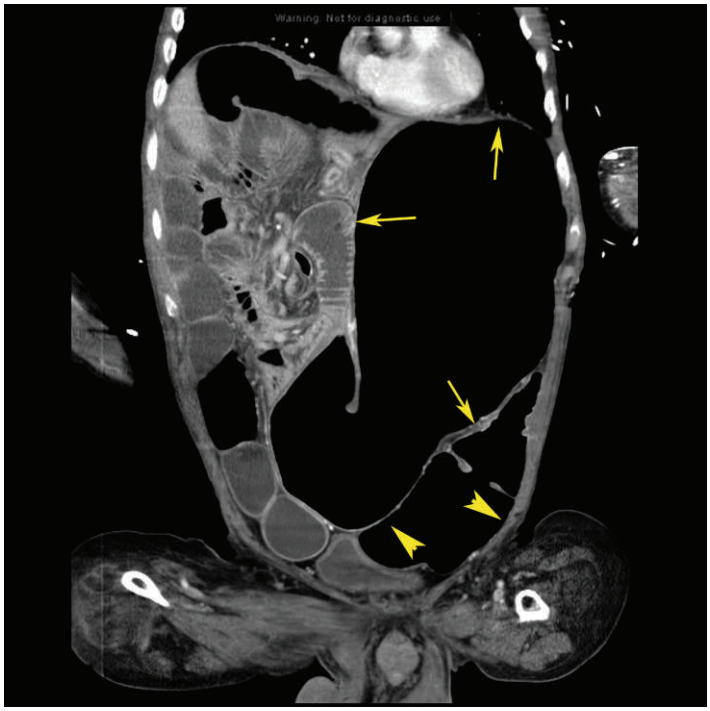

Figure 2. Coronal computed tomography demonstrating the massively dilated stomach (arrows) and dilated bowel loops (arrowheads).

hydration, gastric decompression, and initiation of intravenous metoclopramide and erythromycin.
Esophagogastroduodenoscopy revealed no evidence of gastric outlet obstruction.

Chronic intestinal pseudo-obstruction (CIPO) is a rare disorder of gastrointestinal motility characterized by repetitive/chronic symptoms of bowel obstruction in the absence of a mechanical or metabolic cause of obstruction.[1,2] Radiation enteritis, drugs such as clonidine, opiates, systemic disorders such as diabetes, hypothyroidism, amyloidosis, scleroderma, and multiple sclerosis can also produce a similar clinical picture. Therefore, exclusion of aforementioned secondary causes is mandatory for the diagnosis of idiopathic CIPO. Abnormalities in the integrity of intestinal neural pathways, interstitial cells of Cajal, and smooth muscle cells of the gastrointestinal tract have been implicated in the causation of CIPO.

Acute management involves decompression, appropriate fluid and electrolyte replacement, and nutritional support.[1] Prokinetic agents such as metoclopramide, erythromycin, octreotide, and neostigmine have been shown to help improve bowel transit times.[1,2] Pacing of the stomach or intestine and intestinal transplantation are considered experimental. Increasing awareness about CIPO is essential to ensure early diagnosis, appropriate treatment, and hopefully avoid unnecessary abdominal surgeries in these patients.[1]

Address for Correspondence: Andrew G. DeNazareth, MD, Creighton University Medical Center, Department of Internal Medicine, 601 N 30th St, Ste 5850, Omaha, NE 68131. E-mail: andrewdenazareth@creighton.edu.

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Diplopia from Subacute Bilateral Subdural Hematoma after Spinal Anesthesia

Getaw Worku Hassen, MD, PhD
Hossein Kalantari, MD
New York Medical College, Metropolitan Hospital Center, Department of Emergency Medicine, New York, New York

Supervising Section Editor: Rick A. McPheeters, DO
Submission history: Submitted August 3, 2011; Accepted August 4, 2011
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Subdural hematoma (SDH) is a rare, but life-threatening complication of spinal anesthesia. Subdural hematoma resulting from this procedure could present with vague symptoms such as chronic headache and could easily be missed. Chronic headache is one of the symptoms of chronic SDH in postpartum women. Diplopia as the presenting complaint in SDH secondary to peripartum spinal anesthesia has not, to our knowledge, been previously reported. Here, we report a case of diplopia secondary to postpartum subacute bilateral SDHs with transtentorial herniation after spinal anesthesia in a healthy primagravid 25-year-old woman. SDH can expand gradually and the initial symptoms might be subtle as in our case, despite critically high intracranial pressure. [West J Emerg Med. 2012;13(1):108–110.]

INTRODUCTION

Spinal anesthesia has become very popular for obstetric procedures. Such use was first described in 1901.[1] It involves the injection of anesthetic solution into the spinal peridural or subarachnoid space. Spinal anesthesia offers the advantage of avoiding general anesthesia and the patients' remaining awake during the procedure.[2] Complications of spinal anesthesia include hypotension, postdural puncture headache, meningitis, spinal hematoma and reversible sensory loss, and paraplegia. Subdural hematoma (SDH) and abducens palsy are uncommon complications of spinal anesthesia.[3–8] Increased intracranial pressure (ICP) may manifest as chronic headache and focal neurologic deficits.[9–11]

CASE DESCRIPTION

A 25-year-old woman (G1, P1, A0) presented to the emergency department with a chief complaint of binocular double vision. She stated that she had delivered her first baby vaginally about 4 weeks before presentation, under spinal anesthesia. The entire pregnancy and peripartum period were uneventful except for moderate neck pain with stiffness and spasm starting shortly after delivery, for which she used ibuprofen every 6 hours for 2 to 3 days. The patient reported no headache, nausea, vomiting, change in vision, difficulty speaking, numbness, or weakness during that time. Two weeks after delivery she started noticing double vision, which progressively worsened to the point where she was having difficulty driving her car.

The review of systems was negative for fever, chills, chest pain, dyspnea, nausea, vomiting, abdominal pain, or urinary symptoms. She reported no recent or remote head trauma as well as no bleeding tendency or family history of bleeding disorders. She had not taken any medications except ibuprofen for her neck pain.

The physical examination did not demonstrate any abnormalities except horizontal diplopia on forward gaze and left lateral gaze. Diplopia disappeared on gaze to the right and when covering either eye. Visual acuity was normal when each eye was examined individually. Speech, visual field, motor, sensory, deep tendon reflexes as well as gait and coordination examinations were all unremarkable.

Laboratory tests including complete blood count, complete metabolic panel, and coagulation parameters were within normal limits (platelet count, 247×10^9/L; international normalized ratio, 1.03). Chest radiograph and electrocardiogram were unremarkable. A noncontrast computed tomography (CT) of the brain revealed large bilateral subacute SDH involving the bilateral fronto-temporo-parietal regions

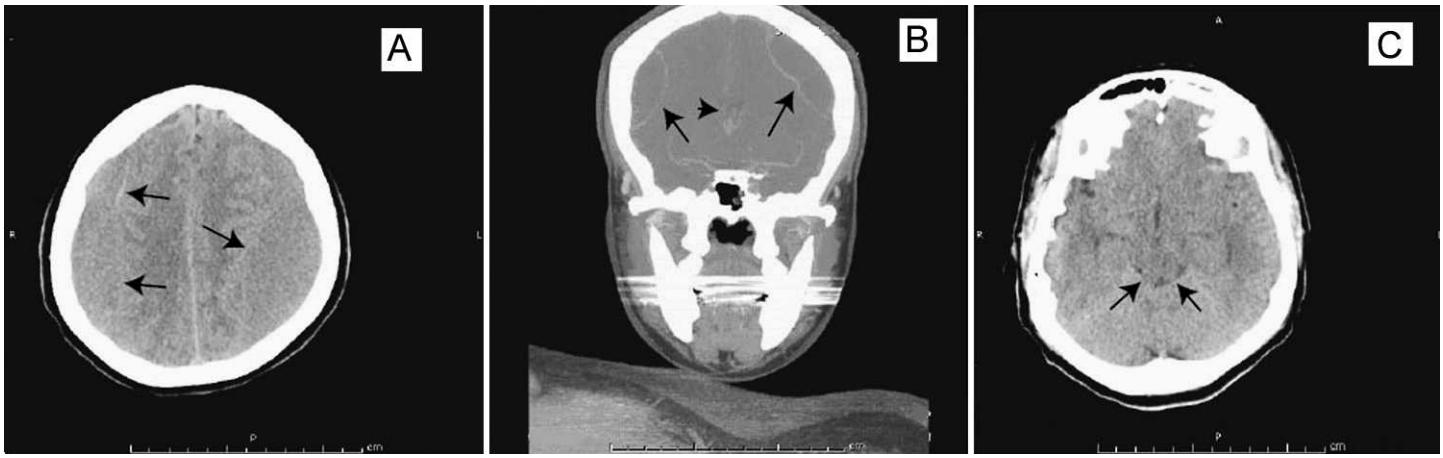

Figure. Computed tomography (CT) of the head showing bilateral subacute hematomas at the convexity of both hemispheres with ventricle compression. A, Transverse CT of the head with bilateral subdural hematoma (arrows). B, Axial CT of the head demonstrating bilateral subdural hematomas (big arrows) and compression of the ventricle (arrowhead). C, The diminishing of the basal cisterns next to the brain stem indicates the increased intracranial pressure and downward displacement of the brain caudally (arrows).

with compression of the lateral ventricles and transtentorial herniation (Figure, parts A through C). A CT angiography of the head and neck did not reveal any vascular abnormality.

Neurology and neurosurgery services were consulted and the neurosurgical team admitted the patient for surgical intervention and further management. The patient underwent craniotomy with evacuation of bilateral SDH. No other abnormalities were noted intraoperatively, nor did any postoperative complications occur. The patient did well after the procedure with marked improvement of her symptoms. During the course of her hospital stay, the diplopia resolved completely and the patient remained asymptomatic without any neurologic deficit.

DISCUSSION

Multiple complications can occur after lumbar puncture (LP), including cerebral and spinal herniation, postlumbar puncture headache (PLPH), cranial neuropathies, nerve root irritation, low back pain, infection, and bleeding.[4,9,12,13]

PLPH is a frequent complication of lumbar puncture and spinal anesthesia. The mechanism that is proposed for this phenomenon is the persistent leakage of cerebrospinal fluid through the dural puncture site, leading to the caudal displacement of the brain with traction on pain-sensitive structures such as blood vessels.[4,14,15] Similar mechanisms may apply traction force on the bridging veins, possibly causing a slow and constant blood leakage from these veins. The blood can accumulate over time causing an SDH and leading to symptoms such as headache or focal neurologic deficit.[3,10,11,16]

Contributing factors to the development of SDH include trauma, cerebral atrophy, the use of an anticoagulant after surgery, and bleeding disorders. Contributing factors to the development of PLPH are cerebral atrophy and dehydration.[17,18]

There is no uniform definition of PLPH, but a widely accepted definition is as follows: a constant headache appearing or worsening significantly upon assuming the upright position and resolving or improving significantly upon lying down. PLPH may persist up to 2 weeks and generally responds well to pain medications; however, occasionally, placement of epidural blood patch may become necessary. Headache from SDH may persist longer and frequently does not respond to analgesics. This headache is more likely due to increased ICP secondary to the development of subdural hygroma and subsequent SDH.[19,20]

Our patient did not develop the characteristic PLPH. She had no other predisposing factors for SDH, such as trauma or coagulation disorders. She only reported moderate neck pain with stiffness and spasm. She did not have any focal neurologic deficit except double vision. The double vision improved initially, but subsequently caused significant lifestyle restrictions leading to her decision to seek medical care.

Abducens palsy has been reported after LP and spinal anesthesia,[13,21] but there was no peripartum or SDH association. Earlier reports of cranial nerve (CN) palsy suggested that the downward traction of the brain and compression of the CN leads to the neurologic deficit.[9,21] In our patient, the downward displacement of the brain secondary to bilateral SDH may have caused compression of the cranial nerve that led to her symptom. Abducens palsy is the most common CN affected in post-LP palsy. The abducens nerve may be more sensitive to this effect, in comparison to other cranial nerves, owing to its long intracranial course. Abducens palsy usually occurs 4 to 14 days post LP and spinal anesthesia. It can be unilateral or bilateral and is usually associated with PLPH.[4,9]

CONCLUSION

Patients with SDH post spinal anesthesia may present with persistent headache, weakness or numbness in their extremities, or isolated cranial nerve palsy. Since a subtle focal neurologic deficit may be the initial presenting sign or symptom of a potentially devastating ICP, with or without impending herniation, physicians should maintain a high index of suspicion in such cases.

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Address for Correspondence: Getaw worku Hassen, MD, New York Medical College, Metropolitan Hospital Center, Department of Emergency Medicine, 1901 First Ave, New York, NY 10028. E-mail: getawh@yahoo.com.

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Therapeutic Hypothermia for Acute Air Embolic Stroke

Matthew Chang, MD
John Marshall, MD
Maimonides Medical Center, Emergency Department, Brooklyn, New York

Supervising Section Editor: Kurt R. Denninghoff, MD
Submission history: Submitted October 19, 2010; Revision received March 21, 2011; Accepted April 11, 2011
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We describe a 65-year-old man who presented to the emergency department with acute cerebral air embolism after receiving computed tomography guided lung biopsy. The patient presented with muscle strengths of 0/5 in left arm and 3/5 in left leg, aphasia, and a right pneumothorax. Our concern was to improve the patient's neurological function, while ensuring stability in a patient who possibly had systemic air embolism and a penumothorax, which is a contraindication to hyperbaric treatment. To that end, we treated the patient with therapeutic hypothermia for 24 hours. The patient went on to recover full neurological functions.

CASE

A 65-year-old man presented to the emergency department (ED) after a computed tomography (CT) guided lung biopsy by interventional radiology with an acute cerebral air embolism. Prior to presentation, the patient had experienced progressive dyspnea for the prior few months and had an outpatient CT that revealed a lung mass. The patient subsequently presented to the interventional radiology suite for a CT guided lung biopsy, which later revealed adenocarcinoma. He received Versed and fentanyl for the procedure but remained alert and conversive during the procedure.

Shortly after the procedure, the patient became unresponsive. The rapid response team was activated immediately, but prior to their arrival, the patient became more alert. Upon assessment, the patient was noted to be aphasic, with left hemipareiss. A head CT was done and revealed right middle cerebral artery watershed area air embolism and hypodensity consistent with acute infarct (Figures 1 and 2). The chest CT showed no evidence of air embolism in the thoracic vasculature, but demonstrated a right pneumothroax, and a chest tube was inserted. The patient was not oriented to self, time, and place, but could follow simple commands intermittently. At that point, which was roughly about 30 minutes after the initial onset of symptoms, the patient was transported to the ED.

Upon initial assessment in the ED, the patient had the following vital signs: temperature of 99.4°F, pulse of 86, respiratory rate of 18, and blood pressure of 120/91. The blood glucose obtained by finger stick was 149. The patient was hemodynamically stable, alert, speaking a few words in a confused manner, and was not following simple commands. Physical exam revealed bilateral breath sounds, with slightly decreased breath sounds on the right and a right-sided pigtail catheter visibly in place. On neurological exam, the patient was noted to be flaccid in the left arm, with 0/5 in motor strength and not responding to noxious stimuli. He had 3/5 in muscle strength on the left lower extremity and was responding sluggishly to noxious stimuli.

Throughout his assessment, the patient was placed in Trendelenburg position while receiving 100% oxygen via nonrebreather mask. Hyperbaric oxygen treatment was entertained during evaluation of the patient. However, the patient presented with a concomitant pneumothorax, which is a contraindication that could be exacerbated by hyperbaric treatment. In addition, although the patient was stable upon presentation, the patient presented acutely following air embolism introduced into the vasculature and needed to be in a closely monitored setting, such as in the ED, that was prepared for resuscitation.

We, therefore, proceeded with the decision to intubate the patient and began inducing therapeutic hypothermia for neuroprotection. We used a commercial cooling device, and the target temperature was set at 31° to 33°C with the intention of maintaining the patient's temperature at 33°C for 24 hours. The patient was also given boluses of 4 mg lorazepam and 10 mg vecuronium and started on a titratable midazolam drip to prevent shivering and maintain sedation. The patient was maintained on therapeutic hypothermia for 24 hours in the surgical intensive care unit, and the patient was extubated on hospital day 2.

Upon initial assessment following extubation, the patient was alert, oriented, and exhibited 2/5 muscle strength in the left upper extremity and 3/5 muscle strength in the left lower extremity, which was an improvement from the left upper

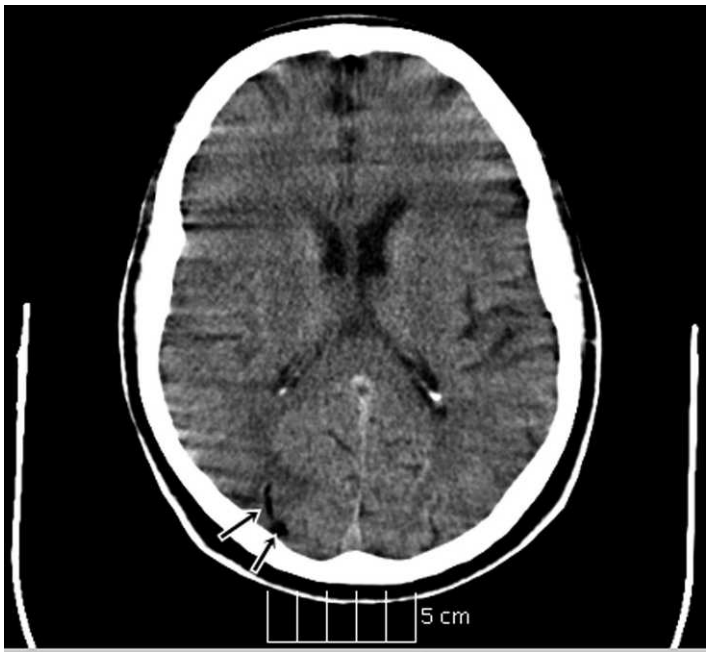

Figure 1. Head computed tomography showing multiple foci of air embolism as indicated by arrows.

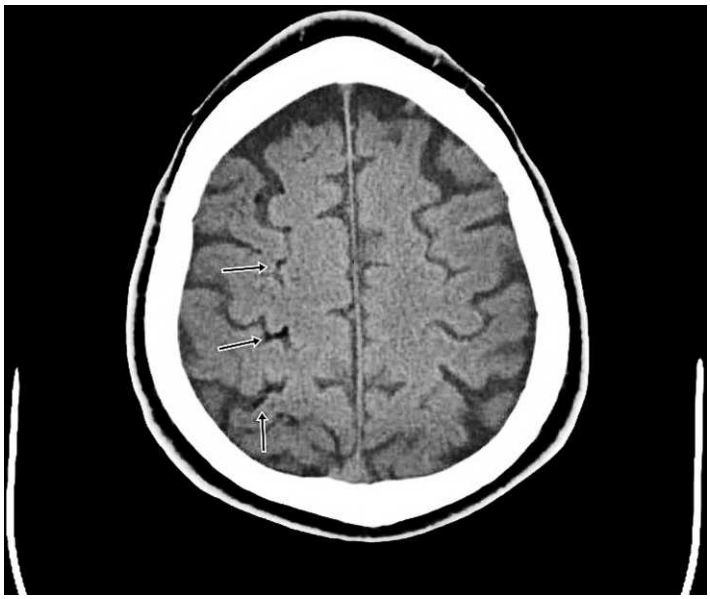

Figure 2. Head computed tomography showing multiple foci of air embolism as indicated by arrows.

extremity muscle strength of 0/5 on initial presentation to the ED. The patient began physical therapy daily and continued to show improvement neurologically. A repeat head CT showed resolution of air emboli on hospital day 4, with evolving right parietal-occipital hypodensity. On hospital day 5, the patient was assessed to have 4/5 in muscle strength in left upper and lower extremities, and left lower extremity muscle strength improved to 5/5 on hospital day 6. With continued effort in encouragement of out-of-bed activities and physical therapy, patient's left upper extremity muscle strength improved to 5/5 on hospital day 8. Overall, the patient made a full recovery neurologically and was later discharged on hospital day 21. The delay in discharge was mainly due to the patient developing Clostridium difficile infection.

DISCUSSION

Air embolism is a known, rare complication of thoracic needle aspiration and biopsy. Its incidence has been noted to be low ranging from 0.02% to 0.4%.[1–3] The standard treatment for air embolism is to immediately initiate 100% oxygen in order to facilitate nitrogen diffusion into the blood serum and increase the rate of resorption of air.[4] There is also evidence that suggests therapeutic advantage by placing the patient in supine position.[5] Furthermore, hyperbaric oxygen therapy is given in order to increase the diffusion gradient between the air bubble and surrounding tissues.[5]

Therapeutic hypothermia after cardiac arrest has been extensively studied and acknowledged for its neuroprotective effect following reperfusion of cerebral tissue.[6,7] Therapeutic hypothermia is thought to suppress many of the chemical reactions associated with reperfusion injury. These reactions include free radical production, excitatory amino acid release, and calcium shifts, which in turn can lead to mitochondrial damage and apoptosis.[8] Although the evidence is not as conclusive as in the situation of postcardiac arrest, there have also been numerous suggestions that therapeutic hypothermia could be beneficial in the case of acute ischemic stroke, especially when the onset of the stroke is recent and reperfusion is anticipated within the timeframe that cerebral tissue is still salvageable.[9–13]

We'd like to point out that the main difference between using therapeutic hypothermia in patients with return of spontaneous circulation (ROSC) following cardiac arrest and in patients who suffered ischemic stroke is that patients with ROSC after cardiac arrest have a more defined pathophysiology with a progression from absent or poor perfusion during arrest to reperfusion during spontaneous circulation. After cardiac arrest, the evidence is clear that cerebral reperfusion has occurred when there is a palpable pulse and perfusing blood pressure. In contrast, it is difficult to determine the amount of perfusion and especially reperfusion following an ischemic stroke caused by an embolus or thrombus. Ultimately, reperfusion is dependent on a variety of variables, including the size and composition of the obstruction, which affect the rate of resolution.

Unlike a solid embolus, an air embolus by definition has properties of gas, such as diffusion and resorption. We believe that the intrinsic properties of gas suggest that the resolution of an air embolus and subsequent reperfusion could be expected to occur in a more timely manner when compared to the typical acute ischemic stroke, in which reperfusion is dependent on the resolution of the solid obstruction.

In addition to neuroprotective benefits during reperfusion,

1 article suggests that intra-ischemic cooling reduces infarct size under animal models.[9] This article further suggests that tissue salvage is commonly achieved when hypothermia is initiated within 60 minutes of stroke onset and less effective when cooling is delayed.

There have also been other case reports of air embolism treated with therapeutic hypothermia.[14–16] In those cases, the patients were under cardiopulmonary bypass and had air inadvertently introduced into the vasculature. Consistently, their patients received suction to remove the air mechanically from the vasculature as well as cardiac massage to expel air in 1 case.[16] The patients were then cooled using cardiopulmonary bypass respectively to 22°C for 1 hour, 24°C for 40 minutes, and 20°C for about 10 minutes.

Although all 3 cases went on to describe favorable neurological outcomes in their patients, there was no radiological evidence, unlike our current case, to indicate that their patients sustained cerebral air embolisms initially, and there was also no clinical evidence that their patients sustained acute neurological compromise since the patients were not alert and were not examined. One can only infer in their cases that therapeutic hypothermia improved neurological outcomes by assuming that there was neurological compromise in the first place. Furthermore, their patients received mechanical air removal prior to therapeutic hypothermia. Therefore, it is uncertain if the favorable neurological outcomes should be attributed to the mechanical air removal or to the therapeutic hypothermia, assuming that there was in fact air embolism remaining following mechanical air removal.

In contrast, the current case describes a symptomatic patient presenting with an acute cerebral air embolism diagnosed clinically by exam and radiologically by CT, in which therapeutic hypothermia is the only primary intervention.

Address for Correspondence: Matthew Chang, MD, Maimonides Medical Center, Emergency Department, 4802 10th Ave, Brooklyn, NY 11219. E-mail: chang_matt@yahoo.com.

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Benign Nuchal Rigidity: The Emergency Department Evaluation of Acute Prevertebral Calcific Tendonitis

Zachary Levy, MD
James Carroll, MD
Heather Farley, MD

Christiana Care Health System, Emergency Medicine Residency Program, Newark, Delaware

Supervising Section Editor: Sean Henderson, MD
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Acute prevertebral calcific tendonitis (APCT) is a rare condition, the exact incidence of which is unknown. It is of particular interest to the emergency physician owing to the other potentially devastating conditions in the differential diagnosis of neck stiffness and/or odynophagia (including retropharyngeal abscess, infectious spondylitis, and meningitis.) In contrast, APCT has a benign clinical course and can be easily managed in the emergency department. We will present a case of APCT, followed by a brief discussion of the disease and current literature. [West J Emerg Med. 2012;13(1):114–116.]

CASE REPORT

A 66-year-old male presented to our emergency department (ED) with a chief complaint of nuchal rigidity and odynophagia. He had been seen earlier in the day by his primary care physician, who subsequently referred him to the emergency department for further work-up.

The patient reported that he had been in his usual state of health until 2 days before his visit, when he experienced the somewhat rapid onset of diffuse neck pain and stiffness, followed by significant pain upon swallowing and a nonproductive cough. He reported no chest pain, shortness of breath, headache, nausea, or vomiting. He did, however, report an intermittent low-grade fever at home since the onset of his symptoms. He reported no significant recent illness or hospitalization. His past medical history was notable only for hyperlipidemia and hypertension, both controlled by medications. He had no known drug allergies and was a nonsmoker.

On arrival, his vital signs were as follows: temperature (oral), 37.3°C; pulse, 97 beats per minute; respirations, 16 per minute; blood pressure, 125/91. His oxygen saturation was 96% on room air. On examination, he was awake, alert, oriented, and in no acute distress. He was able to phonate and was protecting his airway without difficulty. He had marked nuchal rigidity, which was associated with lateral soft tissue neck tenderness. His oropharyngeal examination was somewhat limited secondary to mild trismus, but there was no evidence of tonsilar exudate or peritonsilar abscess. There was no lymphadenopathy. His lungs were clear bilaterally. The remainder of his physical examination was unremarkable.

Lateral soft-tissue radiographs of his neck (Figure 1) revealed prominent prevertebral soft tissues with patent hypopharyngeal and proximal tracheal air columns. The epiglottis was unremarkable and no unusual gas patterns were present; however, abnormal ossification was noted along the inferior aspect of the anterior arch of C1. This prompted follow-up imaging with computed tomography of the neck (Figure 2), which showed an 8-mm retropharyngeal calcific focus with a small, nonloculated, fluid collection along the anterior aspect of the longus colli. Based on these findings, a diagnosis of acute prevertebral calcific tendonitis (APCT) was made.

The patient received 15 mg of intravenous ketorolac in the emergency department. Shortly afterwards, he reported marked alleviation of his symptoms and was able to tolerate per os fluids without difficulty. After a discussion with the on-call otolaryngologist, the patient was discharged home with a prescription for oral non-steroidal anti-inflammatory drugs (NSAID) and a recommendation for outpatient follow-up in the ear, nose, and throat clinic in 1 to 2 weeks as needed. The

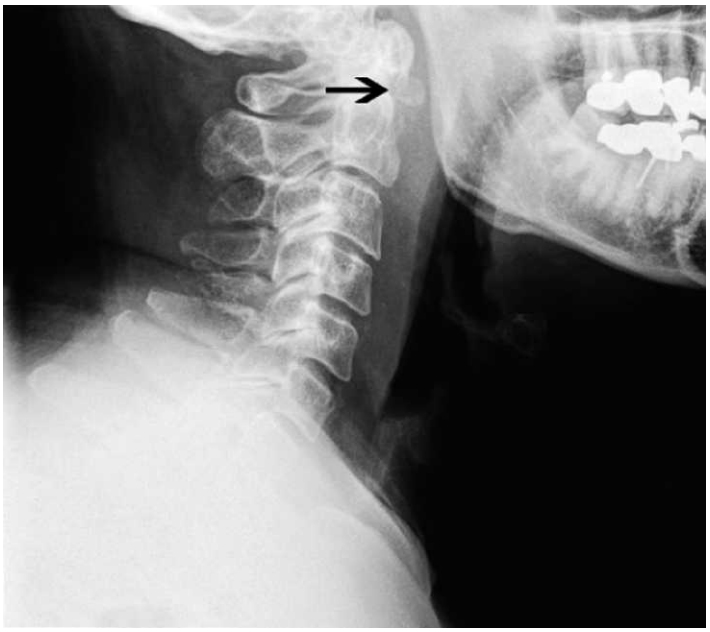

Figure 1. Plain radiographs, though less sensitive than computed tomography, are an acceptable screening examination. A calcific focus is visible just anterior to C1.

patient subsequently reported complete pain relief with oral NSAID therapy.

DISCUSSION

The longus colli traverses the anterior spinal column from C1 to T3 and is divided into 3 portions: superior oblique, inferior oblique, and vertical. Acute prevertebral calcific tendonitis (alternatively referred to as retropharyngeal tendonitis and longus colli tendonitis) is an inflammatory calcification of the longus colli and was first mentioned in 1964 when Hartley[1] described a "large amorphous calcium deposit just anterior to the atlanto-axial joint." Since that time, the disease has remained relatively obscure, and research on the subject has consisted primarily of case reports (although Hviid et al[2] have suggested that this obscurity is primarily the result of underrecognition.)

Seventy-six patients diagnosed with APCT have been identified in the existing literature; in this group, the disease shows a slight female predominance (~58%), with the average age of onset being 46 years. This is fairly consistent with the epidemiology intimated in prior reports,[3] in which the disease is often described as afflicting females in the third through sixth decade of life.

The exact pathogenesis of the condition is unknown, although it is believed to be similar to other forms of tendinitis that occur in the body owing to hydroxyapatite deposition, dystrophic calcification, and subsequent inflammatory response.[4] In particular, it is strikingly similar in epidemiology, pathogenesis, and clinical course to the equally obscure "crowned dens syndrome," which involves calcification of the

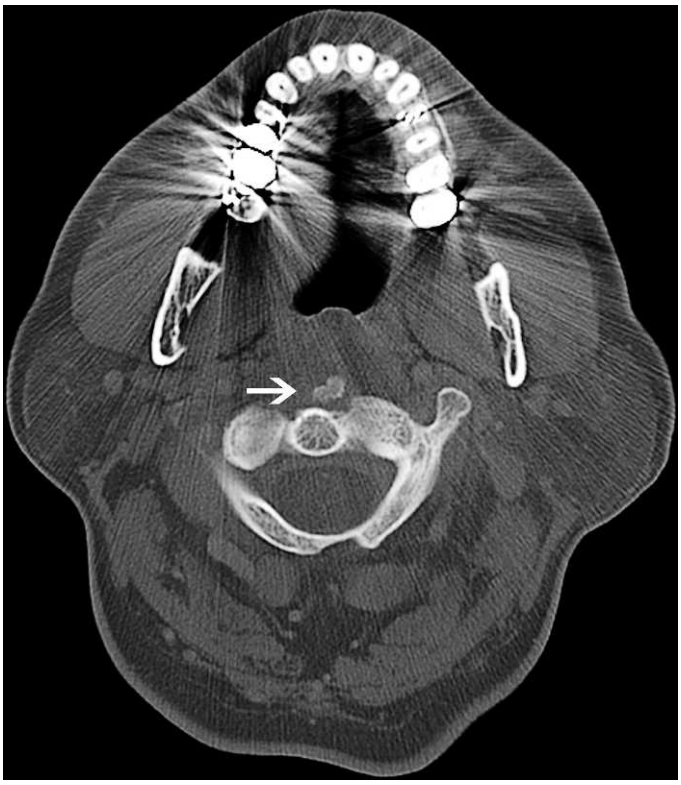

Figure 2. Neck computed tomography bone windows demonstrate the abnormal prevertebral calcium deposit.

ligaments surrounding the odontoid process.[5] Although typically occurring in the anterior C1 to C3 region, APCT has also been described at C5 to C6,[6] at the origin of the vertical portion of the longus colli.

In APCT, nuchal rigidity and odynophagia are the 2 most commonly reported chief complaints. Fever and leukocytosis may be present,[7] as well as elevated levels of C-reactive peptide,[8] often prompting a more extensive work-up in search of an infectious etiology. Imaging will usually demonstrate the pathognomonic calcium deposit[9] and, coupled with the physical examination, will usually allow the clinician to confirm the diagnosis. Computed tomography is more sensitive[7] than plain radiography for detecting the condition, owing to better contrast resolution, and is the confirmatory scan of choice. Plain films, however, will often end up being the initial screening examination and will demonstrate a significantly abnormal expansion of the retropharyngeal space (by as much as 1.5 to 2.0 cm, as reported by Haun[9]), with or without visible calcification. Use of magnetic resonance imaging (MRI) has also been described, showing characteristic edema confined to the longus colli.[10] Vollmann et al[11] have suggested that MRI with diffusion-weighted imaging is the preferred modality when available.

The clinical course of APCT appears universally benign and self-limited,[12] in that no fatalities or significant complications have been described; however, the sample size on which this assumption is based is quite small, and the

possibility of significant airway compromise cannot be entirely dismissed. Nevertheless, symptoms resolve in about 1 to 2 weeks, correlating with calcium deposit resorption[9] (although the resorption process itself may not be radiologically complete for several months.) As mentioned above, oral NSAIDs are the hallmark of outpatient treatment and are usually sufficient monotherapy for pain control. Occasionally, a short course of corticosteroids[13] may be appropriate when NSAIDs provide suboptimal relief. Follow-up with an otolaryngologist may be offered but is usually unnecessary.

Address for Correspondence: Zachary Levy, MD, Christiana Care Health System, Emergency Medicine Residency Program, 4755 Ogletown-Stanton Rd, Newark, DE 19713. E-mail: levy.zachary@gmail.com.

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Tension Gastrothorax in a Child Presenting with Abdominal Pain

Ross Hooker, MD
Ilene Claudius, MD
Anh Truong, MD

Los Angeles County USC Medical Center, Department of Emergency Medicine, Los Angeles, California

Supervising Section Editor: Sean Henderson, MD
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A 4-year-old girl was brought to our hospital by her parents because of abdominal pain. She had suffered minor trauma after rolling from her standard-height bed 2 days prior. Vital signs were appropriate for age. Physical examination was remarkable for decreased breath sounds to the left side of the chest. A chest radiograph (Figure) demonstrated a large gas-filled structure in the left side of the chest with mediastinal shift. [West J Emerg Med. 2012;13(1):117–118.]

In the emergency department the patient began exhibiting respiratory distress, and after nasogastric tube insertion, she became bradycardic and hypoxic, and finally lost pulses. The patient underwent intubation and cardiopulmonary resuscitation was initiated. Vigorous suction from the nasogastric tube was initiated, with prompt return of vital signs. The patient recovered well and underwent an operative procedure in which the spleen and part of the stomach and transverse colon were reduced into the abdomen. A 5-cm posterolateral defect in the diaphragm was identified and repaired. The patient left the hospital at neurologic baseline.

Tension gastrothorax is a rare life-threatening condition occurring when the stomach herniates through a defect in the diaphragm into the chest and becomes distended with air, leading to hemodynamic compromise.[1,2] This is seen most commonly in the perinatal period in a patient with a congenital diaphragmatic hernia, or after acute major abdominal trauma or surgery in an otherwise healthy individual, or sometimes years later.[2,3] In these settings, diagnosis requires a high level of suspicion, as this will often be mistaken for a tension pneumothorax, a far more common and equally life-threatening condition.[1,4] A poorly differentiated diaphragm on chest radiograph, or visualization of a nasogastric tube in the stomach (in the chest), can help differentiate these conditions.[2] Tension

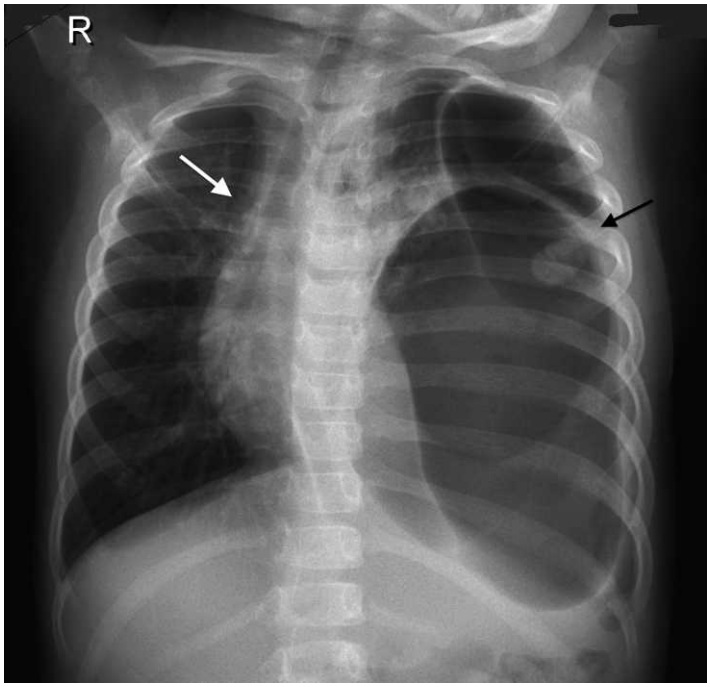

Figure. Chest radiograph of a 4-year-old-girl who presented with abdominal pain after minor trauma, showing a large, gas-filled structure in the left hemithorax (black arrow) and mediastinal shift (white arrow) and poor definition of the left diaphragm.

gastrothorax is treated initially with prompt nasogastric tube placement and vigorous suction of contents and, when stable, intraoperative reduction and repair.[1,2]

Address for Correspondence: Ross Hooker, MD, Los Angeles County USC Medical Center, Department of Emergency Medicine, Room 1011, 1200 N State St, Los Angeles, CA 90033. E-mail: rosshooker@hotmail.com.

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Kohler's Disease

Nirav Shastri, MD
Lauren Olson, MD
Milton Fowler, MD

University of Missouri–Kansas City, Children's Mercy Hospital and Clinics, Overland Park, Kansas

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted December 23, 2010; Revision received January 5, 2011; Accepted January 10, 2011
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We present a pediatric case report of foot pain due to Kohler's disease. [West J Emerg Med. 2012; 13(1):119–120.]

A 4-year-old girl presented with intermittent right foot pain for 1 week. Pain had worsened the previous day after playing outside, and she was now refusing to bear weight on the right foot. On examination, she had pain and tenderness over her right dorsomedial midfoot with no local skin changes. She walked with an antalgic gait with weight bearing on the lateral side of the foot. Her right foot radiograph showed a collapsed, flat, and radiodense navicular bone (Figure).

Kohler's disease is a rare, self-limiting, avascular necrosis of the navicular bone, first described in 1908. It is usually unilateral and most often affects boys. Its usual onset is between 4 to 5 years of age but can present as early as 2 years of age. Girls with this condition are often younger than boys, probably owing to earlier onset of ossification.[1,2] The pathophysiology of this condition is best explained by a mechanical cause associated with a delayed ossification. Navicular is the last tarsal bone to ossify and can get compressed between the already ossified talus and cuneiforms when the child becomes heavier. This in turn compresses the navicular bone's perichondral ring of blood vessels, producing ischemia of the central spongy bone and avascular necrosis. The prognosis remains excellent owing to this radial arrangement of blood supply.[1] Radiologic findings show patchy areas of navicular with sclerosis and rarefaction with loss of normal trabecular pattern. Sometimes the navicular may appear collapsed or may be normal in shape with a uniform increase in density with minimal fragmentation. Treatment includes pain control and using soft arch supports or medial heel wedge. Patients with worse symptoms may benefit from a short leg walking cast for 4 to 6 weeks. Symptoms in untreated patients last longer than in treated patients (15 months vs 3 months).[2,3] Patients with persistent pain should be examined for other conditions such as talar coalition. Radiographic findings may be normal 6 to 18 months after onset and almost all patients eventually recover excellent function. The type of treatment does not alter the radiographic course of the disease or the final result.[3]

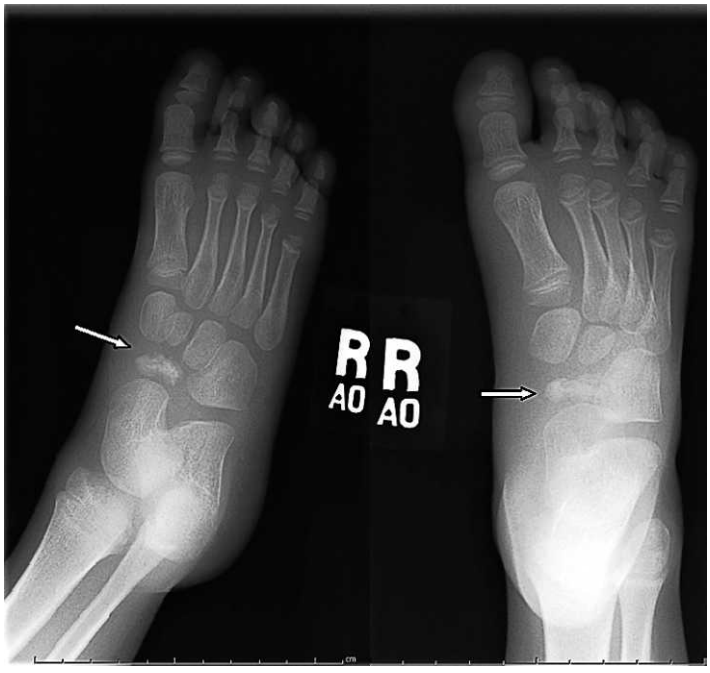

Figure. Radiograph of foot. Arrows point to the navicular bone with avascular necrosis.

Address for Correspondence: Nirav Shastri, MD, University of Missouri–Kansas City, Children's Mercy Hospital and Clinics, 5808 W 110th St, Overland Park, KS 66211. E-mail: nshastri@cmh.edu.

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Chemical Burn Secondary to Propofol Extravasation

Rahul Sharma, MD, MBA
Hana Yoshikawa, RPA-C
Josyann Abisaab, MD

New York Presbyterian-Weill Cornell Medical Center, Department of Emergency Medicine, New York, New York

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted May 29, 2011; Accepted June 10, 2011
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A 58-year-old female presented to the emergency department (ED) with pain and swelling to the right arm after receiving propofol during an outpatient procedure for nasal polyps. During the procedure, it was noted that propofol had infiltrated at the antecubital intravenous (IV) site. A new IV line was inserted, and the procedure was completed. The patient was discharged from the hospital and then presented to our ED.

Examination showed a 7-cm-by-8-cm denuded area with erythema and edema in the antecubital region (1% total body surface area). An hour after presentation, ecchymosis developed medially (Figure). There was small amount of serous drainage and tenderness to palpation. She was neurovascularly intact.

The burn service was consulted. The patient was admitted to the burn unit and treated with antibiotics. She received a skin graft and discharged on post-op day 5 without any complications.

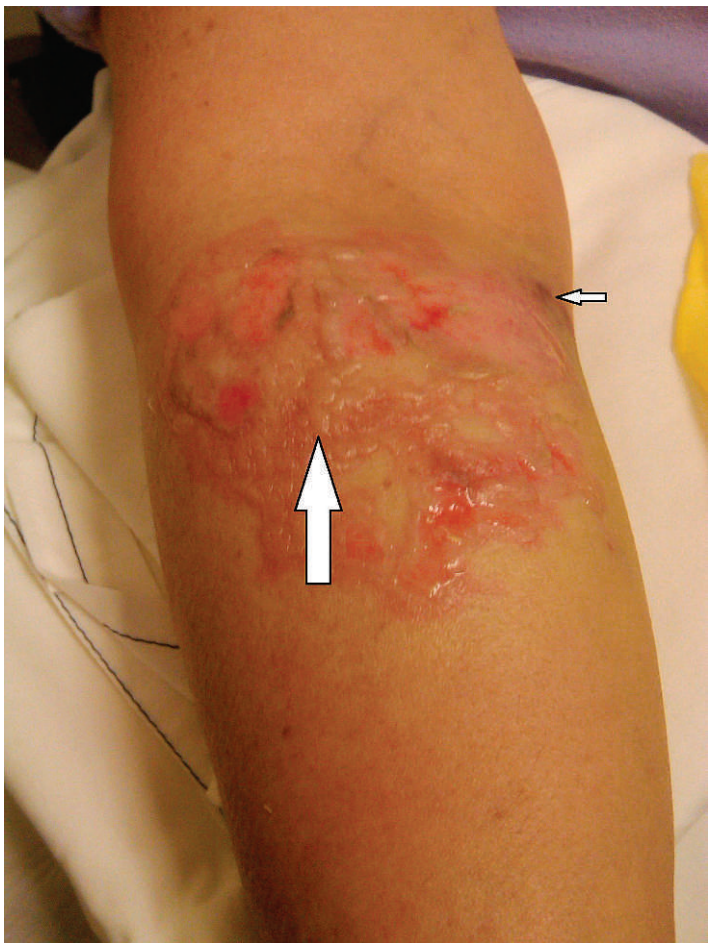

Figure. Second-degree burn of the right upper extremity. The photograph shows unroofed blisters (large arrow) with ecchymosis on the medial aspect (small arrow) of the antecubital fossa after propofol extravasation.

DISCUSSION

Propofol is a widely used anesthetic with many favorable properties, including short half-life, neutral pH, and isotonicity.[1] Owing to these factors, extravasation injuries due to propofol are relatively rare, though cases of tissue necrosis have been reported.[1–3] Risk factors for injury include cytotoxicity of the solution, infusion pressure, regional anatomical peculiarities, and other patient factors, such as preexisting cutaneous or vascular pathophysiology.[4]

When extravasation occurs, the infusion must be stopped immediately. If possible, the extravasated fluid should be aspirated before withdrawing the needle, and consider flushing with Ringer's solution or normal saline.[1,2,4] Immediate surgical consultation should be obtained. The risk for tissue damage after extravasation is often underestimated, resulting in potentially limb-threatening morbidity.[4]

Address for Correspondence: Rahul Sharma, MD, MBA, New York Presbyterian-Weill Cornell Medical Center, Department of Emergency Medicine, 525 E 68th St, Rm M-130, New York, NY 10065. E-mail: rahul_sharma@hotmail.com.

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Unilateral Internuclear Ophthalmoplegia after Minor Head Injury

Richard Bamford, MRCS(Eng)*
Gurpreet Singh-Ranger, MS, FRCS(Eng)†

* St George's Hospital and Medical School, Department of Surgery, London, United Kingdom
† William Harvey Hospital, Department of Surgery, Ashford, Kent, United Kingdom

Supervising Section Editor: Sean Henderson, MD
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Internuclear ophthalmoplegia is a rare condition caused by injury to the medial longitudinal fasciculus in the brainstem. It usually occurs in conditions such as stroke or multiple sclerosis and is extremely rare after head injury. We report a case of unilateral internuclear ophthalmoplegia, which occurred after a minor head injury in a young male. His only symptoms were headache and diplopia. He was treated conservatively, and his symptoms settled after 3 months. [West J Emerg Med. 2012;13(1):123–124.]

A 17-year-old male was admitted to our emergency unit after a head injury. He described running, and hitting the front of his head on a lamppost, with brief loss of consciousness.

On admission, he had complaints of a generalized headache and double vision, but no other symptoms. On examination, vital signs and Glasgow Coma Scale were normal, but he was unable to adduct his right eye, and had double vision in the neutral position, worse on looking toward the left (Figure 1). Findings from the cranial nerve examination were otherwise normal, and there were no other neurologic deficits or injuries found. An initial computed tomography (CT) result was reported as normal, but a subsequent magnetic resonance imaging (MRI) revealed small bilateral frontal lobe contusions (Figure 2). He was admitted, managed conservatively, and after neurosurgical review, discharged with analgesia and an eye patch. Subsequent review at 3 months showed complete resolution of his diplopia.

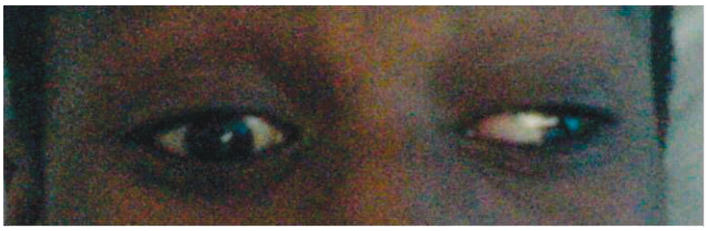

Figure 1. Photo of patient attempting to look to his left. Note inability to adduct right eye.

Unilateral internuclear ophthalmoplegia usually occurs in patients with multiple sclerosis or vascular disease[1] and is extremely rare after head injury.[2] It results from trauma to the medial longitudinal fasciculus (MLF), bundles of nerve fibres

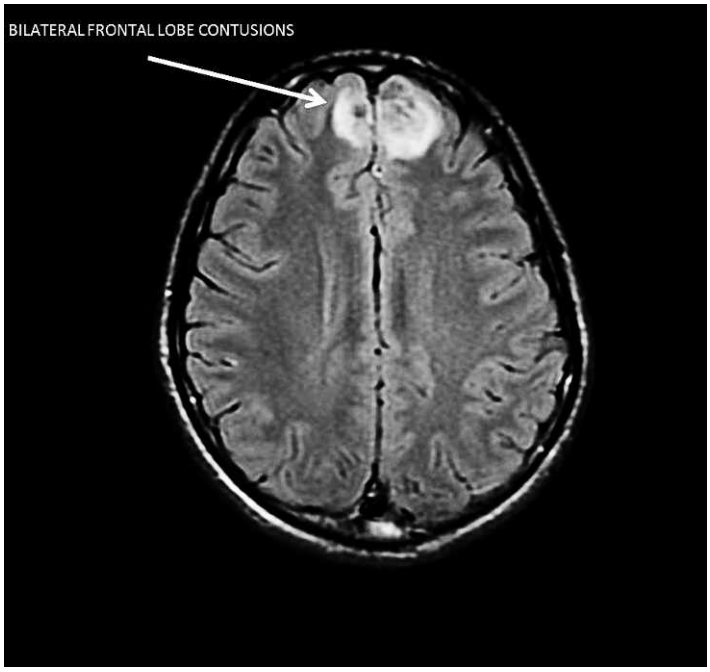

Figure 2. Magnetic resonance imaging of the brain showing frontal lobe contusions.

in the brainstem, connecting nuclei of the cranial nerves controlling head movement and directional gaze. Injury to the MLF is characterized by inability to adduct 1 eye in lateral gaze, and monocular fast-phase nystagmus of the abducting eye. It is important to note that CT imaging findings are frequently normal, and MRI is the imaging modality of choice.[3] Symptoms usually resolve with conservative management after a few months, but sometimes can persist for more than a year.[2]

Address for Correspondence: Gurpreet Singh-Ranger, MS, FRCS(Eng), William Harvey Hospital, Department of Surgery, Kennington Rd, Willesborough, Ashford, Kent, United Kingdom, TN24 0LZ. E-mail: gsinghranger@yahoo.co.uk.

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Cervical Ectopic Pregnancy

Jag S. Heer, MD
Denver K. Chao, MD
Rick A. McPheeters, DO

Kern Medical Center, Department of Emergency Medicine, Bakersfield, California

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted April 18, 2011; Revision received May 5, 2011; Accepted May 9, 2011
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A 31-year-old, who is gravida 2 para 1 at 6 weeks by last menstrual period, presents for vaginal bleeding starting approximately 5 days earlier. The bleeding was initially light, and there was no associated abdominal pain. The bleeding seemed to stop when she laid down and increased upon standing. Past medical, surgical, and social histories were unremarkable. On exam, vital signs were normal. Pelvic exam showed blood at the external orifice of the uterus, no cervical motion tenderness or adenexal tenderness. The remainder of the exam was unremarkable. Quantitative serum human chorionic gonadotropin was 7,470. Transabdominal and transvaginal bedside ultrasounds are shown in Figures 1 and 2, demonstrating an hour-glass deformity of the cervix. Fetal heart tones were present. The findings are consistent with cervical ectopic pregnancy.

DISCUSSION

Cervical pregnancy is a rare, life-threatening form of ectopic pregnancy occurring in approximately 1:9,000 pregnancies. The majority of women with a cervical pregnancy are those of low parity. Thus the current treatment trend is to preserve their reproductive function. The major predisposing factor seems to be dilatation and curettage, but others include previous cesarean delivery and in vitro fertilization. Asherman's syndrome, prior instrumentation or therapeutic abortion use, infertility, and prior ectopic pregnancies have also been implicated as predisposing factors.[1] The hallmark of the presentation is profuse, painless vaginal bleeding. Lower abdominal cramping is conspicuously absent and is found in less than one third of patients. Findings on bimanual exam include a soft, disproportionately large cervix compared to the uterus or an hour-glass shaped uterus. Sonography has led to the correct diagnosis in 81.8% of patients. Emergency physicians should have a high index of suspicion for cervical ectopic pregnancy with a low-lying gestation. Early diagnosis allows early intervention, increasing the likelihood of successful conservative treatment.[1,2] Our patient received conservative treatment with methotrexate and intra-amniotic injection of potassium chloride. She did well and was discharged 2 days later.

Address for Correspondence: Jag S. Heer, MD, Kern Medical Center, Department of Emergency Medicine, 1700 Mount Vernon Ave, Bakersfield, CA 93306. E-mail: jheer@bak.rr.com.

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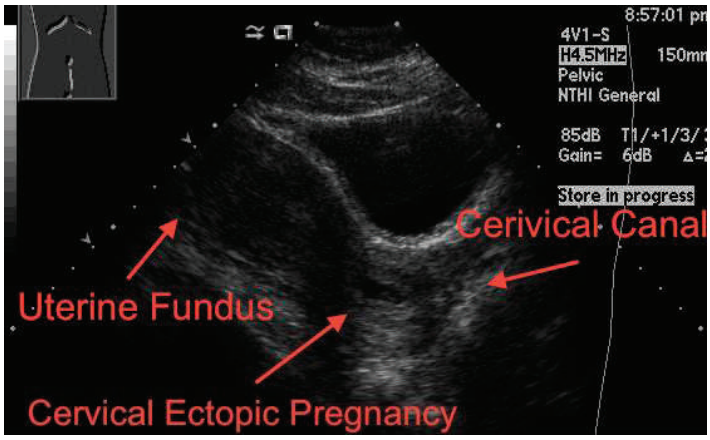

Figure 1. Transabdominal ultrasound in longitudinal plane.

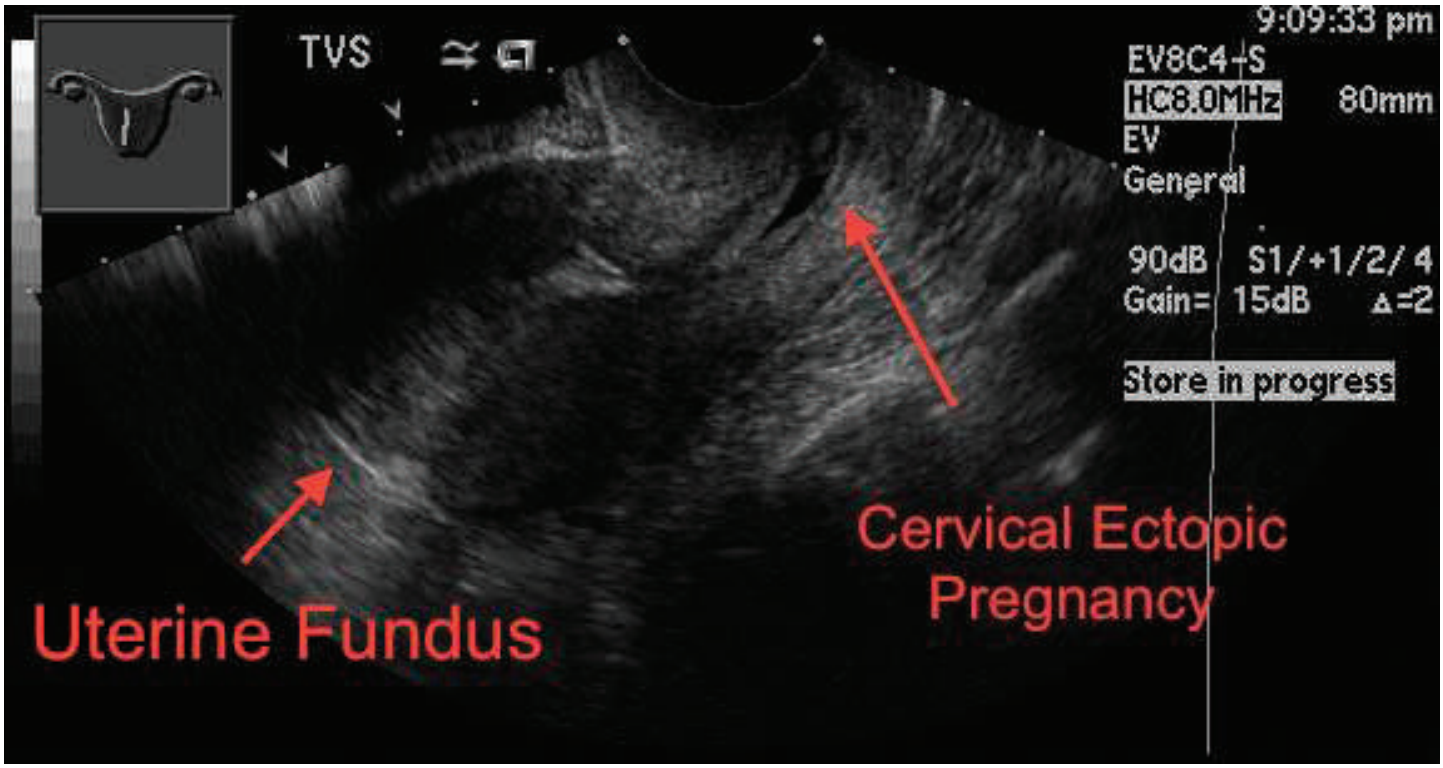

Figure 2. Transvaginal ultrasound in longitudinal plane.

Erosion of Embolization Coils into the Renal Collecting System Mimicking Stone

Jason Phan, MD*
Chandana Lall, MD*
Ross Moskowitz, MD†
Ralph Clayman, MD†
Jaime Landman, MD†

* University of California Irvine, Department of Radiology, Orange, California
† University of California Irvine, Department of Urology, Orange, California

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Urinary tract interventions can lead to multiple complications in the renal collecting system, including retained foreign bodies from endourologic or percutaneous procedures, such as stents, nephrostomy tubes, and others. We report a case of very delayed erosion of embolization coils migrating into the renal pelvis, acting as a nidus for stone formation, causing mild obstruction and finally leading to gross hematuria roughly 18 years post transarterial embolization. History is significant for a remote unsuccessful endopyelotomy attempt that required an urgent embolization. [West J Emerg Med. 2012;13(1):127–130.]

CASE REPORT

The patient is a 51-year-old Turkish man who recently presented to our institution with acute left flank pain and hematuria. Past history is significant for an atrophic nonfunctioning right kidney and a functioning left kidney with longstanding ureteropelvic junction stenosis. Since 1992, the patient has undergone an open dismembered pyeloplasty, 2 endopyelotomy attempts, and several stone treatments with ureteroscopy, holmium laser, and basket extraction.

During the second endopyelotomy attempt in 1992, a left renal subsegmental arterial branch was inadvertently traversed, resulting in acute hemorrhage and development of an arterial-ureteral fistula. This was embolized by interventional radiology with bare metal coils, with satisfactory hemostasis. A left ureteral stent was subsequently placed for the chronic ureteral obstruction.

After this procedure, the patient underwent routine ureteral stent changes every 2 years, which is a much longer period than that during which stents can typically be left in place before problems such as encrustation, infection, or obstruction are estimated to develop (2–4 months). Indwelling 7/14 French 28-cm endopyelotomy stents are used.

In 2010, the patient presented to our institution's emergency department (ED) with significant hematuria and flank pain. Computed tomography (CT) of the abdomen and pelvis without intravenous contrast (stone protocol) was performed and showed several small oval and tubular high densities in the left renal pelvis, all of which were initially interpreted as renal calculi (Figure 1). The preexisting ureteral stent appeared intact and stable since the prior CT. The patient was then brought into the operating room for stent change and stone retrieval.

Intraoperatively, at least 2 bare metal coils were observed to endoscopically erode into the left renal pelvis. Few small renal calculi were also seen, most likely due to the coils acting as the nidus. Upon further review of the CT with multiple different windowing levels, the coils could be subtly distinguished from the stones owing to their tubular appearance and configuration of the tightly wrapped loops.

DISCUSSION

Patients with complex urologic histories present a unique challenge to the evaluating physician, be it an emergency department physician or a urologist assuming the care of a new patient. A thorough review of the surgical history, and identification of potential complications or morbidities, is

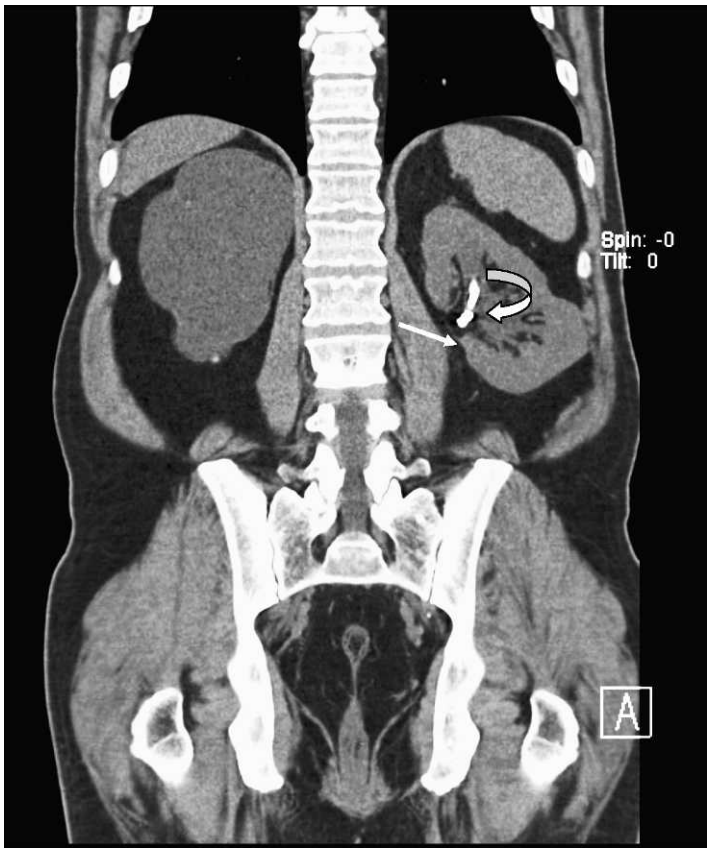

Figure 1. Coronal computed tomography in soft tissue window. Left ureteral stent (curved arrow) and eroded embolization coil (straight arrow) are noted within the left renal pelvis. On this window setting, these are difficult to tell apart.

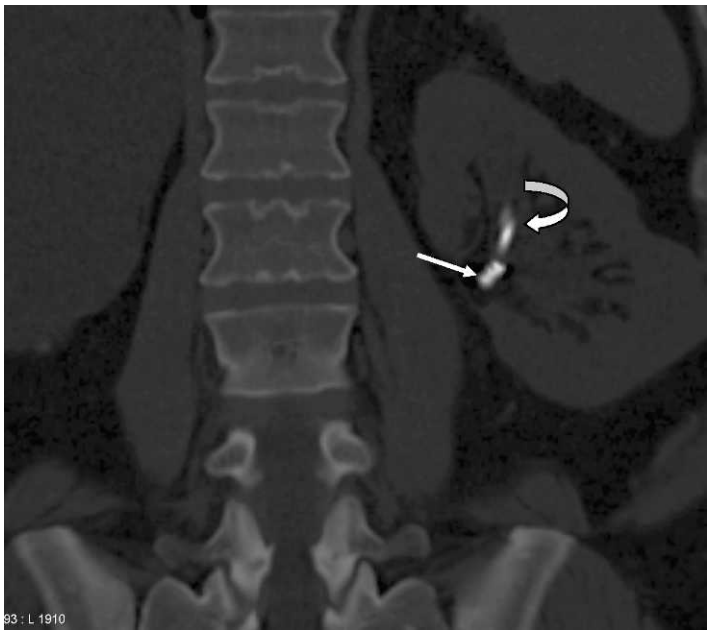

Figure 2. Importance of appropriate window levels and window widths on computed tomography. Bone windows clearly differentiate the ureteral stent (curved arrow) from the embolization coil showing surface detail (white arrow).

essential in diagnosing the acute patient. This patient presented to the ED with complaints of left flank pain and hematuria, which in most situations would be fairly diagnostic of nephrolithiasis. This patient even had a CT showing a small density in the renal pelvis and a stent in place (Figure 1), supporting this diagnosis; however in a patient with this history, the differential is lengthened.

Any patient with gross hematuria should be evaluated for possible malignancy, such as urothelial or renal cell carcinoma (RCC). Stone disease, represented by densities on CT, can exist anywhere in the urinary collecting system, even within the renal parenchyma or embedded within the wall of the ureter, which can cause stricture. As previously mentioned, retained foreign materials from stents or nephrostomy tubes are also represented as densities on CT. These include fragments, strings, or the entire implant itself, and can be found with varying degrees of encrustation. In this case, embolization coils are the culprit (Figures 2 through 5).

Procedures such as laser lithotripsy, endopyelotomy, and pyeloplasty all have inherent risks for vascular injury. Transarterial embolization is very effective in managing such vascular injury, since it has been widely used in the treatment of

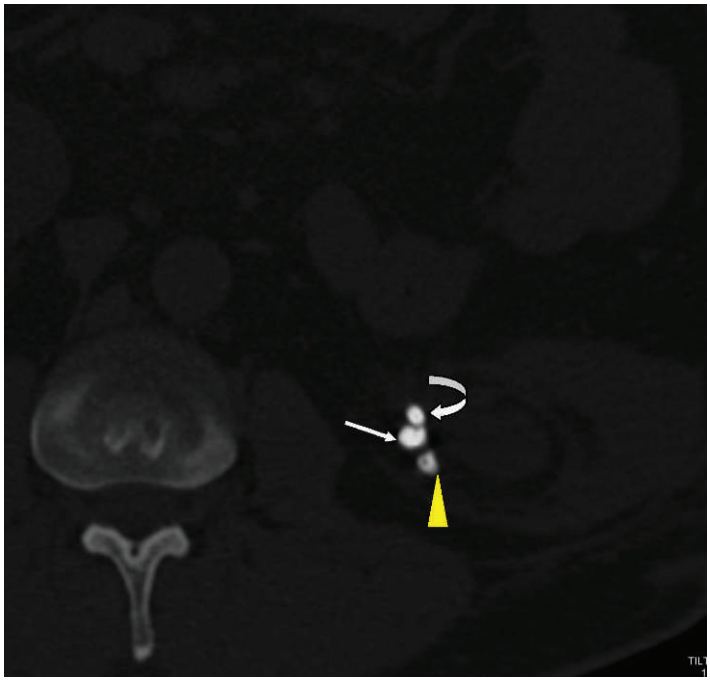

Figure 3. Axial computed tomography computed tomography (CT) in bone window: importance of appropriate window levels and window widths on CT. Bone windows clearly differentiate the ureteral stent (curved arrow) from the embolization coil (white arrow) and stone forming due to coil acting as nidus (yellow arrowhead).

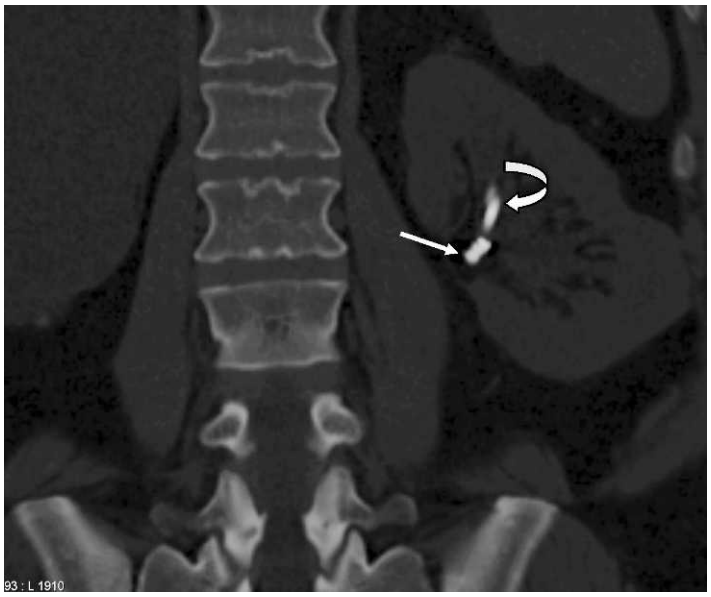

Figure 4. Coronal and curved coronal reformatted image showing different attenuation and appearance of the left ureteral stent (curved arrow) and eroded embolization coil (straight arrow).

renal arteriovenous malformation (AVM), of acute extravasation in trauma patients, and for prophylaxis against operative blood loss before surgical resection of vascular tumors such as RCC and angiomyolipoma.

Complications have been widely reported for transarterial embolization during the procedure course as well as the immediate perioperative period.[1–4] Commonly encountered complications are renal functional impairment and inadvertent embolizations of nontarget organs. Renal functional impairment often results from uremia, sepsis, and acute tubular necrosis owing to segmental parenchymal infarct.

Complications from inadvertent embolization of nontarget organ have been reported in the literature and include infarction and renal failure of the contralateral kidney; arteriovenous shunting within the targeted kidney, which can lead to embolization of the lungs and right-sided heart failure; adjacent bowel and skin necrosis; and thrombosis at the renal vein and inferior vena cava.[5–7] With the newly designed coils, frequent complications encountered in the past, such as incomplete transcatheter expulsion of the coil and withdrawal into the aorta, have been almost completely eliminated.

Coil migration at the time of insertion happens commonly, but migration in the immediate perioperative period happens much less frequently. Patients undergoing prophylactic renal artery embolization within 24 hours before nephrectomy have had migrated coils at the time of nephrectomy.[8] Delayed coil migration several years after the insertion is also very rare. Yoon et al[9] reported a case of migration of coils and guidewires from a treated renal AVM to the descending colon. Reed et al[10] reported a case of passage of coil into the collecting system at 1 year post embolization.

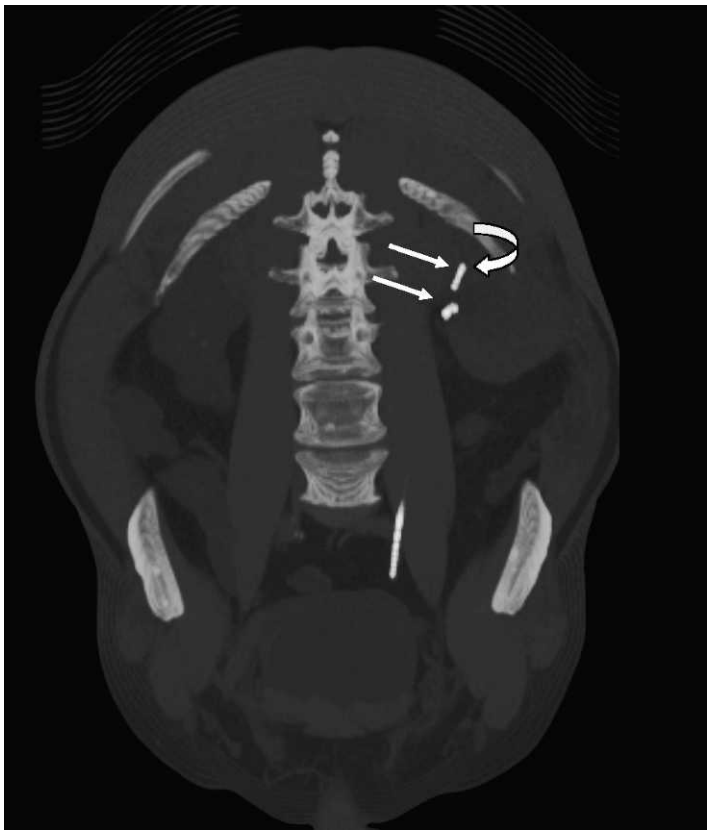

Figure 5. Coronal and curved coronal reformatted image showing different attenuation and appearance of the left ureteral stent (curved arrow) and eroded embolization coil (straight arrow).

Savoie et al[11] reported a case in which the patient passed a stone containing a platinum coil. This event happened 5 years after percutaneous nephrolithotomy and embolization of a lower polar artery branch due to persistent hematuria. This coil was initially deployed too distally and had floated within the pseudoaneurysm cavity. Such encrustation of the coil with renal calculi is compatible with reports of migrated coils acting as the nidus for stone formation.[12]

In comparison to the other case reports, the erosion and migration of the coils in this patient happened at a much later time, about 18 years later. The patient was initially treated and followed up at an outside hospital in a different state for 10 years before transferring to our institution. This case underscores the need to remain vigilant for delayed coil migration beyond the intraoperative and immediate perioperative periods, and to search for the nidus of stone formation and unusual causes of obstruction as in this case.

SUMMARY

Our report highlights the importance of checking for unusual causes of complications in patients who have undergone prior urologic intervention. The importance of viewing a stone protocol CT in different window settings cannot be overstated.

Address for Correspondence: Ross Moskowitz, MD, University of California Irvine, Department of Urology, 333 The City Blvd W, Ste 2100, Orange, CA 92868. E-mail: rmoskowi@uci.edu.

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Testicular Compromise due to Inguinal Hernia

Morgan Eutermoser, MD
Kristen Nordenholz, MD
Denver Health and University of Colorado Hospital, Department of Emergency Medicine, Denver, Colorado

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A 34-year-old male presented to the emergency department with a 3-hour complaint of pain in the right lower quadrant and right testicle. He stated that his pain began suddenly while standing at work. On physical examination, he had a small, firm, unreducible bulge in his right inguinal canal and an enlarged right scrotum. The patient was placed in trendelenburg position; intravenous fentanyl, valium, and dilaudid were administered; and surgery consult was obtained. A testicular ultrasonogram (Figure) was obtained owing to continued pain in the right scrotum and inability to evaluate the testicle. After viewing the ultrasound pattern, the patient was promptly taken to the operating room 6 hours after onset of symptoms. [West J Emerg Med. 2012;13(1):131–132.]

INDIRECT INGUINAL HERNIA WITH TESTICULAR COMPROMISE

Testicular compromise is most commonly due to testicular torsion; however, this case highlights the risk to the testicle with an incarcerated inguinal hernia. In this patient, the physical examination yielded a bulge at the inguinal canal with diffuse scrotal enlargement, tenderness, and firmness. The patient's testicle could not be evaluated, and therefore, ultrasonography was used to evaluate for concomitant torsion. The ultrasonogram revealed bowel inside the scrotal sac with no blood flow to the testicle due to compression of the spermatic cord. The spermatic cord contains 3 arteries, most notably the testicular artery, which experience reduced flow during torsion. Diagnosis of torsion can be confirmed by ultrasonography[1] if examination is questionable.

Although testicular ischemia due to hernias has been well documented in pediatric literature, only 1 case report was found demonstrating this complication in the adult patient.[2] The case report described a 48-year-old male who required only manual reduction to return flow to the testicle. If the mechanism of arterial compression is analogous to torsion, then the time to reduction and return of blood flow should be less than 6 hours to prevent permanent ischemia.[3] Operative visualization of the testicle after hernia reduction revealed a normal testicle without evidence of ischemic change, and our patient was discharged shortly after the operation.

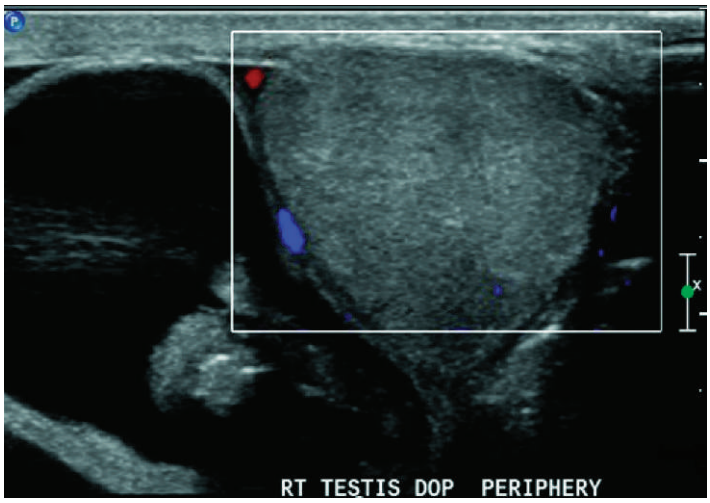

Figure. Ultrasonogram demonstrating no blood flow to the right testicle owing to the adjacent bowel within the scrotum.

Address for Correspondence: Morgan Eutermoser, MD, Denver Health and University of Colorado Hospital, Department of

Emergency Medicine, 777 Bannock St, Denver, CO 80204. E-mail: pinkstonmd@gmail.com.

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Haemophilus influenzae Sepsis and Placental Abruption in an Unvaccinated Immigrant

Paul A. Calner, MB BCh, BAO*
Megan L. Salinas, MD*
Alaina Steck, MD*
Elissa Schechter-Perkins, MD, MPH†

* Boston Medical Center, Department of Emergency Medicine, Boston, Massachusetts
† Boston University, School of Medicine, Boston, Massachusetts

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Background: *Haemophilus influenzae* infections have declined dramatically in the United States since implementation of the conjugate vaccine. However, in countries where widespread immunization is not routine, *H influenzae* remains a significant cause of morbidity and mortality. We report a case of a previously unvaccinated immigrant with confirmed *H influenzae* sepsis and placental abruption leading to spontaneous abortion.

Objectives: To alert emergency medicine practitioners that *H influenzae* should be recognized as a maternal, fetal, and neonatal pathogen. Clinicians should consider this diagnosis in immigrants presenting with uncertain vaccination history, as *H influenzae* can cause significant morbidity and mortality.

Case Presentation: A 36-year-old female was referred to our emergency department (ED) with lower abdominal pain with some vaginal spotting. The patient had an initial visit with normal laboratory investigations and normal imaging results, with complete resolution of symptoms. The patient returned to the ED with sudden onset of vaginal bleeding and abdominal pain. She presented at this time with sepsis, which progressed to septic shock, causing placental abruption and ultimately, spontaneous abortion. The patient was treated with pressors and antibiotics and was admitted to the medical intensive care unit where she received ampicillin, gentamycin, and clindamycin for suspected chorioamnionitis. The patient's blood cultures came back positive after 1 day for *H influenzae*. The patient did well and was discharged from the hospital 4 days later.

Conclusion: *Haemophilus influenzae* should be recognized as a neonatal and maternal pathogen. Clinicians should consider this diagnosis in immigrants presenting with uncertain vaccination history, especially in pregnant females, as *H influenzae* can cause significant morbidity and mortality. [West J Emerg Med. 2012;13(1):133–135.]

INTRODUCTION

Haemophilus influenzae infections have declined dramatically in the United States since the conjugate vaccine became available in 1988.[1] However, in countries where widespread immunization is not routine (including Southeast Asia, Africa, the western Pacific, and the eastern Mediterranean), *H influenzae* remains a significant cause of mortality. There were an estimated 8.13 million infections and 371,000 deaths in 2000 alone. Most deaths worldwide are in Africa and Asia; however, infections from nontypable *H influenzae* continue to pose a problem within the United States.[2] Cases of *H influenzae* infections in pregnant females have been documented in case reports, usually resulting in significant maternal and fetal morbidity.[3–8] The incidence of *H influenzae* bacteremia in pregnant females, causing sepsis and eventual spontaneous abortion, has not been well documented in the literature. We report a case of a previously unvaccinated immigrant with confirmed *H influenzae* sepsis and placental abruption, which resulted in a spontaneous abortion.

CASE REPORT

The patient was a 36-year-old female, gravida 4, para 3, pregnant at 16 weeks and 1 day (confirmed by previous ultrasonography) who presented to an ambulatory health center with complaints of 1 day of lower abdominal and flank pain with some vaginal spotting. She also endorsed a history of mild dysuria with urinary frequency and urgency at that time. She reported no fever, chills, or sick contacts. She was referred to the emergency department (ED) for further evaluation. She had no past medical history but had arrived in the United States from Pakistan 8 months earlier. Her ED vital signs showed a blood pressure of 103/70 mmHg, a heart rate of 133 beats per minute (bpm), a temperature of 99.9°F, and a respiratory rate of 16 per minute with an oxygen saturation of 99% on room air. The patient was well appearing, and her physical examination was notable for tachycardia, suprapubic tenderness without rebound or guarding, and a pelvic examination with slight bleeding but no uterine tenderness. Her laboratory results showed a leukocytosis of 16.1 K/μL with 88% polymorphonuclear leukocytes, which was considered within the normal range for the second trimester of pregnancy. The remainder of the complete blood count, chemistry panels, and urinalysis were unremarkable. An ultrasonogram of her abdomen showed a live intrauterine pregnancy with an estimated gestational age of 16 weeks and 0 days, with a fetal heart rate of 182 beats per minute. Magnetic resonance imaging (MRI) of the abdomen was interpreted as normal, without evidence of appendicitis. She received 4 liters of normal saline, 2 mg of morphine sulfate, and 975 mg of acetaminophen. Hear heart rate improved to 100 bpm and she felt better. She was seen by an obstetrician consultant in the ED and discharged home with obstetrician follow-up and instructions to return with any concerns.

She returned to the ED 1 day later with sudden onset of vaginal bleeding and abdominal pain. Her vital signs were concerning for a heart rate of 139 bpm, a blood pressure of 149/88 mmHg, a respiratory rate of 30 per minute, and oxygen saturation of 100% on room air. Further examination revealed that the patient was actively delivering the products of conception. An obstetrician was emergently consulted and arrived minutes later. They delivered an intact fetus and gestational sac shortly after their arrival. The patient received 800 mg of rectal misoprostol. A tympanic temperature at this time was measured at 105°F. The patient was persistently tachycardic into the 140s. The patient became hemodynamically unstable with a blood pressure of 84/35 mmHg and central intravenous access was obtained. The patient was given acetaminophen, intravenous normal saline, and given vancomycin and piperacillin/tazobactam, with a working diagnosis of sepsis of unknown etiology. Her laboratory results revealed a venous pH of 7.22, an anion gap of 21 with a lactate concentration of 10.8 mmol/L. Her complete blood count at this time showed a white blood cell count of 14.9 K/μL and 85% polymorphonuclear leukocytes. The patient had normal MRI and urinalysis findings and no symptoms to suggest colitis, skin infection, or other soft tissue infections. The patient was given norepinephrine for persistent hypotension and admitted to the medical intensive care unit where she received ampicillin, gentamycin, and clindamycin for suspected chorioamnionitis. An ultrasonogram was suggestive of retained products of conception and that night the patient underwent dilatation and curettage with removal of tissue debris. The patient did well and was discharged from the hospital 4 days later.

The patient's blood cultures came back positive after 1 day for *H influenzae*, non–β-lactamase producing, although further identification of the molecular characteristics of the bacteria, which requires slide agglutination for serotyping or polymerase chain reaction for capsular typing, was not performed. The pathology report showed retroplacental hemorrhage occupying 20% of the placental disc and adjacent infarct, consistent with placental abruption. There was no histologic evidence of acute chorioamnionitis.

DISCUSSION

Placental abruption is the separation of a normally implanted placenta due to decidual hemorrhage before delivery of the fetus. The incidence of placental abruption is approximately 1 in 100 births and accounts for 10% to 15% of perinatal mortality.[9–11] Acute placental abruption can cause significant maternal and fetal compromise; the risk to the fetus depends on the severity of the abruption and the gestational age at which the abruption occurs, and the danger to the mother is primarily dependent on the degree of abruption.[9,11] Abruption is associated with a ninefold increased risk for stillbirth.[9] Although there has been significant epidemiologic and clinical research into the causes of placental abruption, the underlying etiology and sequence of events at a molecular level are still not well understood. A number of risk factors for placental abruption have been identified, including maternal age and parity, cigarette smoking, hypertension, preeclampsia, and intrauterine infection.[11] The role of nonintrauterine maternal infection is less clear, but there is circumstantial and animal-model evidence to suggest that it is implicated in at least a portion of preterm pregnancy complications, and intrapartum fever has been found to be associated with increased risk of placental abruption as well.[12,13] Given the lack of other risk factors for placental abruption and the temporal relationship of the patient's sepsis and abruption, it is likely that sepsis secondary to an acute *H influenzae* infection led to placental abruption and ultimately, spontaneous abortion.

Haemophilus influenzae, primarily serotype B, causes serious invasive diseases, usually in children younger than 5 years.[14] Hib conjugate vaccines were first licensed for children in the United States in 1988, with subsequent licensure in 1990. Since implementation, the number of reported Hib invasive disease cases among children younger than 5 years has declined 99%.[1] Nontypable *H influenzae* is distinguished from the serotyped strains by the absence of a polysaccharide capsule. Nontypable strains cause invasive disease in children less frequently than encapsulated *H influenzae*, but are increasingly recognized as pathogens in adults, particularly for the

immunocompromised person.[15,16] Nontypable *H influenzae*, (particularly biotype 4) can colonize the genital tract of women. It can cause significant neonatal disease, including sepsis and pneumonia, owing to vertical transmission, as well as postpartum maternal sepsis with endometritis, tuboovarian abscess, and chronic salpingitis.[17] There is a sixfold increased risk for *H influenzae* bacteremia in pregnant women aged 18 to 39 years compared with other adults of the same age. Over half of the pregnancies associated with bacteremia in 1 prospective study resulted in fetal death, and a retrospective study found that 65% of fetuses were infected if mothers were, with a mortality rate of 44% in fetuses that were infected.[15,4]

Emergency physicians are frequently required to treat infections before the specific bacterial pathogen is identified. Maternal/fetal sepsis of unclear etiology ought to initially be treated with broad-spectrum antibiotics to empirically cover likely potential infective agents. When diagnostic testing has confirmed infection is due to *H influenzae,* treatment can be tailored to this specific organism. β-Lactam antibiotics, such as amoxicillin, have generally been considered first-line agents against *H influenzae*, although there have been reports both in the United States and worldwide of significant β-lactamase resistance including resistance to amoxicillin-clavulonate,[18,19] requiring a second- or third-generation cephalosporin. Alternative choices of antibiotics with activity against *H influenzae* include macrolides, aminoglycosides, fluoroquinolones, and tetracyclines.

CONCLUSION

Although the epidemiology of *H influenzae* is changing both in the United States as well as worldwide, owing to the Hib vaccine, there is still significant morbidity from this illness.[16,20] The illness described in our patient exemplifies how *H influenzae* should be recognized as a maternal, fetal, and neonatal pathogen. Clinicians should consider this diagnosis in immigrants presenting with uncertain vaccination history, especially in pregnant females, as *H influenzae* can cause significant morbidity and mortality.

Address for Correspondence: Paul A. Calner, MB Bch, BAO, Boston Medical Center, Department of Emergency Medicine, One Boston Medical Center Pl, Boston, MA 02118. E-mail: Paul.Calner@bmc.org.

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Dermatomyositis with Extensive Calcification in an Adult

Bradley C. Presley, MD
Jeffrey S. Bush, MD
Simon C. Watson, MD

Medical University of South Carolina, Division of Emergency Medicine, Charleston, South Carolina

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This report reviews a case of dermatomyositis presenting with weakness and extensive calcification in an adult. While dermatomyositis is not uncommon in adults, it is uncommon for calcifications to be present. Children develop calcifications more frequently than adults. When present in adults, small calcifications on areas of frequent trauma such as elbows and fingers are more common. However, this patient presented with large calcified deposits in his abdomen and extremities. His treatment and course are described. [West J Emerg Med. 2012;13(1):136–138.]

CASE REPORT

A 26-year-old Hispanic male presented to the emergency department with progressive weakness during the previous year. This weakness initially started in his lower extremities, when he noticed difficulty when ascending stairs; however, it progressed to involve his upper extremities in the following months. He also had complaints of joint pain, generalized fatigue, "bumps," and "hard places" over his abdomen and thighs, as well as a rash that was more pronounced on his face, arms, and legs. Of note, he had been admitted to another hospital several months before and treated with aggressive hydration for rhabdomyolysis. He improved slightly after that admission, but the weakness subsequently progressed to the point that he was unable to ambulate on his own power. He was given a prescription for an unknown medication on discharge from his previous hospitalization but it was never filled. His past surgical history was significant for an appendectomy about 1 year prior; his family history was unremarkable.

On physical examination, the patient had normal vital signs. He was noted to have a diffuse hyperpigmented rash over his face, more prominent on his cheeks and other sun-exposed portions of his body. He was also noted to have taut skin and a firm, nonmobile, tender mass in his right lateral abdominal and flank area that began at the costal margin and continued into his right pelvis. This firm area extended from the posterior axillary line to almost the midline of his abdomen. He had other similar but smaller masses in his left upper and lower quadrants as well as on both thighs. On examination, the patient was unable to raise his arms above his head without assistance. His neurologic examination showed intact sensation and reflexes throughout with marked weakness in his extremities and trunk. He demonstrated 2–3/5 strength in both legs proximally with 4/5 strength in his upper extremities. He had difficulty sitting up without assistance.

His laboratory tests in the emergency department included a basic metabolic panel that was within normal limits, with normal calcium levels. The creatine kinase (CK) level, however, was elevated to 3,501 IU/L, while serum aldolase was 29.7 U/L (reference, 1.5–8.1 U/L) and lactate dehydrogenase (LDH) was 728 IU/L (reference, 100–240 IU/L). He had a mild transaminitis with an aspartate aminotransferase level of 207 IU/L and an alanine aminotransferase level of 148 IU/L. A computed tomography of the abdomen and pelvis was completed. The official read commented on calcifications in the right rectus abdominis muscle, external oblique, and subcutaneous fat, as well as calcifications in the left external oblique muscle and left rectus abdominis. The patient was admitted for further management.

In the hospital, he was aggressively hydrated and given high-dose steroids as well as methotrexate (Trexall; Teva Pharmaceuticals USA, North Wales, Pennsylvania). Rheumatology service was consulted, and a muscle biopsy confirmed the diagnosis of dermatomyositis with calcinosis cutis. The patient was then given azathioprine (Imuran; Prometheus Laboratories Inc, San Diego, California) and he showed continual improvement throughout his hospital course.

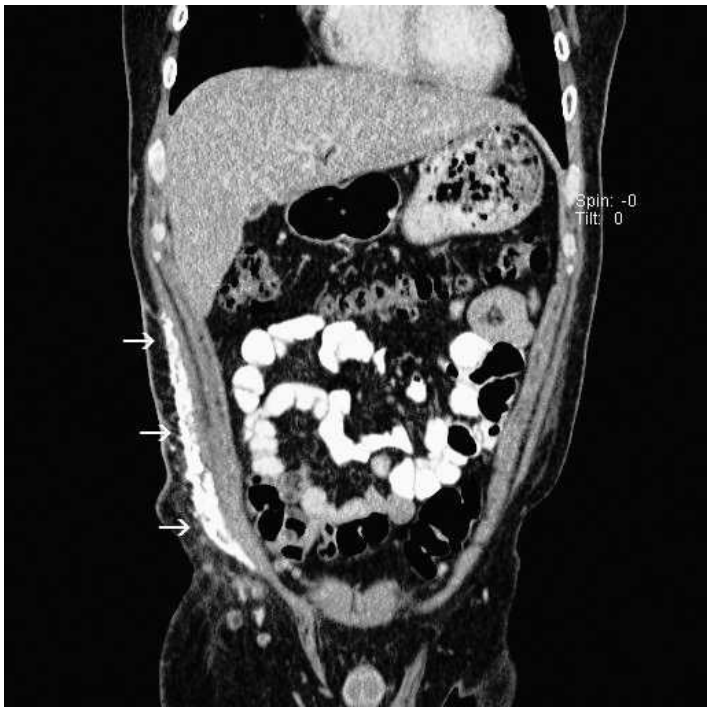

Figure 1. Coronal view of computed tomography showing extensive calcification of right flank.

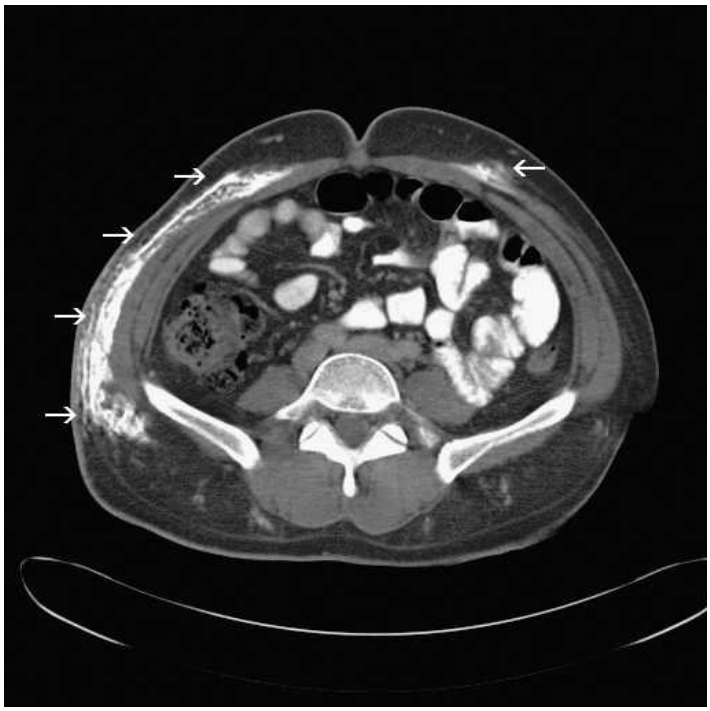

Figure 2. Sagittal view of computed tomography showing right-sided calcification as well as small calcific foci on left anterior abdomen.

His CK levels improved with hydration, and he received intensive physical therapy throughout his hospitalization. At hospital discharge, the patient had 4/5 strength globally and was able to sit in a chair unassisted. He continues to work toward ambulation through outpatient physical therapy.

DISCUSSION

Dermatomyositis is a disease that can present in individuals of all ages, with peak incidence in adults during the fifth and sixth decades of life. It has an incidence of 5.5 per million people. The exact mechanism for dermatomyositis is not known, but it is postulated to have an autoimmune component. Some cases are felt to be paraneoplastic. In most cases, the rash and proximal weakness appear simultaneously. However, 30% of patients experience the cutaneous symptoms without weakness (dermatomyositis sine myositis), and 10% have muscle weakness without cutaneous symptoms. Initial cutaneous symptoms often include burning and pruritus, which may be associated with exposure to ultraviolet light or sunlight. Muscle weakness is characterized predominantly by proximal hip and shoulder muscle involvement: patients may have complaints of difficulty standing from a sitting position or raising their arms above their heads. They also have complaints of pain and tenderness to their muscles.[1]

Laboratory tests to support the diagnosis of dermatomyositis include serum muscle enzyme concentrations as well as autoantibody tests. Often, CK, LDH, aldolase, and aminotransferases are elevated from muscle breakdown. Patients usually have autoantibodies ranging from nonspecific antinuclear antibodies to the more specific anti–155 kDa. Electromyography (EMG) is characterized by increased irritability with spontaneous fibrillation and sharp waves. Often, skin and muscle biopsies show inflammatory changes, segmental necrosis, or other nonspecific findings. The diagnosis is confirmed through clinical history and examination of proximal muscle weakness with skin findings and 2 of 3 laboratory criteria. These include elevated muscle enzymes, EMG changes, and tissue biopsy, as described above.[1]

Although this patient had the typical findings of dermatomyositis, with confluent photosensitive rash over the malar area of his face and proximal muscle weakness, he also suffered from extensive calcinosis. While described as a complication of dermatomyositis in pediatric and adolescent patients, calcinosis is much less common in adult patients. Among the few cases seen in adults, calcinosis is often located in hard deposits around areas that experience frequent trauma (elbows and fingers).[2] Socioeconomic status may play a role in the progression of calcinosis, as demonstrated in case reports[3]; this patient is a migrant worker without insurance. He demonstrated problems with timely follow-up to ensure he was getting the appropriate medicines after his initial diagnosis of rhabdomyolysis.

This patient developed extensive calcifications in the subcutaneous tissue of his right flank (Figure 1) and abdomen (Figure 2). This area is not typically affected in adults; however, the trauma from his appendectomy may have initiated an

inflammatory response in this region. This inflammation likely precipitated the formation of the calcium deposits in his abdominal wall with resultant calcinosis.[4]

Treatment is largely based on controlling the likely autoimmune component of the disease. One possible etiology is complement-mediated inflammation at the vascular level; another is a direct cytotoxic effect of lymphocytes on the muscle cells. Initial therapy consists of high-dose steroids. Immunosuppressant and cytotoxic agents are often given early in the course in order to wean off steroids, thereby limiting the toxic effect of chronic steroids. Methotrexate, azathioprine, and mycophenolate mofetil are common agents used in dermatomyositis. If this combination of drugs is unsuccessful, intravenous immunoglobulins have shown promise for short-term treatment.[5]

Calcinosis is a difficult complication to treat. Some studies have shown success with diltiazem, aluminum hydroxide, and even alendronate in children.[6,7] However, refractory cases of calcinosis that cause pain or interfere with function may need to be referred for surgical excision.[8]

Treatments for the rash are first focused on controlling systemic processes, but providing protection is also extremely important through limiting sun exposure and using sun protective clothing and sunscreen.

Current theories indicate calcinosis may be a consequence of untreated or unaggressively treated dermatomyositis. In juvenile dermatomyositis, early aggressive intervention offers the best protection from development of calcinosis. This adult patient had been experiencing symptoms for about 14 months before he received aggressive treatment, most likely another factor in his development of calcinosis.[3] This patient suffered to the point at which he was no longer able to complete his activities of daily living. Luckily, he showed rapid signs of improvement with high-dose steroids and azathioprine. He was given prednisone and azathioprine also as an outpatient with continued improvement in his symptoms. Initially, intravenous immunoglobulin was considered because of the severity and progressive nature of his symptoms; however, it was never given owing to his response to other medicines.

The overall prognosis for patients treated with dermatomyositis is good, with a 5-year survival rate of 95% and a 10-year survival rate of 84%. Those who die from the condition often have pulmonary or cardiac manifestations as well. While most persons with dermatomyositis improve and respond to therapy, up to 30% experience long-term consequences.[9] At a 2-month follow-up appointment, the patient was ambulating without assistance and was able to complete activities of daily living. He still reports some difficulty in standing from low sitting positions but continues to improve as his disease process is better controlled. The patient continues to take immunosuppressants and diltiazem. His calcifications remain, but his pain and symptoms are controlled.

Address for Correspondence: Bradley C. Presley, MD, Medical University of South Carolina, Division of Emergency Medicine, 169 Ashley Ave, MSC 300, Charleston, SC 29425. E-mail: brad.presley@gmail.com.

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