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Comparison of the Evaluations of a Case-Based Reasoning Decision Support Tool by Specialist Expert Reviewers with Those of End Users

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Background: Decision-support tools (DST) are typically developed by computer engineers for use by clinicians. Prototype testing DSTs may be performed relatively easily by one or two clinical experts. The costly alternative is to test each prototype on a larger number of diverse clinicians, based on the untested assumption that these evaluations would more accurately reflect those of actual end users.

Hypothesis: We hypothesized substantial or better agreement (as defined by a κ statistic greater than 0.6) between the evaluations of a case based reasoning (CBR) DST predicting ED admission for bronchiolitis performed by the clinically diverse end users, to those of two clinical experts who evaluated the same DST output.

Methods: Three outputs from a previously described DST were evaluated by the emergency physicians (EP) who originally saw the patients and by two pediatric EPs with an interest in bronchiolitis. The DST outputs were as follows: predicted disposition, an example of another previously seen patient to explain the prediction, and explanatory dialog. Each was rated using the scale Definitely Not, No, Maybe, Yes, and Absolutely. This was converted to a Likert scale for analysis. Agreement was measured using the κ statistic.

Results: Agreement with the DST predicted disposition was moderate between end users and the expert reviewers, but was only fair or poor for value of the explanatory case and dialog.

Conclusion: Agreement between expert evaluators and end users on the value of a CBR DST predicted dispositions was moderate. For the more subjective explicative components, agreement was fair, poor, or worse.
[*West*JEM. 2008;9:74-80.]

INTRODUCTION

Decision-support tools (DST) are typically developed by computer engineers who rely heavily on feedback from clinicians as they build and test the DST prototypes. Often developers will collaborate with one or two clinicians with a particular expertise in the field for which the DST is being targeted. An alternative approach is to test each prototype on a larger number of diverse clinicians, anticipating that these evaluations of the evolving DST will more accurately reflect those of actual end users. This latter approach is logistically far more difficult than the former, adding time and expense to the development of DSTs. Furthermore, the underlying assumption that testing a DST on a larger number of clinicians is better is an untested one.

To test this assumption we compared the evaluations of a DST performed by two sub-specialists with a particular interest in the field targeted by the DST with those of 12 other clinicians. We did this using a case-based reasoning (CBR) tool designed to predict the disposition of children with bronchiolitis. This DST provides three distinct outputs. It predicts disposition. It provides an example of a previously treated patient and that patient's outcome from a database of previously treated patients as evidence supporting its prediction. It provides an explanatory dialog to 'explain' its decision.

We hypothesized substantial or better agreement (as defined by a κ statistic greater than 0.6) between the evaluations of the DST performed by the clinically diverse end users, to those of two sub-specialist reviewers who evaluated the same DST output.

METHODS

The study was approved by our Institutional Review Board. A CBR tool for use in infants with bronchiolitis was developed and prospectively tested in an academic emergency department. This has been described in detail elsewhere.[1] Briefly, the DST compares the patient presented to it with previous patients in its database. It uses nine clinical features, including response to treatment to match the patient as closely as possible to a previous patient for whom the clinical outcome is known. These clinical features are shown in the appendix, which gives a sample DST output. Based on a previously treated patient in the database whose outcome is known, the DST predicts the current patient's disposition. It then presents the case from its database that most supports the prediction and generates a dialog comparing and contrasting the previously seen patient and the current patient.

Following enrollment, a detailed history and physical exam was performed on each child and the results recorded on a specifically mandated data-collection sheet. This information was entered in a customized Filemaker-pro database.[2] The DST extracted the data points it required directly from this database. To prevent the DST from influencing a clinician's disposition decisions, we delayed presenting the DST printed output until the clinician's next shift. Each physician was asked to rate the usefulness of each of the three components of DST output (disposition, case to justify the disposition, and explanatory dialog) using the scale Definitely Not, No, Maybe, Yes, and Absolutely. This was converted to a Likert Scale from 1 to 5 respectively for analysis. An example of this output is shown in Appendix 1. We also analyzed the data compressing the ordinal five-point Likert scale to a three-point scale, as two people could mean nearly exactly the same thing by 'No' and 'Definitely not.'

Two pediatric EPs, both of whom have previously published research on bronchiolitis, acted as the expert reviewers. These experts reviewed the data collection sheet DST output and the CBR-DST output in the same manner as the original end users.

One of these reviewers also performed a blinded review of the cases without the DST output to provide some measure of disagreement that could be attributed solely to the use of chart review rather than due to disagreement with the DST output. This was performed four months before review of the DST output to minimize recall bias.

Severity of illness of the patients was calculated using the NCH bronchiolitis severity model.[3] A predominance of mildly or severely ill patients would render a DST less useful and potentially could affect physicians' evaluations of it.

Inter rater agreement was calculated using a weighted kappa (κ) statistic. The κ statistic was interpreted as recommended by Landis and Koch.[4] Confidence intervals for the weighted κ statistic were calculated using a bootstrap technique.[5] Mean scores, their distribution and interquartile ranges (IQR) for the end users and the expert evaluators, were calculated. Overall distributions of scores were compared using the non parametric sign rank test. Statistical analysis was performed using Stata 9.2 software.

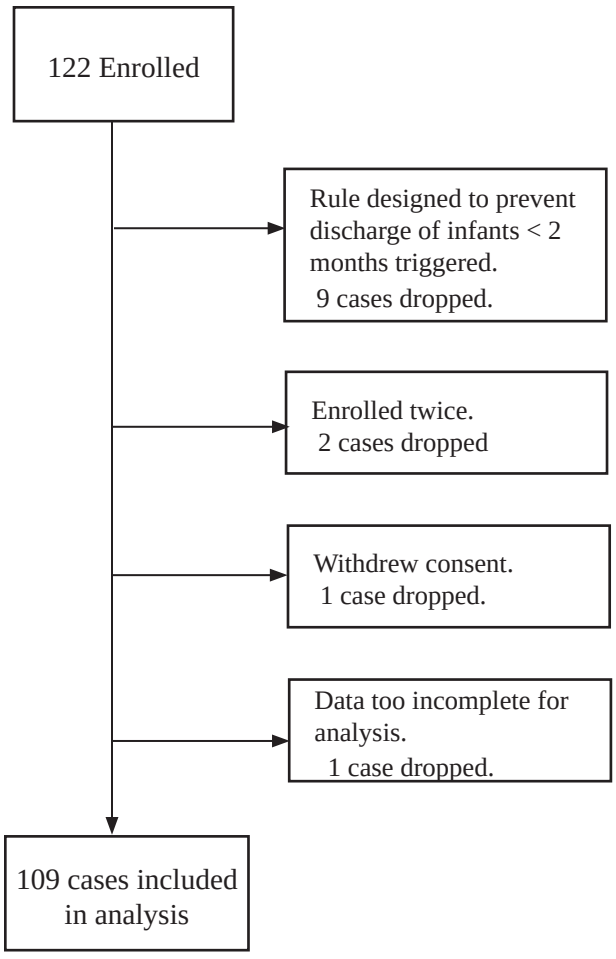

Figure 1. Case flow through the study.

Table 1. Agreement between evaluators on the predicted disposition. The values in parentheses are the results obtained when the five categories are collapsed to three.

CBR DST predicted disposition: Do you agree with the suggested course of action?					
Evaluator 5 point scale (3 point scale)	**Observed Agreement**	**Agreement expected by chance alone**	**κ**	**95% C.I.**	**Interpretation**
End users & Expert 1	93.5% (89.9%)	87.2% (79.6%)	0.49 (0.51)	0.25 - 0.69 (0.25 - 0.71)	Moderate (Moderate)
End users & Expert 2	93.6% (91.6%)	86.4% (79.9%)	0.53 (0.58)	0.33 - 0.68 (0.36 - 0.76)	Moderate (Moderate)
Expert 1 & Expert 2	94.5% (91.6%)	87.3% (80.9%)	0.56 (0.56)	0.38 - 0.70 (0.33 - 0.74)	Moderate (Moderate)

Table 2. Agreement between evaluators on the value of the explanatory case. The values in parentheses are the results obtained when the five categories are collapsed to three.

CBR DST explanatory example: Did you find the explanation case useful?					
Evaluator 5 point scale (3 point scale)	**Observed Agreement**	**Agreement expected by chance alone**	**κ**	**95% C.I.**	**Interpretation**
End users & Expert 1	89.2% (70.8%)	83.0% (58.4%)	0.36 (0.30)	0.19 - 0.53 (0.14 - 0.46)	Fair (Fair)
End users & Expert 2	83.96% (46.0%)	84.3% (43.2%)	-0.02 (0.05)	-0.10- 0.04 (0.04 - 0.14)	None* (Poor)
Expert 1 & Expert 2	87.03% (59.2%)	87.3% (52.6%)	-0.01 (0.14)	-0.08 - 0.07 (0.03 - 0.26)	None* (Poor)

Table 3. Agreement between evaluators on the value of the explanatory dialog. The values in parentheses are the results obtained when the five categories are collapsed to three.

CBR DST explanatory dialog: Did you find the supporting dialog useful?					
Evaluator 5 point scale (3 point scale)	**Observed Agreement**	**Agreement expected by chance alone**	**κ**	**95% C.I.**	**Interpretation**
End users & Expert 1	87.2% (66.0%)	83.6% (56.7%)	0.21 (0.21)	0.03 - 0.40 (0.05 - 0.38)	Fair (Fair)
End users & Expert 2	84.1% (62.0%)	83.4% (58.7%)	0.04 (0.08)	(0.13 - 0.22) (-0.10 - 0.26)	Poor (Poor)
Expert 1 & Expert 2	78.3% (60.3%)	79.2% (56.9%)	-0.04 (0.08)	-0.20 - 0.12 (-0.09 - 0.25)	None (Poor)

RESULTS

One hundred and twenty-two patients were enrolled. Patient flow and exclusions are shown in Figure 1. Following exclusions, 109 remained in the analysis. Expert reviewer evaluations were available for all of these. Attending physicians performed end-user evaluations on 97 of the CBR predictions of disposition and 96 of the CBR explanatory dialogs. Midlevel providers performed the evaluations on 12 cases, three of which had no attending evaluations.

The mean number of years following residency training for the faculty was nine (range one to 28). All were board prepared or certified and all but one residency trained in emergency medicine.

Severity of illness and age characteristics are shown in

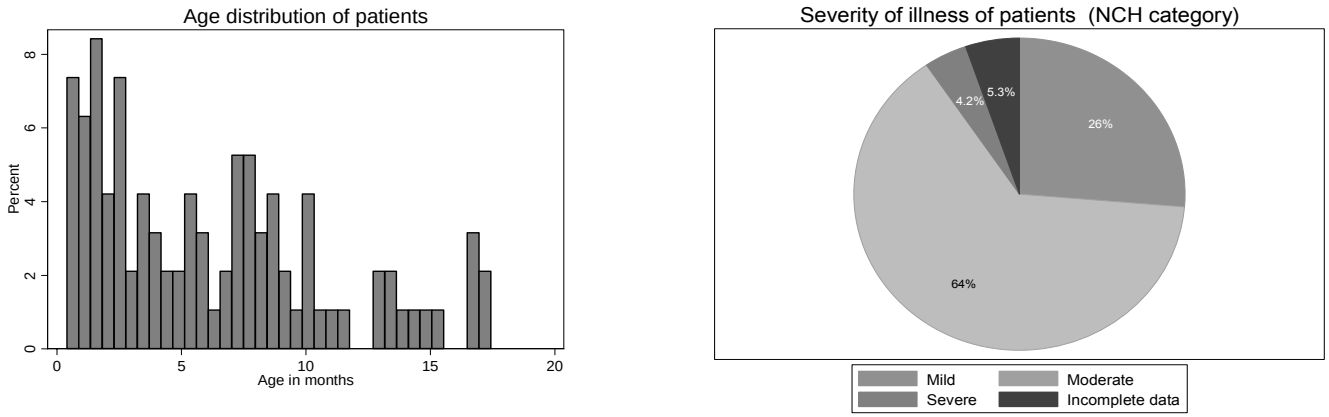

Figure 2. Age and severity of illness of patients.

Figure 2 and showed a broad range of cases. The expert reviewer who performed the chart review agreed with the disposition of the end user 93/109 (85.3%) of the time (expected by chance alone 50%) $\kappa = 0.66$ (95% CI 0.53 to 0.80) demonstrating moderate agreement.

The raw scores and their distribution for the evaluations are shown in Figure 3. Agreement between the end users and expert reviewers and the reviewers with each other are shown in Tables 1 to 3.

DISCUSSION

We found moderate agreement between our expert reviewers and actual end users for disposition, but this decreased progressively as the inherent subjectivity of the DST output being evaluated increased. The expert reviewers did not agree any more with each other than they did with the end users when the case was more ambiguous. For DST developers this is disheartening as it suggests that when developing these tools prototype testing requires feedback from a group representative of actual end users rather than one or two interested clinical experts. This former approach to DST development is logistically much more difficult and costly to perform than the latter. The silver lining for developers was that the end users consistently scored the DST more highly than did the expert reviewers.

LIMITATIONS

The management of bronchiolitis is inherently controversial,[6-8] and some disagreement between clinicians on disposition is to be expected leading to an immeasurable random bias towards poorer agreement in disposition and presumably DST output. On the other hand, it is precisely for such less than clear-cut conditions that CBR may offer some benefit. The use of chart review by the expert reviewers introduces potential bias to decreased agreement. Eliminating this systematic bias would require that the patients were independently seen by both the treating clinician and the expert reviewer at the same time. Such a methodology is unlikely to be feasible in emergency medicine. We addressed this by having one expert perform an initial blinded review of disposition, at least providing some measure of the effect of this. Agreement for this was 85% (compared with 50% expected by chance alone), suggesting that this effect was relatively modest. It implies a methodologically introduced potential upper limit of substantial agreement (κ=0.6 to 0.8) for what might be obtained between the end users and expert reviewer by virtue of the use of chart review by the experts. This is important; the observed agreement was moderate (κ=0.49) for the DST-predicted disposition, suggesting that for this outcome at least the agreement may be better than it appears after initial review.

The role of chance in leading to artificially poor agreement between DST users is minimized by using larger numbers of patients and clinicians. While the number of clinicians involved in the study is relatively small, it strikes a balance between having too few evaluations carried out by many evaluators and the feasibility of obtaining a reasonable sample size of patients. This was particularly the case for our study, which required written informed parental consent for every patient.

The generalizability of our work is limited because we considered a single DST at a single site. We have previously noted a higher discharge rate (with more discharge failures) at our site compared with a second ED. We addressed this in part by using an expert reviewer from another center. While preferable to using a single reviewer from the study site, this likely further decreased agreement. A potential confounder arises from using experts in a field to evaluate a decision support tool. By virtue of their expertise they may find

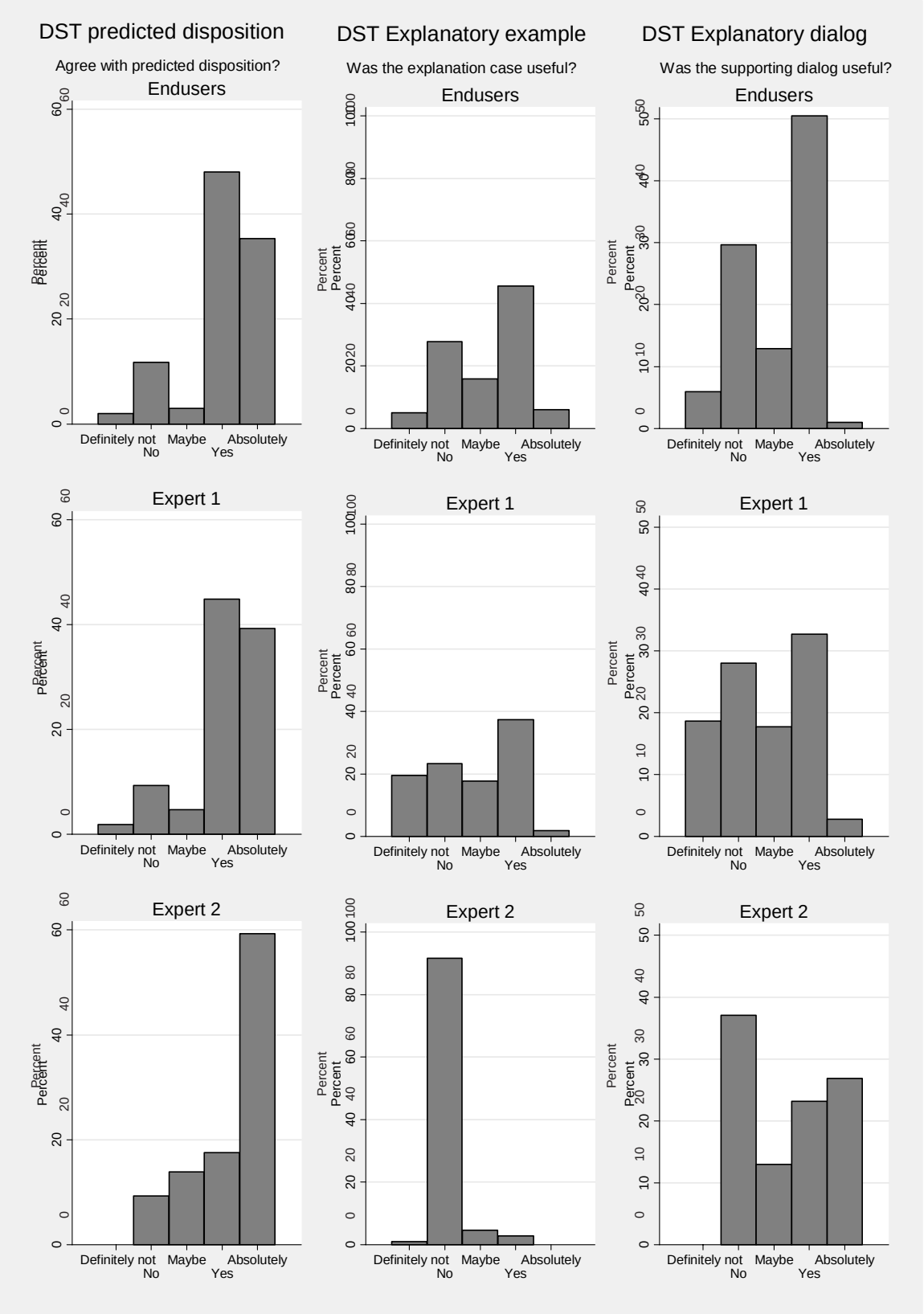

Figure 3. Evaluation (raw scores) of the DST by the end users and expert reviewers.

any DST less useful than their more generalist colleagues, and there is some evidence in this study pointing to this. However, there were few cases according to these reviewers where their opinion of the DST would have been changed regardless of whether they rated its output for their own use or what they perceived as appropriate for a more general audience.

Other potential confounding factors can be missed. Local limitations on bed availability, proximity of the patient's residence to the hospital, and the reliability of the parents affect dispositions in ways unmeasured by the decision support tool we tested. While the arrival of a clearly intoxicated parent will likely be documented, subtler yet important considerations may not be recorded on the patient's chart. For instance, dirty maternal fingernails have been associated with increased infant dehydration[9] but are not often noted on a child's chart. Moreover, estimating the magnitude of the effect of such variables on disposition decisions is difficult. The DST will not reflect these considerations, so such cases will tend to decrease agreement between end users and a subsequent reviewer on the correctness of the DST output. All these considerations tend to make our estimate more conservative. This lends support to using expert reviewers for objective criteria; however, even if our estimate of agreement on subjective DST output is overly conservative, this agreement was so weak that it seems difficult to justify their use.

Answering our question in the general will require replicating experiments like ours with a variety of DST types in various clinical settings for a variety of clinical conditions. In the meantime DST developers must at least consider the implications of this work when prototype testing DSTs.

CONCLUSION

Agreement between expert evaluators and end users with predicted disposition for children with bronchiolitis by a CBR-based DST predicted was moderate. For the more subjective explicative components of the DST output, agreement was fair, poor, or worse. The general clinical end users ranked the DST more highly than the specialist clinical reviewers.

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APPENDIX

Sample of the DST output.

Features	Patient	Explanation case
Age	1.2	1.8
Birth	Vaginal	Vaginal
Smoking Mother	No	No
Hydration before treatment	Normal	Normal
O2 saturation before treatment	99.0	98.0
Retraction severity before treatment	None	Mod
Heart rate after treatment	129	129
Overall increase in work of breathing	None	None
Oxygen saturation under 92 after treatment	No (100.0)	No (99.0)
Respiratory rate over 60 after treatment	No (42)	No (38)
Temperature over 100.4 after treatment	No (98.0)	No (99.9)
Work of breathing after treatment	Same	Improved
Disposition		Admit

We suggest that this patient should be admitted to hospital.

In support of this prediction we have the Explanation Case that was older and had a better response to treatment but was still admitted to hospital.

However, it should be noted that the patient's lower heart rate after treatment and less severe retractions and higher O2 saturation before treatment in relation to the Explanation Case are features that go against our argument that the explanation case is healthier than the patient.

We have a reasonable confidence in our prediction

	Definitely Not	No	Maybe	Yes	Absolutely
Q1. Do you agree with the suggested course of action?	☐	☐	☐	☐	☐
Q2. Did you find the explanation case useful?	☐	☐	☐	☐	☐
Q3. Did you find the supporting dialog useful?	☐	☐	☐	☐	☐

Leukocytosis as a Predictor of Severe Injury in Blunt Trauma

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Objective: The objective of this study was to determine if the white blood cell count can predict severity of injury in blunt trauma victims.

Methods: This was a retrospective study comparing two groups of blunt trauma victims by severity of injury, one with significant injury and one without significant injury, and comparing their initial WBC in the emergency department (ED). We also examined if WBC correlates with degree of injury using Injury Severity Score (ISS) in both groups combined. Further, we examined the WBC as a predictor of serious injury.

Results: Our study showed a difference in mean WBC between the two groups that was statistically significant ($p<0.001$). A positive relationship between ISS and WBC was found, although the association was weak (correlation coefficient = 0.369). While the WBC had moderate discriminatory capability for serious injury, it could not, in isolation, reliably rule in or out serious injury. Nevertheless, this study supports using WBC on presentation to the ED as an adjunct for making disposition decisions.

Conclusion: A significant elevation in WBC in a blunt trauma patient, even with minimal initial signs of severe injury, should heighten suspicion for occult injury.
[*West*JEM. 2008;9:81-85.]

INTRODUCTION

Identifying significant injury in the trauma patient remains a difficult task. Having early markers of injury may help the clinician prevent serious decompensation of patients and thus improve their outcome and hasten their disposition. We set out to determine if WBC can aid physical exam, history, and other tests in detecting severe injury in blunt trauma victims.

Leukocytosis in trauma/stress is due to neutrophilia, caused by neutrophil margination, and not due to increased marrow production or release of immature cells or bands. The phenomenon is short-lived, lasting only minutes to hours.[1] In theory, patients with significant injury should have a higher degree of leukocytosis compared to patients with minor injuries. Since the CBC is one of the first tests obtained from trauma patients in the emergency department (ED), WBC level could serve as an easy-to-obtain marker for serious injury.

METHODS

The study was conducted at Kern Medical Center, a metropolitan county teaching hospital, Level II trauma center with approximately 50,000 yearly ED visits, and 2,400 trauma activations per year. The hospital is the only trauma center in Kern County, California, serving a population of approximately 800,000. This study was a retrospective analysis. Initial patient identification utilized the hospital trauma database. Entry criteria included all ED patients treated for blunt trauma, whether admitted or discharged from the ED, between January 1, 2003 and July 31, 2004. Exclusion criteria were non-blunt trauma (such as penetrating trauma or burn victims), or lack of white blood cell count determination on admission. Medical records for all entered patients were retrieved and examined by a single research assistant. Accuracy was reviewed by a 100 percent review of all charts and data by the principal investigator for the data outlined below. Standard definitions and criteria were used to obtain the data. IRB approval was obtained for this study.

Data collected included WBC on admission, patient age, sex, race, co-morbid conditions, prescription medication use, drug/alcohol intoxication, presenting Injury Severity

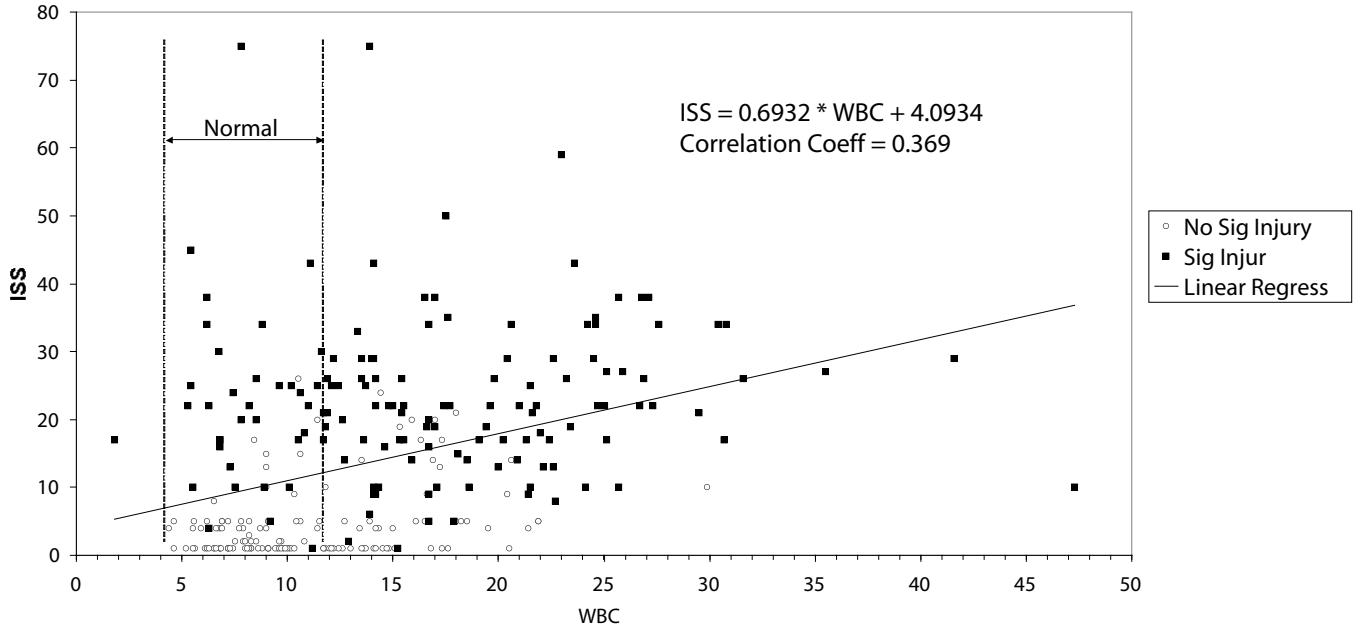

Figure 1. White Blood Count (WBC) vs. Injury Severity Score (ISS)

Score (ISS), disposition, and time elapsed between injury and WBC blood draw. Co-morbid conditions that might affect the degree of leukocytosis were recorded, including pregnancy, chronic infection, diabetes, cardiovascular disease, pulmonary disease, cancer, liver disease, pancytopenia on presentation, and immunologic diseases. Prescription drug use such as steroids, immuno-suppressants, lithium, beta agonists, as well as recreational drug use and drug toxicology results, including methamphetamine, cocaine, opiates, benzodiazepines, and alcohol, were also recorded.

Patients were divided into two groups by severity, one with significant injury and one without significant injury. Significant injury was defined as any of the following injuries: all intra-abdominal and intracranial injuries, spinal and skull fractures, pelvic diastasis, pulmonary contusions, hemothorax, pneumothorax, flail chest, and bronchial tree, great vessel or major arterial injuries. These injuries were documented in the medical records either on admission, via radiologic or operative reports, during the hospital course, or on discharge diagnosis. Also included in the significant injury group were all patients with associated symptoms of hypotension (systolic blood pressure equal to or less than 90 mmHg) on arrival in the ED, death, requirement of transfusion, or operative management within the initial 24 hours following injury. Patients requiring operative management within 24 hours included injuries such as severe degloving injuries, pelvic fractures, severe facial injuries, bronchial tree lacerations, arterial injuries, open extremity fractures, open joint injuries, urogenital injuries requiring surgery.

The non-significant injury group included all remaining patients with minor injuries not meeting the significant injury specifications outlined above, such as patients with non-emergent extremity fractures, as well as all patients that may have been admitted for observation who did not develop significant injuries.

A one-tailed design was selected, since prior reports have not shown trauma to be a cause of lowered, only elevated, white counts. Assuming an alpha error of 0.05, a beta error of 0.20, a standard deviation of 5,000 WBC and a detectable effect size of 1500 WBC/mm^3, 137 patients per group would be required. Evaluation of whether a significant difference between groups existed was determined by assessing WBC as a predictor of serious injury and carried out using receiver operator characteristic (ROC) analysis. Continuous data were summarized as mean ± one standard deviation, and were compared using the student's t-test. Categorical data were compared using the chi-square test. Statistical significance was set at an alpha error of 0.05.

Outcome among patients discharged home from the ED was assessed by review of the entire patient's medical record, which include all patient visits. Records were reviewed for each study participant. If patients were followed up in trauma clinic or other specialty clinic visits, the clinic visit was available for review in medical records.

RESULTS

A total of 279 blunt trauma patient records were reviewed. Of these, 142 were found to have significant injury and 137 did not have significant injury. Of those patients with significant injury, seven were dead on arrival or died in the ED, 127 were admitted to the hospital (44 via the operating room), seven were transferred to another institution, and one was discharged home after observation. Of those without

Table 1. Confounding Variables

	Significant Injury (142)			Non-Significant Injury (137)		
	Number	**Percent**	**95% Conf Interval**	**Number**	**Percent**	**95% Conf Interval**
Sex						
Male	92	64.8%	0.563-0.726	83	60.6%	0.519-0.688
Female	50	35.2%	0.274-0.437	54	39.4%	0.312-0.481
Race						
Caucasian	66	46.5%	0.381-0.550	66	48.2%	0.396-0.569
Hispanic	57	40.1%	0.320-0.487	55	40.1%	0.319-0.489
African American	10	7.0%	0.034-0.126	7	5.1%	0.021-0.102
Asian	8	5.6%	0.025-0.108	6	4.4%	0.016-0.093
Other	1	0.7%	0.000-0.039	3	2.2%	0.004-0.063
Age						
0-25	52	36.6%	0.287-0.451	60	43.8%	0.353-0.525
25-50	61	43.0%	0.347-0.515	51	37.2%	0.291-0.459
50-75	21	14.8%	0.094-0.217	21	15.3%	0.097-0.225
>75	8	5.6%	0.025-0.108	5	3.6%	0.012-0.083
Co-Morbidity						
Diabetic	6	4.2%	0.016-0.090	6	4.4%	0.016-0.093
Cardiac	3	2.1%	0.004-0.060	4	2.9%	0.008-0.073
Pulmonary	3	2.1%	0.004-0.060	11	8.0%	0.041-0.139
Modulating**	8	5.6%	0.025-0.108	4	2.9%	0.008-0.073
Pregnancy	0	0.0%	0.000-0.026	2	1.5%	0.002-0.052
Meds						
Prescription meds	9	6.3%	0.029-0.117	9	6.6%	0.030-0.121
Meds (unknown)	11	7.7%	0.039-0.134	2	1.5%	0.002-0.052
Ethanol	26	18.3%	0.123-0.257	20	14.6%	0.091-0.216
Amphetamines/cocaine	11	7.7%	0.039-0.134	15	10.9%	0.063-0.174
Opiates	16	11.3%	0.066-0.177	10	7.3%	0.036-0.130
Benzodiazepines	16	11.3%	0.066-0.177	0	0.0%	0.000-0.026
Time						
0-90	79	55.6%	0.471-0.640	81	59.1%	0.504-0.674
90-180	45	31.7%	0.241-0.400	45	32.8%	0.251-0.414
>180	10	7.0%	0.034-0.126	11	8.0%	0.041-0.139

* p-values calculated using Chi-square
** liver disease, AIDS, blood transfusion, ulcerative colitis, pancytopenia, Sjorgen's S., mononucleosis, cancer.

significant injury, 85 were admitted and 52 were discharged home.

Confounding variables were compared and occurred with similar frequency in the two groups with some exceptions (Table 1). Pulmonary co-morbidities were more frequent in the non-injury group (8% vs. 2%); prescription medication use was more common in the significant injury group (7.7% vs. 1.5%) and positive benzodiazepine screens were seen more often in patients with significant injury (11% vs. 0%). The mean WBC for the significant injury group was 16,900 ± 7,590. Mean WBC for the non-significant injury group was 11,050 ± 4,560. The difference in WBC values between the two groups was statistically significant (Student's t=7.77, $p<0.001$).

In order to examine the relationship between admission WBC and Injury Severity Score (ISS) we combined groups (Figure 1). Although a positive relationship between ISS and WBC was found (ISS = 0.69 x WBC + 4.1) the association was weak (correlation coefficient = 0.369).

We next examined the admission WBC as a predictor of

Table 2. Sensitivity and Specificity with Various WBC Cutoff Values

False Negative Cutoff* (000/mm^3)	False Negative	True Negative	True Positive	False Positive	Sensitivity	Specificity
6.0	5	9	137	128	0.965	0.066
6.5	9	13	133	124	0.937	0.095
7.0	13	27	129	110	0.908	0.197
7.5	15	34	127	103	0.894	0.248
8.0	18	39	124	98	0.873	0.285
8.5	19	49	123	88	0.866	0.358
9.0	23	53	119	84	0.838	0.387
9.5	24	65	118	72	0.831	0.474
10.0	25	72	117	65	0.824	0.526
10.5	27	78	115	59	0.810	0.569
11.0	30	82	112	55	0.789	0.599
11.5	34	84	108	53	0.761	0.613
12.0	40	89	102	48	0.718	0.650
12.5	43	93	99	44	0.697	0.679
13.0	46	95	96	42	0.676	0.693
13.5	47	97	95	40	0.669	0.708
14.0	53	102	89	35	0.627	0.745
14.5	62	107	80	30	0.563	0.781
15.0	64	110	78	27	0.549	0.803
15.5	69	113	73	24	0.514	0.825
16.0	72	114	70	23	0.493	0.832
16.5	72	116	70	21	0.493	0.847
17.0	79	120	63	17	0.444	0.876
17.5	83	124	59	13	0.415	0.905
18.0	87	125	55	12	0.387	0.912
18.5	88	128	54	9	0.380	0.934
19.0	90	129	52	8	0.366	0.942
19.5	92	129	50	8	0.352	0.942
20.0	94	130	48	7	0.338	0.949
25.0	122	136	20	1	0.141	0.993
30.0	135	137	7	0	0.049	1.000

*WBC values equal to or greater than that listed considered a positive test.

serious injury. After ranking subjects in order of ascending WBC values, sensitivities and specificities were calculated for various cut-offs for WBC values. Among patients with WBC values at or above the cut-off level, those with serious injury were counted as true positives, those without serious injury as false positives. Among patients with WBC values below the cut-off, those with no serious injury were counted as true negatives, those with serious injury as false negatives (Table 2). An ROC curve plot of the results of this analysis is shown in Figure 2. The area under the resulting ROC curve was 0.743 (moderate discriminating power).

DISCUSSION

Even among patients not sustaining significant injury, patients presenting to the ED following blunt trauma tended to have elevated, or at least high normal, WBC values, suggesting that the stress of the trauma incident itself can result in marked demargination, even in the absence of major injury. It is clear from our sample that WBC levels falling within the normal range cannot be used to rule out major injury. While WBC alone cannot be used to effectively rule in or rule out serious injury, its moderate ability to discriminate between patients with and without serious injury suggests that it may contribute

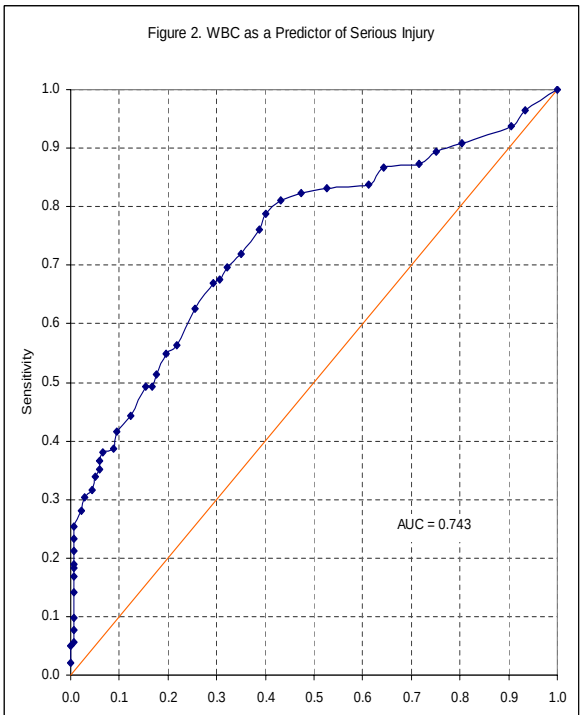

Figure 2. WBC as a Predictor of Serious Injury

meaningfully to the constellation of data used to make disposition decisions. Further study incorporating this variable into prediction models would clarify its value in this regard.

Past studies have either focused on subpopulations of blunt trauma patients, i.e., pediatric or head trauma patients, or studied several variables affecting WBC, and have shown mixed results. Review of the literature reveals several related studies:

Akköse et al[2] performed a retrospective study evaluating 713 blunt trauma patients showing that WBC was positively correlated with ISS. Chang et al[3] prospectively studied 882 patients admitted to a Level 1 trauma center evaluating admission WBC (only available for 786 patients) and GCS, race, injury mechanism, BP, and patients requiring early transfusion vs. no early transfusion, and found that only ISS greater than 15, GCS less than or equal to 8, and white race were associated with increases in white blood cell count. Harris et al[4] retrospectively studied 46 patients after blunt abdominal trauma. They found that, in patients without obvious indications for invasive evaluation of the abdomen (e.g., peritoneal lavage, laparoscopy, laparotomy), leukocytosis was associated with intestinal injury.

Holmes et al[5] retrospectively evaluated 1040 patients <15 years of age with blunt traumatic injury admitted to Level 1 trauma center and concluded that in children hospitalized for blunt torso trauma who are at moderate risk for intraabdominal injury, elevated WBC was associated with intra-abdominal injury.

Rovlias et al[6] prospectively studied 624 patients with head injury and found patients with severe head injury had significantly higher WBC than those with moderate or minor injury. In severe head injury patients, WBC was also associated with unfavorable outcome.

LIMITATIONS

Our study sample of all patients presenting to a trauma center included patients admitted and discharged from the ED. However, our trauma study population proved to be predominantly young (mostly under 50 years of age) and white/hispanic.

Retrospective analysis is always limited to available data within medical records. In order to more accurately apply results of this study to our typical patient population, we elected not to exclude patients with conditions that may affect the degree of leukocytosis, such as age, medical history, prescription medication use, or drug/alcohol intoxication. Some of these confounding variables may have skewed the results.

Assessment of outcome was limited to patients seen at Kern Medical Center. It was assumed that patients with new or recurrent symptoms would return to their initial visit site at Kern Medical Center, the only trauma center in Kern County. However, any patients that may have left the county or returned to an outside hospital may have been missed. No follow-up calls to discharged patients were conducted.

CONCLUSION

The white blood cell count, taken in isolation, cannot be relied upon to rule in or rule out serious injury in blunt trauma patients. Nevertheless, this study supports using WBC on presentation to the ED as an adjunct for making disposition decisions. A significant elevation in WBC in a blunt trauma patient, even with minimal initial signs of severe injury, should heighten suspicion for occult injury.

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Patient Satisfaction and Return to Daily Activities Using Etomidate Procedural Sedation for Orthopedic Injuries

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Objectives: With regard to sedative agents used in procedural sedation and analgesia (PSA), such as etomidate, the focus has been on variables usually related to side effect profile and the success rates of various procedures, with both variables specifically taking place during the patients' stay in the emergency department (ED). There have been no extensive data on the functional status of patients after they leave the ED following PSA.

Methods: Prospective questionnaire evaluating functional status among consecutive adult patients discharged from the ED after undergoing etomidate PSA for orthopedic procedures.

Results: The study sample consisted of 26 cases using only etomidate for closed orthopedic reductions. The mean age was 50.1 years (SD: 20.5), mean weight 86.3 kg (SD: 17.2), and 61.5% were males. The average dose of etomidate given was 0.14 mg/kg with 26.9% requiring a second dose of 0.11 mg/kg. The average dose of analgesic given was 0.11mg/kg in morphine equianalgesic units. The median time between procedural sedation and return to normal sleep was 36 hours, while return to operating a motor vehicle or return to work was 72 hours. Overall, 80% to 100% of respondents felt that any temporary dysfunction was secondary to the orthopedic problems and not to the procedural sedation.

Conclusion: In this small follow-up study, adult patients undergoing PSA with etomidate for orthopedic closed reduction attribute post-discharge functional disability to the injury sustained and not to the PSA itself.
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INTRODUCTION

Procedural Sedation and Analgesia (PSA), as noted from the 2005 American College of Emergency Physicians clinical policy,[1] is defined as "…a technique of administering sedatives or dissociative agents with or without analgesics to induce a state that allows the patient to tolerate unpleasant procedures while maintaining cardio-respiratory function" (p.178).[1] Although emergency department (ED) physicians use PSA for the repair of complex pediatric lacerations and electrical cardioversions, PSA is also commonly used for reducing fractures and dislocations.[2]

Many drugs are available for procedural sedation. Etomidate has been used as an anesthetic agent for over a quarter century.[2-4] It is a non-barbiturate hypnotic that induces sedation through g-aminobutyric acid (GABA) receptors in the central nervous system (CNS).[5] Its rapid onset, short duration of action, clinically insignificant hemodynamic alterations and minimal side effects have accorded it prominence as an adjunct to rapid sequence intubation in emergency medicine. Etomidate's potential adverse effects include nausea/vomiting, potential loss of the protective airway reflexes, myoclonus, adrenal suppression,

Table 1. Etomidate and Analgesic Given

Dislocation	n	1st dose etomidate mg/kg (range)	2nd dose etomidate	2nd dose mg/kg (range)	Received analgestic also	Average IV opiate dose mg/kg (range)*
Shoulder	16 (59.3%)	0.13 (.09-.21)	5 (31.2%)	0.12 (0.12-0.12)	11 (68.7%)	0.09 (0.02 - 0.28)
Hip	5 (19.2%)	0.11 (.09-.13)	1 (20.0%)	0.09	2 (40.0%)	0.06 (0.02 - 0.09)
Elbow	3 (11.5%)	0.16 (.10-.20)	1 (33.3%)	0.12	3 (100%)	0.07 (0.05 - 0.10)
Ankle	2 (7.7%)	0.26 (.25-.28)	0	0	2 (100%)	0.33 (0.31 - 0.35)
Total	26 (100%)	0.14 (.09-.28)	7 (26.9%)	0.11 (0.07-0.16)	18 (69.2%)	0.11 (0.02 - 0.35)

* Oploid dose in morphine equianalgesic units

and hypotension. Electrolyte imbalances and oliguria may occur with prolonged use.[5,6]

Previous research regarding sedative agents focused on variables usually related to the side effect profile of the medication and the success rates of the procedures involved. In addition, some research focused on more qualitative variables. These variables, however, are limited to questions regarding patient satisfaction and appropriate recovery time until discharge.[2,7] Quantitative studies on the functional status of patients after discharge from the ED following procedural sedation needs investigation. This study examined the effects on the recovery and return to daily activities of adult patients who underwent procedural sedation for orthopedic reductions in the ED.

METHODS

This is a prospective questionnaire evaluating functional status among consecutive adult patients discharged from the ED after undergoing etomidate PSA for orthopedic procedures. The study used a 10-item telephone questionnaire format. One to two weeks after discharge, investigators completed the formal questionnaire. Consecutive patients were identified from the ED log for all patients undergoing procedural sedation from September 2004 to June 2005. The study was performed at an urban community hospital that serves 37,000 annual patient ED visits.

Study Protocol

This prospective study initially examined medical records for specific demographic data about the patient to assure inclusion parameters (use of etomidate, ED patients older than 18 years of age and procedures involving orthopedic reduction with or without use of analgesics). Exclusion criteria included pregnant patients, non-English speaking patients, history of organic brain disease (not alert, and oriented to person, place, or time), and procedures conducted with analgesics but without the use of sedatives.

Measurements

Abstracted data included age, gender, weight, arrival and discharge times, time elapsed from sedation to discharge, diagnosis, names and doses of all medications related to the procedure, and complications. Anticipated complications reviewed included oxygen de-saturation (<94 %), blood pressure <90/60 mm Hg, blood pressure >140/90 mm Hg, respiratory rate <10 breaths per minute, respiratory rate > 20 breaths per minute, respiratory assistance, respiratory failure requiring intubation, mental status abnormality, vomiting, and rhythm changes. Investigators also recorded major medical conditions, allergies, use of tobacco, type of work, and exercise habits. Following acceptance by the Institutional Review Board, all data, including responses to the questionnaire, were recorded on preformatted data sheets, coded, and entered into an electronic database. Figure 1 shows the data collection tool.

Data Analysis

The Statistical Package for Social Sciences (SPSS) for Windows Version 11.5 (Chicago, IL, 2002) was used to calculate descriptive statistics.

RESULTS

Thirty patients received procedural sedation for closed orthopedic reductions during the study period. Two patients were excluded secondary to dementia. The remaining 28 patients were successfully contacted and agreed to participate in the study. However, one patient received both etomidate and diazepam for sedation and another patient received both etomidate and midazolam. These two cases were removed from analysis.

1. How effective was the medicine in putting you to sleep for the procedure?
   ☐ Not at all ☐ Somewhat ☐ Moderately ☐ Extremely
2. How effective was the medicine in making you forget what happened during the procedure?
   ☐ Not at all ☐ Somewhat ☐ Moderately ☐ Extremely
3. How nauseated were you after being discharged from the emergency department?
   ☐ Not at all ☐ Somewhat ☐ Moderately ☐ Extremely
   a. How long? (Half-day increments) ☐ 0 hours ☐ 12 hours ☐ 24 hours ☐ 36 hours ☐ 48 hours ☐ 60 hours
4. How likely would you undergo the same sedation medication in a future procedure?
   ☐ Not at all ☐ Somewhat ☐ Moderately ☐ Extremely
5. Do you drive a car? ☐ Yes ☐ No
   a. If yes, how long before you drove a car?
      ☐ 0 hours ☐ 12 hours ☐ 24 hours
      ☐ 36 hours ☐ 48 hours ☐ 60 hours
   b. Was this mainly a result of the medications or disability from the procedure?
      ☐ Medications ☐ Unsure
      ☐ Disability ☐ Other
6. How long before you ate a regular meal?
   ☐ 0 hours ☐ 12 hours ☐ 24 hours
   ☐ 36 hours ☐ 48 hours ☐ 60 hours
7. How long before you slept as you normally did?
   ☐ 0 hours ☐ 12 hours ☐ 24 hours
   ☐ 36 hours ☐ 48 hours ☐ 60 hours
   a. Was this mainly a result of the medications or disability from the procedure?
      ☐ Medications ☐ Unsure
      ☐ Disability ☐ Other
8. How long before you returned to work?
   ☐ 0 hours ☐ 12 hours ☐ 24 hours
   ☐ 36 hours ☐ 48 hours ☐ 60 hours
   a. Was this mainly a result of the medications or disability from the procedure?
      ☐ Medications ☐ Unsure
      ☐ Disability ☐ Other
9. Do you exercise? ☐ Yes ☐ No
   a. If you exercise, how long before you returned to a sports or exercise regimen?
      ☐ 0 hours ☐ 12 hours ☐ 24 hours
      ☐ 36 hours ☐ 48 hours ☐ 60 hours
   b. Was this mainly a result of the medications or disability from the procedure?
      ☐ Medications ☐ Unsure
      ☐ Disability ☐ Other
10. Did you require any help from friends or family for daily activities for which ou are normally independent?
   ☐ Yes ☐ No
   a. How long?
      ☐ 0 hours ☐ 12 hours ☐ 24 hours
      ☐ 36 hours ☐ 48 hours ☐ 60 hours
   b. Was this mainly a result of the medications or disability from the procedure?
      ☐ Medications ☐ Unsure
      ☐ Disability ☐ Other

Comments:

_____

Figure 1. Patient Interview

The mean age was 50.1 years (SD: 20.5), the mean weight was 86.3 kg (SD: 17.2) and 61.5% of the sample were males. The majority of patients were employed with only eight (30.8%) retired. An associated medical condition was noted in 10/26 (38.5%) patients and five of those had either hypertension or heart disease. Table 1 provides the type of orthopedic reduction and the average dose of etomidate used. A second dose of etomidate was required in 26.9% of cases.

Table 1 also provides the median dose of associated analgesic drugs used during procedural sedation. An intravenous analgesic along with etomidate was given in 18 (69.2%) patients. Morphine was given solely in nine (34.6%)

Table 2. Return to Normal Activities and Function Following Shoulder Reductions n=16

	Responses	Median hours (Inter-quartile range)	Respondent felt temporary dysfunction related to orthopedic problem rather than procedural sedation
Time before a regular meal	16/16	18.0 (12.0-24.0) 16 responses	
Time before regular sleep	16/16	24.0 (24.0-48.0) 16 responses	12/14 (85.7%)
Time before patient drove a car	12/12 drivers	72.0 (60.0-120.0) 11 responses	11/12 (91.7%)
Time before return to work	7/8 working	96.0 (48.0-120.0) 7 responses	7/7 (100%)
Time requiring help from family or friends	14/16	24.0 (12.0-48.0) 14 responses	8/14 (57.1%)

Table 3. Return to Normal Activities and Function Following Other Type Reductions n=10

	Responses	Median hours (Inter-quartile range)	Respondent felt temporary dysfunction related to orthopedic problem rather than procedural sedation
Time before a regular meal	9/10	24.0 (12.0-24.0) 9 responses	
Time before regular sleep	9/10	48.0 (30.0-48.0) 9 responses	9/9 (100.0%)
Time before patient drove a car	8/8 drivers	72.0 (48.0-114.0) 4 responses	8/8 (100.0%)
Time before return to work	4/4 working	66.0 (60.0-72.0) 2 responses	4/4 (100.0%)
Time requiring help from family or friends	10/10	48.0 (24.0-72.0) 9 responses	8/10 (80.0%)

cases, while six (23.1%) were given fentanyl only, and three (11.5%) received both.

Our study found no patients that experienced oxygen de-saturation, emesis, rhythm changes, or mental status abnormalities; however, four (15.4%) patients had slightly decreased or increased respiratory rates. None required intervention. Overall, 14 (53.8%) patients had alterations of systolic blood pressure, but only one patient required intervention. This patient received enalapril for elevated blood pressure. On the follow-up survey all 26 reported that the procedural sedative was extremely effective for amnesia to the events. All 26 also were willing to undergo the same sedation again. All 26 experienced nausea following ED discharge, but only one patient reported nausea for more than 12 hours.

Table 2 and Table 3 show the median time for return to normal activities and function. It is seen that in 80% to 100% of cases, patients felt that their temporary disability was secondary to their orthopedic issue rather than related to their procedural sedation.

DISCUSSION

This study examined the effects of procedural sedation on ED-discharged adult patients and their recovery and return to daily activities. Etomidate was the primary sedative used for orthopedic reductions. Overall, patients receiving etomidate for PSA in the ED were satisfied and PSA was well tolerated. Of the 26 patients in our study, only one patient required any intervention for an adverse response. These results are consistent with previously published reports.[2,4,5,8,9]

The median time of functional disability for the patients

in our study was one to three days. However, over 80% of the respondents felt that their disability, i.e., returning to normal sleep, operating motor vehicles, and returning to work, was not related to PSA. Almost all of the patients required family/friend assistance for a median of 1.5 days, and 57.1% of the patients felt that this was directly a function of PSA. These findings directly affect how emergency medicine physicians can educate their patients prior to discharge following PSA. Certainly, the medical condition that necessitated PSA will limit their activities for 1-3 days, but they should also be aware that they may need extra assistance from family and friends.

LIMITATIONS

A major limitation to this study is the small sample size. However, given the emerging popularity of etomidate PSA among emergency physicians and that much of PSA in the ED is performed for orthopedic reductions,[2,4,9] these results are clinically relevant for many practicing emergency physicians. Finally, this study relied on accurate patient perceptions and recall. Some patients may have had trouble truly delineating the cause of the limitations, in other words, the medication involved in the procedure, or the medical condition necessitating the procedure.

CONCLUSIONS

Etomidate for orthopedic reductions in the ED is well tolerated with very high patient satisfaction and low adverse events; however, patients will have some temporary disability following discharge. Adult patients will likely have some functional disability for 36 to 72 hours. The vast majority of patients related their temporary functional disability to their underlying orthopedic problem and not to the procedural sedation itself.

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The Imminent Healthcare and Emergency Care Crisis in Japan

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Objectives: Japan has a universal healthcare system, and this paper describes the reality of the healthcare services provided, as well as current issues with the system.

Methods: Academic, government, and press reports on Japanese healthcare systems and healthcare guidelines were reviewed.

Results: The universal healthcare system of Japan is considered internationally to be both low-cost and effective because the Japanese population enjoys good health status with a long life expectancy, while healthcare spending in Japan is below the average given by the Organization for Economic Corporation and Development (OECD). However, in many regions of Japan the existing healthcare resources are seriously inadequate, especially with regard to the number of physicians and other health professionals. Because healthcare is traditionally viewed as "sacred" work in Japan, healthcare professionals are expected to make large personal sacrifices. Also, public attitudes toward medical malpractice have changed in recent decades, and medical professionals are facing legal issues without experienced support of the government or legal professionals. Administrative response to the lack of resources and collaboration among communities are beginning, and more efficient control and management of the healthcare system is under consideration.

Conclusion: The Japanese healthcare system needs to adopt an efficient medical control organization to ease the strain on existing healthcare professionals and to increase the number of physicians and other healthcare resources. Rather than continuing to depend on healthcare professionals being able and willing to make personal sacrifices, the government, the public and medical societies must cooperate and support changes in the healthcare system.
[*West*JEM. 2008;9:91-96.]

INTRODUCTION

While universal health insurance is commonly viewed in the United States (U.S.) as a means to guarantee adequate healthcare to all residents of the community, the system in Japan does not actually function this way. In fact, the Japanese healthcare system is breaking down rapidly. Offering inexpensive care equally to all citizens has depended on heavy individual sacrifices by healthcare professionals, especially pediatricians, obstetricians and gynecologists, and critical care/emergency care physicians. Doctors are exhausted and lack healthcare support and resources. Emergency and pre-hospital care systems are paying one of the heaviest tolls. Recent incidents, such as ambulances not being able to find hospitals with the appropriate resources to treat patients, are making national headlines.

In 2006 there were 8,943 hospitals (defined as a medical facility with 20 beds or more), with national or public hospitals accounting for 18.4%, and medical corporations (private entities) and private practice hospitals accounting for 70.5%. The number of clinics without inpatient facility (or

19 beds or less) was 98,609, while medical corporations and private practice accounted for the majority at 83.8%.[1] Local clinics and hospitals serve as the primary emergency medical facility for treatment of minor cases; these local facilities also take turns attending to night-time and holiday duties. Larger hospitals and university hospitals, regardless of whether privately or publicly owned, are responsible for providing secondary or tertiary emergency medical care.

The Fatigue of Healthcare Professionals

Shortages of physicians in all specialties are apparent. According to a report by the OECD released in July 2007, the number of physicians per 1,000 people is two; this ratio is among the lowest in industrialized countries and below the 30-member OECD average of three.[2] In the same 2007 OECD report, health expenditures as a share of GDP was reported to be 8% in Japan during the year of 2004, which is also below the 9% OECD average, while in the United States 15.3% was spent on healthcare in 2005. Health expenditure per capita was reported as $2,358 for Japan, $2,753 for the OECD average, and $6,401 for the United States (Figure 1). Therefore, the number of healthcare professionals and the amount spent on healthcare are possibly the lowest of the G7 nations (United States, Japan, Germany, France, United Kingdom, Italy, and Canada).

Nurses are chronically in short supply. In most hospitals the ratio of the number of nurses to patients is more than 10 to 1. The Ministry of Health, Labor, and Welfare is proposing a 7 to 1 ratio in 60% of hospitals with 300 beds or more by 2009. However, with this ratio the shortage of nurses is expected to be 70,000 by April 2008.[3]

On the other hand, the number of licensed paramedics in Japan is increasing. According to April 2007 data published by the Fire and Disaster Management Agency, there are 20,059 licensed paramedics in Japan, 99.9% of fire stations have in-house paramedics, and of 4,940 emergency response teams, 4,201 have licensed paramedics.[4] Japan has a state-of-the-art emergency communication system with highly equipped ambulances. Because Japan is geographically small, the average arrival time for an ambulance is 6.5 minutes. Therefore, paramedics arriving at the site is not considered to be a healthcare issue.

Overall the Japanese population enjoys a very long life expectancy (82 years for the entire population) and an extremely low infant mortality rate of 2.8 deaths per 1,000 live births in 2005 – close to half of the OECD average.[2] Interestingly, Japan has the highest number of doctor consultations per capita of 13.8; Japanese people visit a healthcare facility on average almost 14 times a year, while the OECD average is 6.5 times, and the U.S. average is 3.9 times.[2] In the Japanese medical system, this number of visits

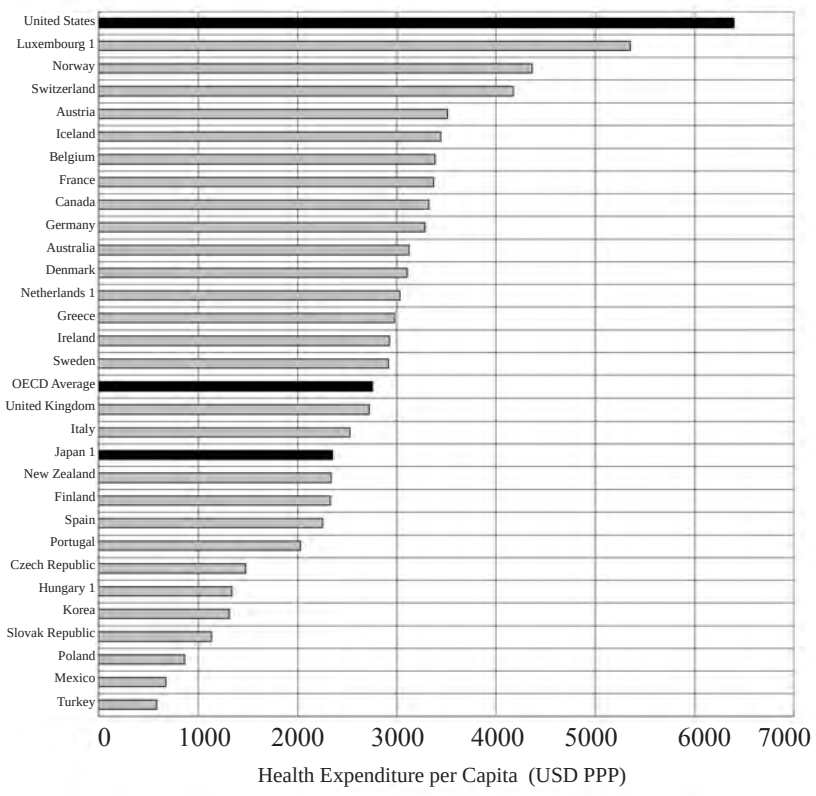

Figure 1. Healthcare Spending in Various Nations

is considered acceptable, despite the low number of practicing physicians per population of 1000 (two in Japan, 2.4 in the U.S., and more than three in many European countries).[2]

Many factors, such as genetics, diet, culture and technology, can account for the seemingly good health of the Japanese. Despite those favorable circumstances, only so much can be done in terms of serious diseases or injuries. The greatest contributing factor by far is that the Japanese healthcare system depends on physicians and healthcare professionals making many personal sacrifices. These individuals work long hours, often without either documentation of or compensation for overtime. According to a 2006 survey of 3,388 medical doctors by the Ministry of Health, Labor, and Welfare, Japanese physicians worked 66.4 (±18.0) hours/week, with the maximum reported at 152.5 hours, and physicians younger than 30 years old worked an average of 77.3 hours/week.[5] While U.S. physicians receive an annual compensation of $150,000 to $300,000,[6] the average compensation for Japanese physicians was about $104,000 for those in their early forties and $77,000 for those in their mid-thirties.[7]

Traditionally because the Japanese public has viewed working in healthcare professions as "sacred," sacrificing personal time and pleasure was expected. This cultural attitude has been used by many public hospitals as a reason to limit the labor rights of their physicians, who have been unable to organize strikes or protest for higher wages or shorter hours. Times have changed, and the Japanese healthcare system is no longer exempt from legal and patient issues. Malpractice lawsuits are on the rise, healthcare spending by government is decreasing, and patient expectations have increased. These factors, along with the increased availability of information through the media and the Internet, are contributing to a change in public attitude toward healthcare.

Old Values Have Led to System Issues

When faced with physician shortages, other countries have delegated various jobs to other qualified healthcare professionals, such as nurses, pharmacists or medical technicians. Such delegation is a new concept in the Japanese medical systems. In Japan healthcare professionals, especially physicians, are granted high levels of authority; others refrain from assisting because the act of helping the physician is perceived as an insult to the physician's ability and knowledge and as an invasion of the medical profession.

With the shortage of physicians and of interaction between professionals, training other technicians for job delegation becomes especially difficult. In addition, there are not enough emergency care physicians to train emergency medical technicians (EMTs), who could possibly become leaders in the field. This is compounded by the fact that EMTs have a relatively low public status in Japan compared with their situation in other developed nations. In turn, this low level of respect leads to low motivation and to lack of leaders and role models. More public awareness of the importance of the role of EMTs and a publicity effort may be needed. Although most EMTs in Japan are stationed at fire departments, in 1991 the official role and regulation of paramedics in Japan was established via a national licensing system. Five years of working experience as ambulance staff, more than 500 hours of lecture, and more than 400 hours of hospital training or simulation in a training facility are required to become an EMT in Japan. However, unlike in the EMT system of the U.S., in Japan there is only one level of EMT, and on-the-job training or continuing education is not required.[8]

The Nara Incidents

On August 7, 2006, a mother in the delivery room of Oyodo Hospital in the southern part of Nara prefecture became unconscious. The attending doctor diagnosed eclampsia and contacted the Nara Medical University Hospital. The facility refused the patient because no bed was available. Other hospitals were also contacted, but none could accommodate the patient. Finally, the National Cardiovascular Center in Osaka prefecture accepted the patient, and she was diagnosed with cerebral hemorrhage. An emergency operation and cesarean section was performed, but the mother died about a week later. The family sued Oyodo Hospital for civil damages.[9]

In the same Nara prefecture about a year later, a pregnant woman called an ambulance because of abdominal pain early in the morning of August 29, 2007. Nine hospitals refused the patient because of insufficient resources. The woman was not under prenatal care and had a history of miscarriage; she delivered a stillborn child before eventually arriving at Takatsuki Hospital in Osaka.[10]

Media reaction further aggravated the local situation, forcing Oyodo hospital to close its obstetrics/gynecology department indefinitely, despite the fact that investigators concluded that making a diagnosis of cerebral hemorrhage was difficult in the situation, and no criminal case was established.[10] The Oyodo case captured tremendous media attention and became one of the causes of a national trend in physicians leaving regional or public hospitals. Some physicians can afford to leave the hospital in favor of solo private practices, while others simply resign their hospital position for part-time or temporary work at other hospitals or clinics. The physician author Hideki Komatsu coined a phrase "tachisarigata sabotage," which means "sabotage by leaving."[11] This expression is an apt description of how hospitals are losing already scarce doctors.

The shortage of physicians in emergency medicine is especially critical, as reported from a survey in the Fukuoka prefecture conducted by Ezaki et al.[12] Fukuoka prefecture is home to the city of Fukuoka, one of Japan's major urban

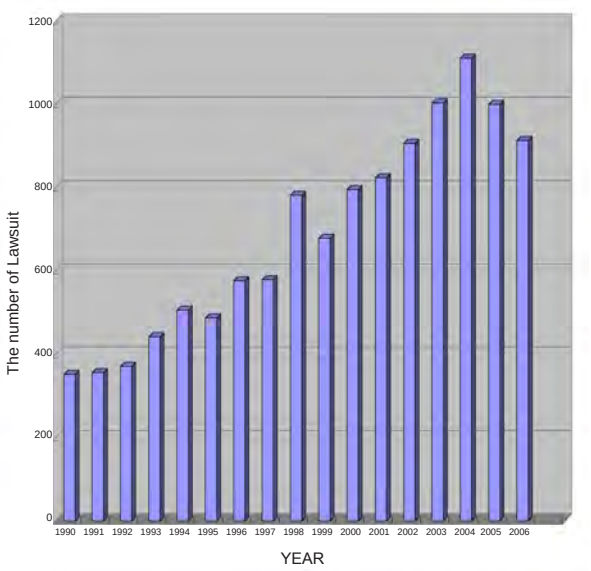

Figure 2. Increase in the Number of Malpractice Cases in Japan

centers, and an industrial area with the nation's ninth largest population – approximately five million as of 2006. Despite an adequate social and economic infrastructure, only 24.4% (32 out of 131) of its emergency medicine facilities responding to a survey had emergency medicine specialists or acute care physicians. Most other facilities were attended by non-emergency or acute care physicians with certifications from organizations, such as the Japan Surgical Society or Japan Society of Internal Medicine.

More Patient Issues

The Japanese healthcare system traditionally does not feature a family doctor or primary care physician, and insurance providers allow patients to see specialists directly without the need for a referral. Most hospitals and clinics routinely accept walk-in patients who usually must wait for long periods of time to see a doctor. One result is that specialists must spend time seeing minor cases that do not require specialist care. For instance, a patient with red eyes goes directly to an ophthalmologist in a university hospital when only minor treatment is needed for mild conjunctivitis. This lack of an organized primary care system overwhelms Japan's limited medical resources. Some facilities are starting to adopt booking systems to manage appointments with doctors. However, when a fully booked schedule causes a patient to be turned away, the Japanese public views this as impersonal and immoral because healthcare professionals are supposed to help people in need whenever asked. Therefore, the facilities using appointment systems usually must accept walk-ins as well, making the appointment system essentially meaningless. This idea stems from the same misconception that the arrival of an ambulance automatically guarantees medical care. The idea that health and safety is guaranteed in Japan has created another issue—that of the so-called "monster patient," who makes unreasonable demands of healthcare professionals. Some patients demand that medicine be perfect and that medical care should always cure the disease. Of course, in reality, this is impossible. The rise in the number of medical malpractice suits in recent years shows that the attitude of Japanese patients toward the healthcare profession has certainly changed, and it is being reflected in the rise of malpractice suits. During the mid 1980s to the early 1990s, the number of new medical malpractice suits in all of Japan numbered in the three hundreds. However, during the late 1990s to early 2000s, the number increased rapidly (Figure 2) while the total number of all civil cases remained at about 150,000 in Japan.[13,14] In the past few years, the effort to obtain informed consent from patients and families may be contributing to a leveling off in the number of new malpractice lawsuits. However, the psychological barrier that had inhibited lawsuits against a physician who does a sacred job has clearly been lowered. Despite such change in public attitudes, the Japanese legal system has not yet developed a method of properly judging medical fault versus unreasonable demand or expectation; in other words, the legal system is not currently prepared to make decisions in medical cases.

Another issue highlighted by the Nara cases is that many medical facilities are reluctant to establish emergency care systems in obstetrics because they have only half the necessary number of doctors, and therefore, accommodating emergency cases will likely lead to malpractice suits.[15]

Recent Responses from the Japanese Administration

One positive result from the Nara incidents and other issues was a response from government and prefectural administrations that ordered the prefectures to improve transportation systems. Japan has been reducing its number of physicians since the 1980s, in an effort to limit healthcare costs. This policy is now being reconsidered, and Yoichi Masuzoe, Minister of Health, Welfare and Labor, announced the formation of a study group on solving various healthcare-related issues, such as the insufficient number of physicians and regional differences in the level of healthcare.[16] This group will consider inviting foreign physicians to practice under certain agreements and mandating medical school graduates to work in rural regions. The prefectural governors are asking the group to identify ways to increase the number of medical school admissions, to delegate some of the work currently being performed by doctors to registered nurses, and to educate primary care physicians to practice at a local level.

In the Nara prefecture the governor of Nara, Shougo Araki, is leading the "Nara Prefecture Board Meeting of A Survey on Issues in Emergency Transportation of Pregnant Women" and implementing a plan to assign a "high-risk pregnant women transport coordinator" system, as well as

a network of private practitioners to take turns handling emergency on-call duties. The prefecture started simulation trainings for transport coordinators in November 2007, and four coordinators were assigned for weekends and holiday duties at Nara Prefectural Medical College Hospital as of November 27, 2007.[17] The Board published an emergency response manual for these services, emphasizing the importance of sharing critical patient information between paramedics and hospitals.[18] The Board also intends to create a seamless system through collaboration of emergency personnel, hospital personnel, and other medical professionals. The need for medical control of emergency and pre-hospital care should be reemphasized throughout.

Introduction of True Medical Control and Medical Director as a Solution

The proposed coordinator system is considered adjunctive to medical control of pre-hospital care. Currently in the Japanese emergency department (ED), physicians working in the ED are the ones making decisions on either to accept or refuse the arrival of patients by ambulance.[19] Both public and healthcare professionals hope this new system would eliminate situations in which ED physicians refuse ambulance arrival without recognizing the severity of the case or knowing the availability of beds in the hospital. In Japan the medical control of pre-hospital care is defined as ensuring the quality of medical activities by having physicians instruct, advise, or evaluate the medical activities of paramedics during transport of patients from incident sites to medical facilities.[20]

The idea for medical control of emergency and pre-hospital care is relatively new in Japan. It was brought into focus by a 1998 Ministry of Health report on the study of the pre-hospital care situation in various regions of the U.S.[21] and by two other Japanese reports, namely, a 2000 report from a study group on the future of the emergency service system, Ministry of Welfare, and a 2001 report from a study group on promoting advancement of emergency services of the Fire and Disaster Management Agency (FDMA).

Despite the fact that the study groups did not provide specific recommendations on changing the emergency service system in Japan, they emphasized collaboration among prefectures, regardless of differences in prefectural administrations, as well as collaboration between physicians and the FDMA, which most Japanese paramedics belong to. The groups were generally optimistic that developing and implementing such a system could be realized, especially considering the current level of medical technology and the expertise of the FDMA. In fact, the Tokyo metropolitan area has established a medical control system in which the efforts of administration, the fire department, and local emergency medicine physicians have been coordinated to function as one group.[22]

A proposed next step would be to support the creation and development of a role for a medical director. This individual would oversee the entire operation and improve its efficiency while ensuring the quality of healthcare and transport activities. Within Japan's current medical control system, emergency care physicians have neither the authority to assign or terminate personnel nor the power to make decisions on how local programs should continue. The Japan Society of Emergency Medicine and other researchers in emergency medicine are currently studying the U.S. system of emergency medical services, especially the activities and achievements of the National Association of EMS Physicians.[23]

Implementing a system in Japan that is similar to that of the United States would certainly have some difficulties. For example, the Japanese administration would first need to define the role and responsibilities of a medical director, and then establish standardized programs and educational courses at the national level for medical directors. However, it can be done—the medical associations in the United States showed strong leadership to establish and maintain such programs, and a similar effort is needed in Japan to develop and implement a more coordinated and higher quality system.

LIMITATIONS

The subject of this article is topical, and comparison to other nations or systems is difficult because of differing cultural and economic backgrounds.

CONCLUSION

Serious issues in the Japanese healthcare system need immediate attention. Adopting an efficient medical control system is needed to ease the strain on existing healthcare professionals and to increase the number of physicians and amount of resources. Cooperation and support from the government, the public and medical societies are essential. When contemplating universal healthcare and finding the best method of pre-hospital and emergency care and transport, other nations should heed the current status of the healthcare situation in Japan as an example.

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Wide Complex Tachycardias: Understanding this Complex Condition
Part 2 - Management, Miscellaneous Causes, and Pitfalls

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INTRODUCTION

Patients who present with electrocardiograms (ECGs) demonstrating wide complex tachycardias (WCTs) are often challenging to clinicians. Not only may the patient present with (or be at risk for) hemodynamic compromise, but their treatment may result in hemodynamic collapse if the incorrect pharmacologic agent is selected. In Part 1 of this article,[1] the identification, epidemiology, and electrophysiology of WCTs were discussed. In this article, treatment of WCTs and miscellaneous causes of this condition will be described.

In patients presenting with WCTs, correct interpretation of the ECG should not be the primary concern of emergency physicians. In fact, it is appropriate (and may be preferred) to "diagnose" the ECG as "wide complex tachycardia of unknown (or uncertain) etiology." This may allow the treating clinician to focus on the patient and his or her hemodynamic status, rather than the academic exercise of ECG interpretation. The most important aspect of treating patients who present with WCTs is to select a therapeutic approach that does no harm. Several diagnostic algorithms were provided for the interpretation of WCTs, although none have proven superior over others, and great debate continues among researchers as to the preferred approach. Given this circumstance of uncertainty, proper treatment will be described for conditions resulting in WCTs.

It is critical to note that once a WCT is identified, the

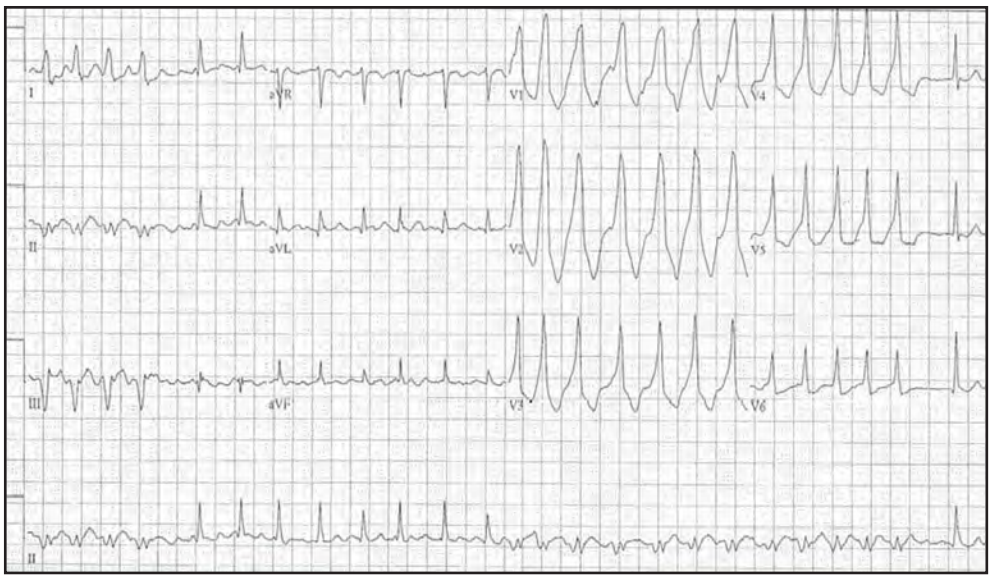

Figure 1. ECG representing Wolff-Parkinson-White Syndrome (WPWS) with Atrial Fibrillation (AFIB). Note the wide QRS complexes, and the irregularly irregular rhythm. In lead II, the axis changes. A slurred delta wave can be identified in the left precordial leads, since the overall rate is not as fast as it might be when these conditions coexist. The R-R interval of the first two QRS complexes seen in leads V1-3 approaches 300, which is extremely dangerous.

Table 1. Synopsis of Advanced Cardiac Life Support guidelines for *Tachycardia with pulses* algorithm[4]

Is the patient stable?

A. If NO, perform immediate synchronized cardioversion (consider sedation), and obtain expert consultation.
B. If YES, assess QRS width and heart rate.

If QRS width greater than or equal to 0.12 seconds, assess if the rhythm is regular (and obtain expert consultation).

A. For **regular WCTs**, if VT or uncertain rhythm, amiodarone; prepare for elective synchronized cardioversion. If SVT with aberrancy, treat with IV adenosine (vagal maneuvers).
B. For **irregular WCTs**, if atrial fibrillation with aberrancy, consider expert consultation; control rate. If pre-excited atrial fibrillation (AFIB + WPWS), expert consultation is advised. Avoid AV nodal blocking agents, consider amiodarone. If recurrent polymorphic VT, seek expert consultation. If torsade de pointes, treat with IV magnesium.

use of certain pharmacologic therapies may have deleterious effects on patients. Verapamil and diltiazem are calcium channel blockers (CCBs) that should be avoided in WCTs, as cardiac arrests from hemodynamic collapse have been reported following their administration.[2,3] This is especially true in infants. Not only do these agents cause negative inotropy and at times profound vasodilation, but they may also allow WCTs to degenerate into VFIB. CCBs slow conduction at the AV node so that accessory pathways can be preferentially used; this results in ventricular rates that approach atrial rates. In addition, verapamil may shorten the effective refractory period of accessory pathway tissues, which is especially dangerous if AFIB and WPWS coexist (Figure 1). Digoxin should be avoided as well in the pharmacologic management of WCTs, as it may facilitate conduction via the accessory pathway. Shortening the refractory period of this pathway is particularly concerning in individuals with anterograde conduction over an accessory pathway (such as the case in AFIB with WPWS), as lethal ventricular rates may develop in response to fast or chaotic atrial rates. Propranolol (or other beta-blocking agents) may facilitate AV conduction via the accessory pathway, which can increase the ventricular rate to dangerous levels during AFIB. Lidocaine is no longer considered by the American Heart Association (AHA) as the drug of choice in WCTs of uncertain origin; good evidence supports alternative agents as being superior. If lidocaine is used, it should be administered with extreme caution, as it has been demonstrated to speed conduction through bypass tracts in experimental models. The use of oral agents in the emergency management of WCTs has no current therapeutic role.

The clinical dictum "do no harm" is especially important when it comes to the management of WCTs, as delays to definitive therapy or their mismanagement (or both) can result in considerable morbidity and mortality. The patient's clinical condition, not the ECG tracing, should be addressed and treated. If the patient is unstable without a pulse, this patient (and the WCT rhythm) should be treated as any patient in ventricular fibrillation, with defibrillation, airway management, and ACLS drugs according to current AHA algorithms. All unstable patients with a pulse should be treated using the unstable VT AHA algorithm, with consideration for sedation prior to synchronized cardioversion. The airway in these patients should be managed with appropriate aggressiveness. The amount of electrical energy needed to cardiovert this WCT to a stable rhythm is generally less than that required for VFIB. In short, all undetermined WCTs should be treated as if they are ventricular in origin (VT). Unless the clinician is absolutely certain that the WCT is supraventricular in origin, pharmacologic agents used in the treatment of SVTs should not be administered (Table 1).

The use of adenosine as a rapid IV push in patients presenting with WCTs deserves special mention. This agent has tremendous efficacy converting SVTs, but on occasion has both diagnostic use as well as success converting some idiopathic causes of VT, especially cases originating in the fascicle.[5,6] Adenosine should be used with caution in the diagnosis and management of WCTs, as prolonged AV block is likely to occur. The rapid administration of adenosine may help identify the underlying etiology of certain WCTs by slowing the heart rate or unmasking accessory tracts.[7] Despite it being considered safe to use in patients presenting with WCTs,[8] there are a few reports of complications following adenosine use. Adenosine may predispose the heart to atrial fibrillation, and may cause the cardiac rhythm to deteriorate into very rapid ventricular rates in patients with pre-excitation syndrome.[9] Fortunately, this increase in ventricular rate was not shown to consistently cause hemodynamic deterioration in one study.[10] Adenosine has also been reported to induce torsade de pointes[11], and has proarrhythmic potential, especially in patients with aberrant conduction pathways, which may produce VT or other WCTs.[12] Other authors share concern that the extranodal effects of adenosine may ultimately lead to pitfalls and misidentification of WCTs.[13] The current AHA guidelines for tachycardia with pulses mentions the use of adenosine for regular wide QRS complexes if SVT with aberrancy is identified as the rhythm. It neither suggests nor recommends its use in a diagnostic trial.[4]

In several areas of the 2005 AHA Tachycardia with pulses algorithm, "expert consultation" is either advised or should be considered. Once the patient is deemed clinically

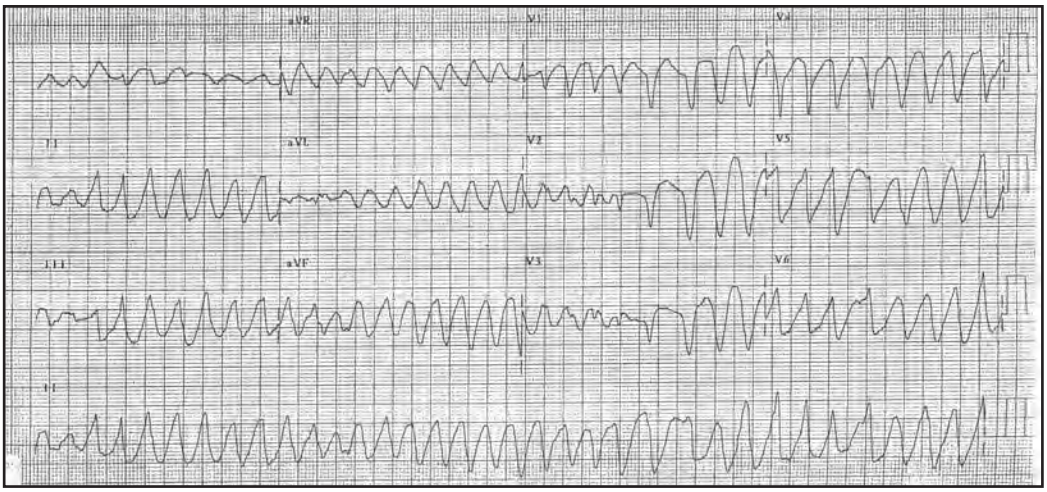

Figure 2. Torsade de pointes (polymorphic VT) captured on 12-lead ECG. The baseline literally "twists of the points." Treatment is with intravenous magnesium, and possibly overdrive pacing (Image courtesy Jeff A. Tabas, MD). Used with permission, ACEP.

stable, and a candidate for pharmacologic intervention, the determination of regular versus irregular rhythm is necessary. For *regular* rhythms, any VT or uncertain rhythm should, according to the guidelines, be treated with IV amiodarone, with preparation for elective synchronized cardioversion (as long as the patient remains clinically stable). For patients with SVT with aberrant conduction, adenosine is suggested. If, on the other hand, the WCT is *irregular*, pharmacologic therapy depends on whether or not AFIB with aberrancy (in which diltiazem or beta-blockers are appropriate) or AFIB with WPWS (in which amiodarone is the agent of choice, and AV nodal blocking agents [*e.g.*, adenosine, digoxin, diltiazem, verapamil] should be avoided) is most likely. According to the 2006 Guidelines for the Management of Patients with Atrial Fibrillation published in *Circulation*, "… in hemodynamically stable patients with pre-excitation, type I antiarrhythmic agents or amiodarone may be administered intravenously." These guidelines further state that if the dysrhythmia causes hemodynamic compromise, early direct current cardioversion is indicated.[14]

Torsade de pointes is a special type of WCT, during which the electrical axis "twists" around the horizontal axis (Figure 2). It is categorized as a form of polymorphic VT. Cycles of alternating electrical polarity of the QRS complexes occur about the isoelectric axis. Torsade de pointes can be initiated when a premature ventricular contraction (PVC) falls on the T wave, or by ventricular couplets or triplets. It commonly has a long-short initiation sequence, and may be more likely during episodes of bradycardia.[15] It has been associated with prolonged QT syndrome, multiple electrolyte abnormalities, many antiarrhythmic agents (ibutilide, sotalol, and the Class Ia agents, for example), phenothiazines, TCAs,

Table 2. Miscellaneous causes of WCTs[1,19]

Torsade de pointes (Polymorphic VT)
Pacemaker-mediated tachycardia (PMT)
Drug overdose (digitalis, TCAs, lithium,[20] cocaine,[21,22,23] diphenhydramine[24])
Sodium channel blocking agents[25]
Hyperkalemia
Post-resuscitation
Malingering[26] and/or ECG artifact[27]

Table 3. Summary pearls for Wide Complex Tachycardias

- Interpretation of ECGs demonstrating WCTs should be based on clinical circumstances, not the ECG itself.
- Although many algorithms and diagnostic criteria exist to help clinicians correctly interpret WCTs, these are not foolproof. Therefore, clinicians must be comfortable accepting an interpretation of an ECG representing a WCT as "WCT of uncertain etiology." It is important to consider this when selecting therapies for patients presenting with WCTs, as some therapies can cause hemodynamic compromise or cardiovascular collapse.
- Clues may be found in a patient's previous ECG, or from capture and/or fusion beats identified in the ECG or rhythm strip. AV dissociation (if identified) makes the diagnosis of VT much more likely.
- In patients with structural heart disease, especially older patients with previous MIs or previous episodes of VT, the diagnosis of VT is far more likely and should be considered until proven otherwise.

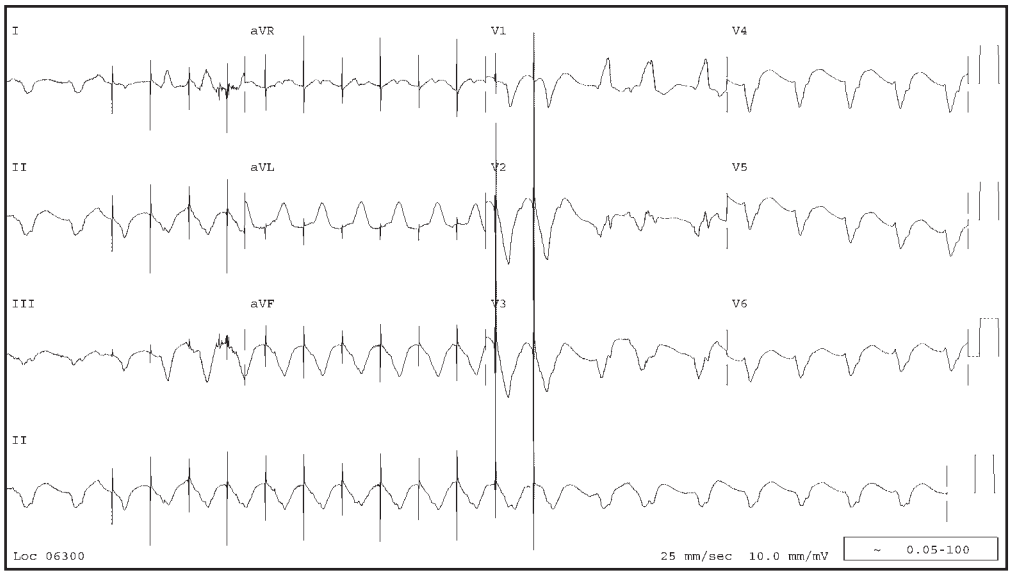

Figure 3. Permanent transvenous pacemaker results in wide QRS complexes.

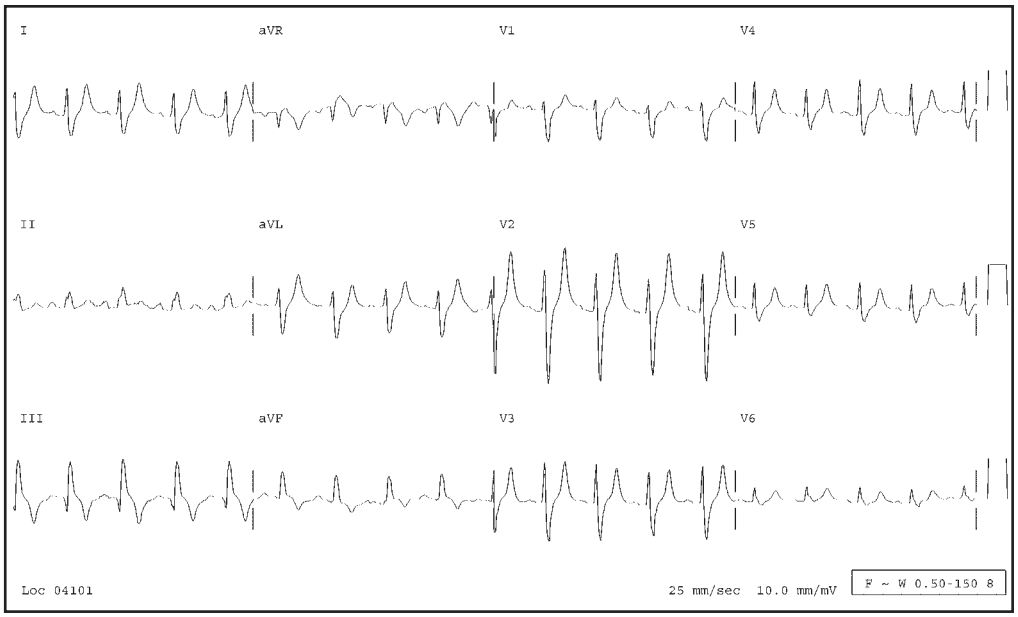

Figure 4. Hyperkalemia causing a WCT in a patient with chronic renal failure (K+ 9.1). The patient's heart rate is 108 bpm, the QRS duration is 134 msec. Note the prominent, narrow peaked T waves (esp. V2, V3), which, with historical factors, should help identify this diagnosis.

and pentamidine.[16,17] In addition to withdrawing the causative agent and correcting electrolyte abnormalities, pharmacologic treatment is with a loading dose of IV magnesium sulfate, followed by infusion. Isoproterenol is no longer recommended as treatment for torsade de pointes, as it increases myocardial oxygen consumption and the likelihood for rhythm disturbances. Overdrive pacing to a heart rate of 90 - 130 beats per minute is generally successful in terminating torsade de pointes if it does not respond to magnesium.

It should be noted that neither lidocaine nor procainamide appear in these updated 2005 AHA ACLS treatment algorithm flow diagrams. Procainamide and sotalol do appear in the accompanying text as alternative drugs for wide complex regular tachycardias.[18]

Miscellaneous WCTs

Several additional causes of WCTs should be considered when evaluating and treating individuals presenting with

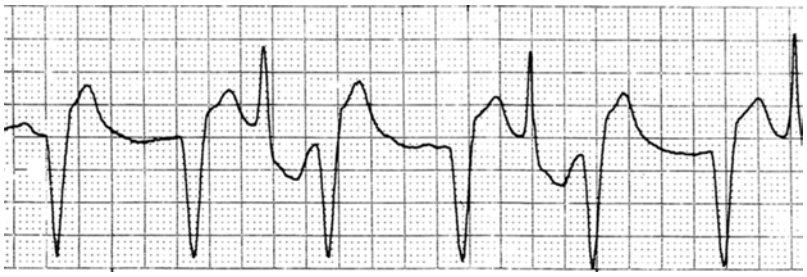

Figure 5a. Bidirectional ventricular tachycardia due to digitalis toxicity. From: Piccini J, Zaas A. Images of Osler: Cases from the Osler Medical Service at Johns Hopkins University. Am J Med 2003 Jul;115(1):70-1. Used with permission.

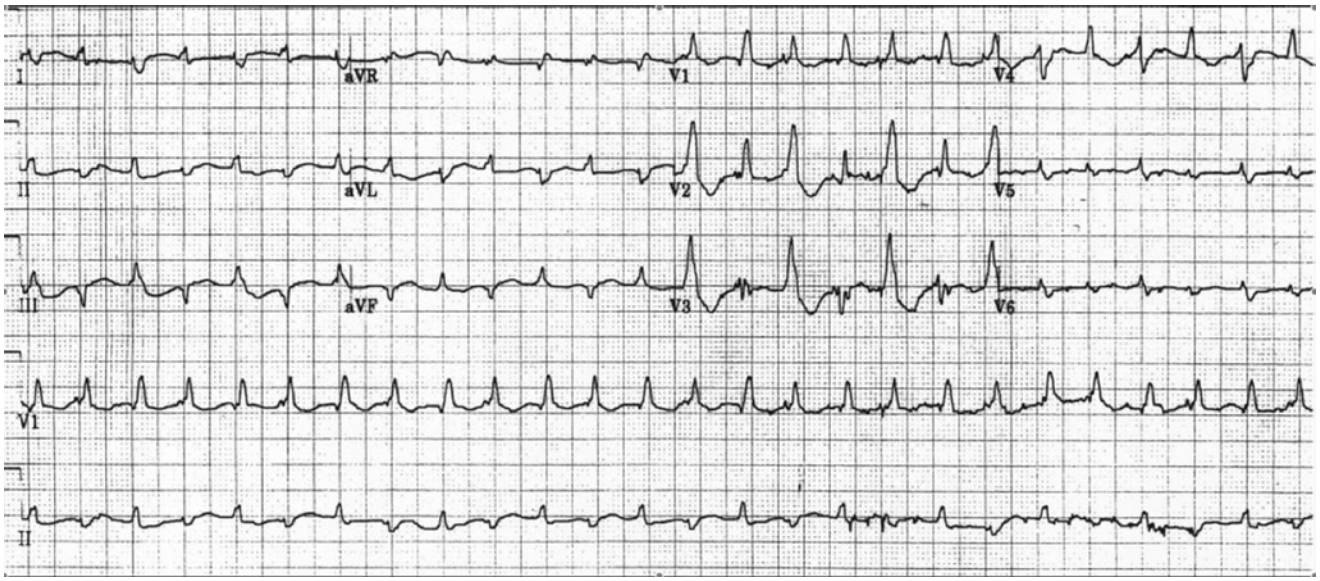

Figure 5b. 12-lead ECG from a different patient demonstrating bidirectional ventricular tachycardia from digitalis toxicity. The axes of the RBBB-morphology QRS complexes alternate. From: Kummer JL, Nair R, Krishnan SC. Images in cardiovascular medicine. Bidirectional ventricular tachycardia caused by digitalis toxicity. Circ 2006 Feb 21;113(7):e156-7. Used with permission.

ECGs demonstrating WCTs (Table 2). Hemodynamic stability remains the main principle directing patient management. These additional causes of WCTs have markedly different treatment strategies, depending on their etiologies.

Pacemaker-mediated tachycardias can be investigated using a pacemaker magnet over the pacemaker generator. As transvenous pacemaker leads are placed in the right ventricle, the QRS complexes generated by the pacemaker are widened. Any pacemaker-mediated tachycardias will therefore be WCTs. Adenosine may elicit hidden atrial activity, and it is particularly safe as the pacemaker should respond if prolonged AV block or a brief period of asystole occurs. With newer pacemakers, this condition is less common.[28] Figure 3 demonstrates a wide complex tachycardia in a patient with a pacemaker, although not pacemaker-mediated.

Tricyclic antidepressant (TCA) overdose causes a WCT due to the quinidine-like effects of these drugs. The class IA sodium channel blocking activity widens the QRS complex, with tachycardia being part of the toxidrome caused by these agents. A high index of suspicion should help identify this clinical scenario. Treatment is with IV bicarbonate, aggressive airway protection, and supportive measures. Agents with similar properties have been reported to result in WCTs due to quinidine-like effects on the sodium channels, especially in toxic ingestions or overdoses.

Severe hyperkalemia may present with WCT as a late ECG manifestation of this condition (Figure 4). A high index of suspicion should cause one to consider this etiology, especially in the setting of renal failure. Initial treatment includes IV calcium to stabilize the myocardium, IV bicarbonate, IV glucose and insulin, and inhaled albuterol. An additional therapeutic regimen includes sodium polystyrene sulfonate (Kayexalate) resin, although this treatment does not take immediate effect and can be challenging to administer.

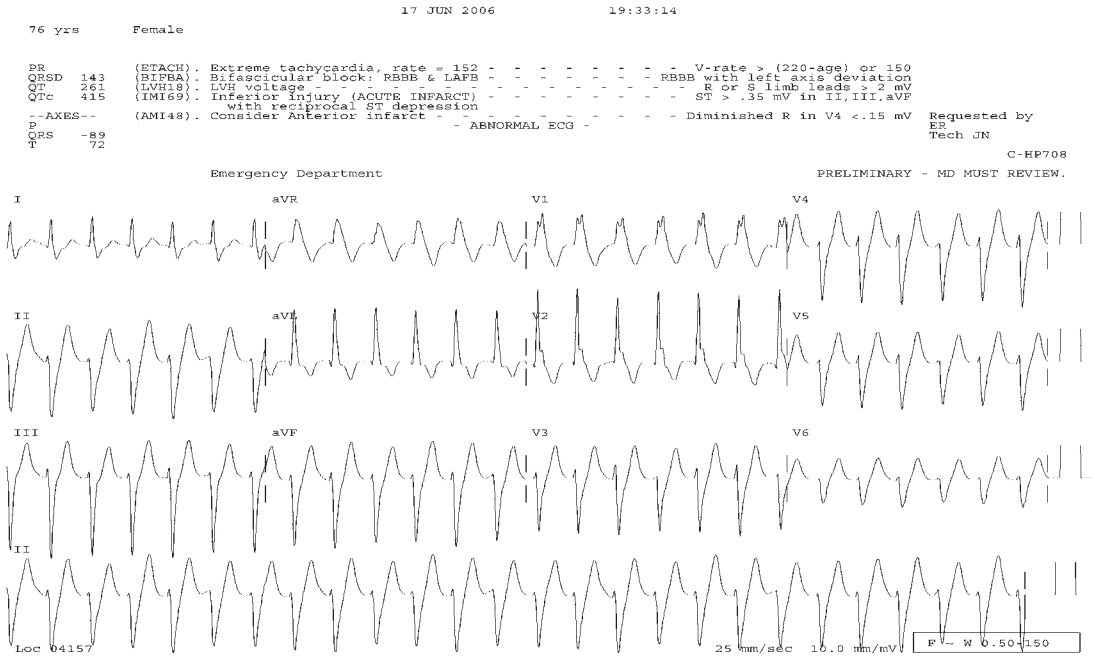

Figure 6. Example of an unusual WCT known as Idiopathic (benign) Ventricular Tachycardia. This is caused by a re-entry loop in the apex of the LV, resulting in the left superior axis and RBBB pattern. Also called Left Ventricular Tachycardia, it often responds to Verapamil. (Note -- CCBs should not be used in WCTs unless recommended by a cardiologist or electrophysiologist.)

Emergent hemodialysis is the definitive treatment for acute hyperkalemia with WCT, and should be arranged as quickly as possible.

Severe digitalis overdose can cause a bidirectional VT.[29,30,31] This WCT generally has a RBBB pattern, with the QRS complexes alternating between right and left axis deviation in the limb leads. This distinct pattern of alternating axis deviation is due to alternating impulse formation in the left posterior and left anterior fascicles (Figures 5a, b). Again, a high index of suspicion should assist in making this diagnosis, as individuals with severe digitalis overdose may have symptoms such as visual changes (especially color vision), changes in behavior, gastrointestinal disturbances, and access to this medication. This condition is more likely in elderly patients with mild dehydration or renal insufficiency, or in individuals following overdose. Treatment includes withdrawal of the offending agent and supportive care; definitive therapy is administration of digitalis-specific antibodies.

SUMMARY

The treatment of patients presenting with wide complex tachycardias should be based on the clinical or hemodynamic status of the individual, not the ECG. The ECG in WCTs is not always helpful in determining the etiology of the rhythm disturbance. It may not even have clinical relevance in the emergent setting.[32,33,34] None of the sets of diagnostic criteria described previously has proven to have 100% accuracy. Therefore, clinicians should always treat the patient, not the ECG, when managing patients presenting with WCTs. Unstable patients require defibrillation or emergent cardioversion (with sedation considered). Pharmacologic treatment of stable patients should occur according to the most updated AHA ACLS guidelines, with "expert consultation" advised. As research continues, newer pharmacologic agents and treatment modalities are likely to become more common in the ED setting.

When the ECG diagnosis of a WCT is uncertain, clinicians should consider it to be VT and treat it as such. Even given referral bias, the literature consistently identifies VT to be much more likely than SVT with aberrant conduction, which makes up part of the rationale behind this recommendation. More important, however, is that treating WCTs of uncertain etiology as rhythms originating above the ventricle may result in clinical deterioration, including cardiovascular collapse. Clinicians should be familiar with agents to avoid when treating individuals who present with WCTs. Furthermore, some pharmacologic agents used to treat VT may in fact terminate a WCT of supraventricular origin. Finally, clinicians must remain vigilant when evaluating and managing patients presenting with WCTs. They must look

for ECGs demonstrating irregular wide QRS complexes, as such ECGs may represent atrial fibrillation with Wolff-Parkinson-White Syndrome (WPWS). Caution should be maintained in patients presenting with a fast, irregular, wide-complex rhythm, as serious consequences may occur from its misidentification or the use of pharmacologic agents that decrease refractory periods in or speed conduction through alternative pathways.

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Takotsubo Cardiomyopathy: A Case Series and Review of the Literature

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Takotsubo cardiomyopathy (TCM) is an unusual form of acute cardiomyopathy showing left ventricular apical ballooning. It is often triggered by intense physical or emotional distress. We report here four cases of TCM and a review of the literature on the topic.
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INTRODUCTION

First described in Japan in 1990 by Sato et al,[1] Takotsubo Cardiomyopathy (TCM) is an acute cardiac condition that involves left ventricular apical ballooning and mimics acute myocardial infarction (MI). It is also known as 'transient left ventricular (LV) apical ballooning syndrome,' 'tako-tsubo-like left ventricular dysfunction,' 'ampulla cardiomyopathy,' 'stress-induced cardiomyopathy,' and 'broken heart syndrome.' In Japanese, "tako tsubo" translates to "octopus pot," which is a fishing jar with a narrow neck and wide base used to trap octopus, and describes the visual appearance of the heart on left ventriculography.

TCM patients present with symptoms of chest pain, electrocardiograph (ECG) ST-segment elevation, and cardiac markers consistent with an acute coronary syndrome (ACS). However, angiography finds no significant coronary stenosis, and the LV apex is found to balloon, which usually resolves in weeks. The syndrome appears to be triggered by emotional or physical stress. TCM is being diagnosed more frequently, possibly because of increasingly stressful times and public attention to ACS. Although well known in cardiology, awareness of this entity is still developing in emergency medicine (EM). It is important to consider this diagnosis, as patients may present to the emergency department (ED) with what appears to be uncomplicated ACS. We discuss four patients with chest pain, ECG changes and cardiac markers consistent with ACS, who were diagnosed with TCM.

CASE 1

A 67-year-old Caucasian female was sent from a clinic by ambulance to the ED for chest pain longer than 24 hours and ST elevations on ECG. Her chest pain began the previous day while mowing her lawn. It was 5/10, "stinging" in character, located substernally, and associated with shortness of breath (SOB), and radiated to her left arm. After rest, her SOB and chest pain resolved over one hour, but she continued to have intermittent chest discomfort throughout the day and again the next morning. At the primary care physician office, ECG was "concerning."

In the ED, vital signs were: blood pressure (BP) of 140/86 mm Hg, pulse of 86 beats/min, respirations of 14 breaths/min, oxygen saturation (O_2 sat) of 100% on 2 liters/minute via nasal cannula, and temperature of 37.1°C. She denied active medical issues but reported she had a rib resection in 1970 for thoracic outlet syndrome. Cardiac risks included age and 20 pack-year smoking history. When asked about family history for heart disease, she tearfully stated that the day before onset of symptoms, her sister had died of a heart attack at age 64. Apart from anxiety, her physical exam was otherwise unremarkable.

Initial ECG showed normal sinus rhythm (NSR), rate of 79 beats/min; small Q waves in inferior leads with T-wave inversions in leads II, III, and AVF; a trend toward ST elevation in lead V3; poor R wave progression; Q waves in V4 and V5, with a very small Q wave in V6; and deep T-wave inversions in V3, V4, and V5, with a small degree of T wave

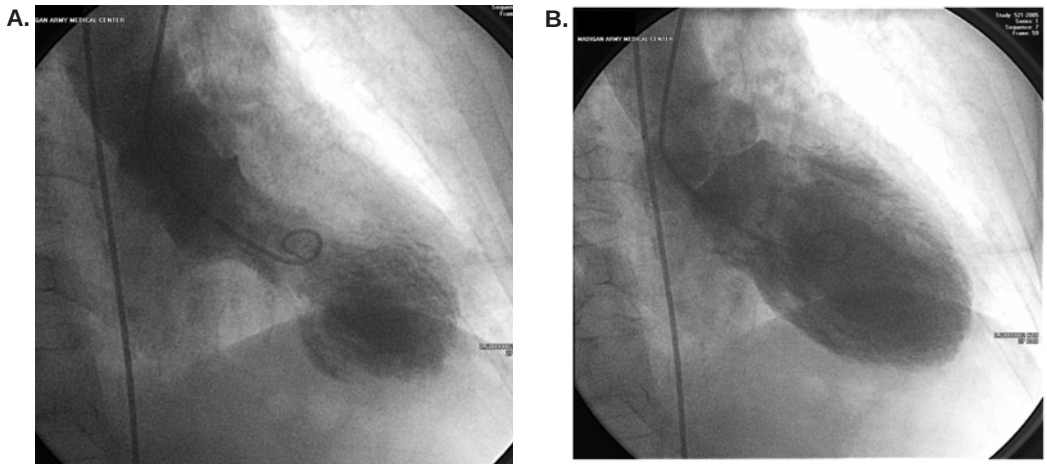

Figure 1. Cardiac catheterization demonstrating the patient's left ventricular severe anterior-apical akinesis with compensatory inferior-posterior hyperkinesis during systole (A) and diastole (B).

inversion in V6. A chest x-ray revealed no acute disease. Lab results were troponin I of 2.5 ng/ml (normal, 0.0-0.04 ng/ml), creatine kinase (CK) of 24 ng/ml (normal for females, 30-135 U/L; for males, 55-170 U/L), CK-MB quotient of 11 (normal 0.3-4.0 ng/ml), and a white blood count 11,200/mm^3. Serum electrolytes and coagulation studies were normal.

The patient had immediate cardiac catheterization that revealed no source of cardiac ischemia. Left ventriculography revealed significant apical segment akinesis. Follow-up echocardiogram seven days later showed normal LV size and function with ejection fraction (EF) of 78%. Apical wall motion was normal, but the base still appeared to contract more vigorously than the apex.

CASE 2

An 86-year-old Asian female presented to the ED with chest pain of ten hours duration, which began while watching television. The pain was 9/10, substernal and non-radiating, pressure-like, without associated symptoms. Vital signs were: BP of 185/88 mm Hg, pulse of 71 beats/min, respirations of 20 breaths/min, O2 sat of 98% on room air, and temperature of 35.7°C. She reported medical history of hypertension, hypothyroidism, gout, and a hysterectomy. Cardiac risks included age, hypertension, and family history of coronary disease. She never smoked and reported a healthy diet and exercise five times per week. Her daughter stated that her mother had been under extreme stress due to sudden accidental death of her son two weeks ago. Her physical exam was unremarkable.

Initial ECG revealed a NSR with ST elevation in V2 and V3, ST depression in V4 and V5, and T wave inversions in inferior and precordial leads. Chest radiograph revealed no acute disease. Lab studies showed troponin I of 3.23 ng/ml and a white blood cell count 12,000/mm^3. Serum electrolytes were normal.

She had cardiac catheterization with left ventriculography, which showed mid-anterior and apical akinesia with preserved anterobasal and posterobasal function, with an EF of 30%. Coronary arteries were unremarkable Echocardiogram showed apical akinesis with reduced LV function with EF of 34%. The mid septum showed marked hypertrophy with a thinned apex.

CASE 3

A 76-year-old Caucasian female presented to the ED with chest pain of 30 minutes duration. This began 20 minutes after she received the news that her husband was in critical condition in the intensive care unit (ICU) and needed emergent surgery. The pain was described as 5/10 nonradiating, substernal, with associated lightheadedness.

Vital signs were: BP of 110/65 mm Hg, pulse of 72 beats/min, respirations of 24 breaths/min, O2 sat of 96% on room air, and temperature of 36°C. Past medical history included osteoporosis and mitral valve prolapse. Cardiac risks included age and 45 pack-years smoking, but she quit 28 years ago. Her only medication was alendronate. Physical exam revealed a 2/6 systolic cardiac at the apex.

Initial ECG showed NSR with ST elevation in leads II, III, AVF, V2-V6, poor R wave progression, and Q waves in V2 and V3. A right-sided ECG showed no ST segment elevation in V4R. Chest radiograph showed mild pulmonary edema. Complete blood count and electrolytes were normal, but troponin I was elevated at 0.15 ng/ml.

The patient immediately had cardiac catheterization with left ventriculography. This revealed EF of 20% with only basal kinesis. Coronary arteries were unremarkable. Cardiac catheterization (Figure 1) showed severe anterior-apical akinesis with compensatory inferior-posterior hyperkinesis.

CASE 4

A 42-year-old Caucasian female presented with a two-

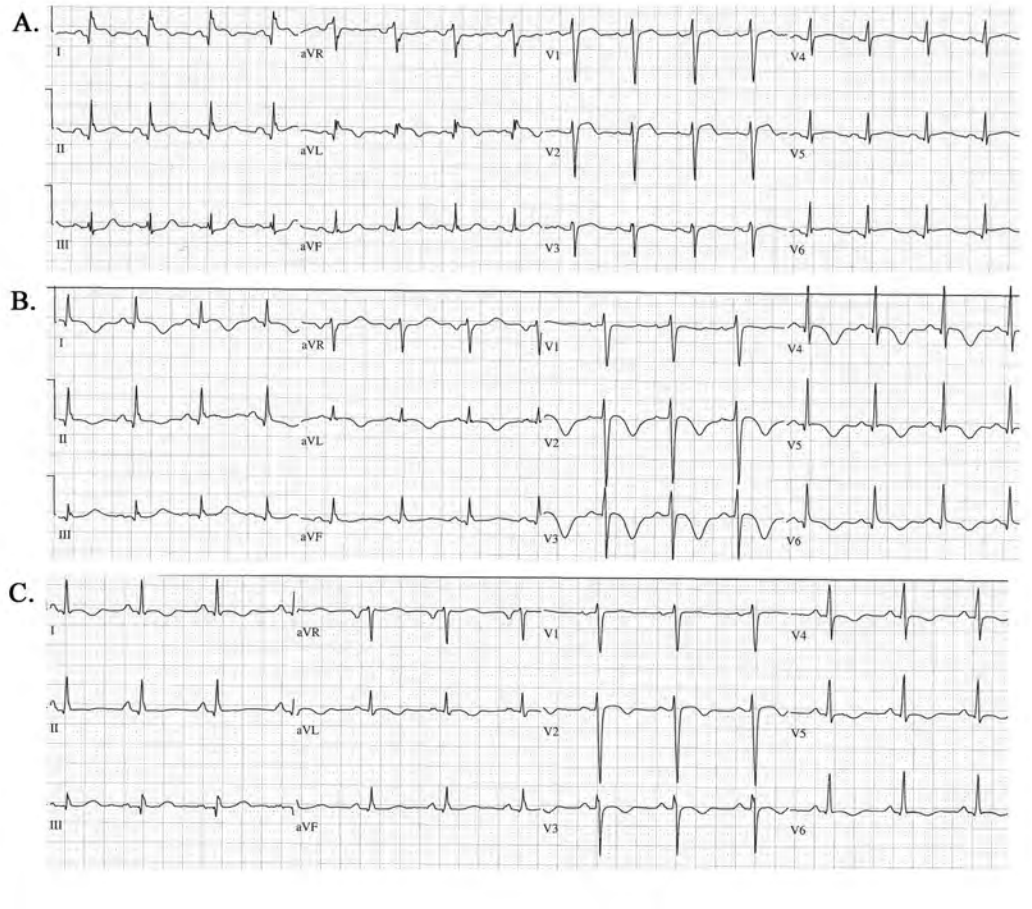

Figure 2. Electrocardiograms of patient showing (A) ST-segment elevation is leads V1-V3, I and aVL at 15 minutes after presentation, (B) T-wave inversion in leads V2-V6, I and aVL and a lengthened QT at one day after presentation, and (C) improvement of the T-wave inversion and normalization of ST-elevation at four days after presentation.

hour history of chest pain. She had just been told that one of her sons had died in a car accident and had visited her other son who was in the (ICU) in critical condition. Her chest pain began during the ICU visit and she was brought to the ED. She had a history of seizures, anxiety disorder, fibromyalgia, irritable bowel syndrome, and gastroesophageal reflux disease. The patient had one other episode of chest pain three months prior, but MI was ruled out.

In the ED, vital signs were: BP of 101/78 mm Hg, pulse of 109 beats/min, respirations of 20 breaths/min, O2 sat of 97% on room air, with pain 10/10. She was awake and alert, diaphoretic and vomited three times. The physical exam was normal. An ECG showed ST elevation in leads I, aVL, and V_1-V_3, with a rate of 98 beats per minute and a QTc interval of 437 ms (Figure 2a).

Initial cardiac markers were elevated with a troponin I of 5.1 ng/ml (normal, 0.0-0.3 ng/ml), a total CK of 173 U/L (normal, 25-145 U/L), and a CK-MB level of 24.7 ng/ml (normal, 0.0-5.0 ng/ml).

Emergent catheterization revealed normal coronary arteries, with a right-dominant system, and severe hypokinesis of the LV anteroapical wall, and mid to distal septum with apical ballooning. Echocardiograms performed at day 1 (Figure 3) and 4 showed a severely hypokinetic LV anteroapical wall and mid-to distal septum.

DISCUSSION

Etiology

Although the clinical presentation of TCM may be identical to ACS, the suspected pathogeneses of TCM and ACS differ greatly. The etiology of Takotsubo cardiomyopathy remains speculative. Proposed mechanisms include: (1) multivessel coronary artery spasm, (2) impaired cardiac microvascular function, and (3) endogenous catecholamine induced myocardial stunning and microinfarction.

Lacy et al[2] have shown that during a mental stressor of simulated public speaking patients have a decrease in coronary artery diameter, which returned to baseline after five minutes. Furthermore, these changes were similar in patients with and without coronary artery disease. Physicians who originally reported on and named 'Takotsubo cardiomyopathy' have

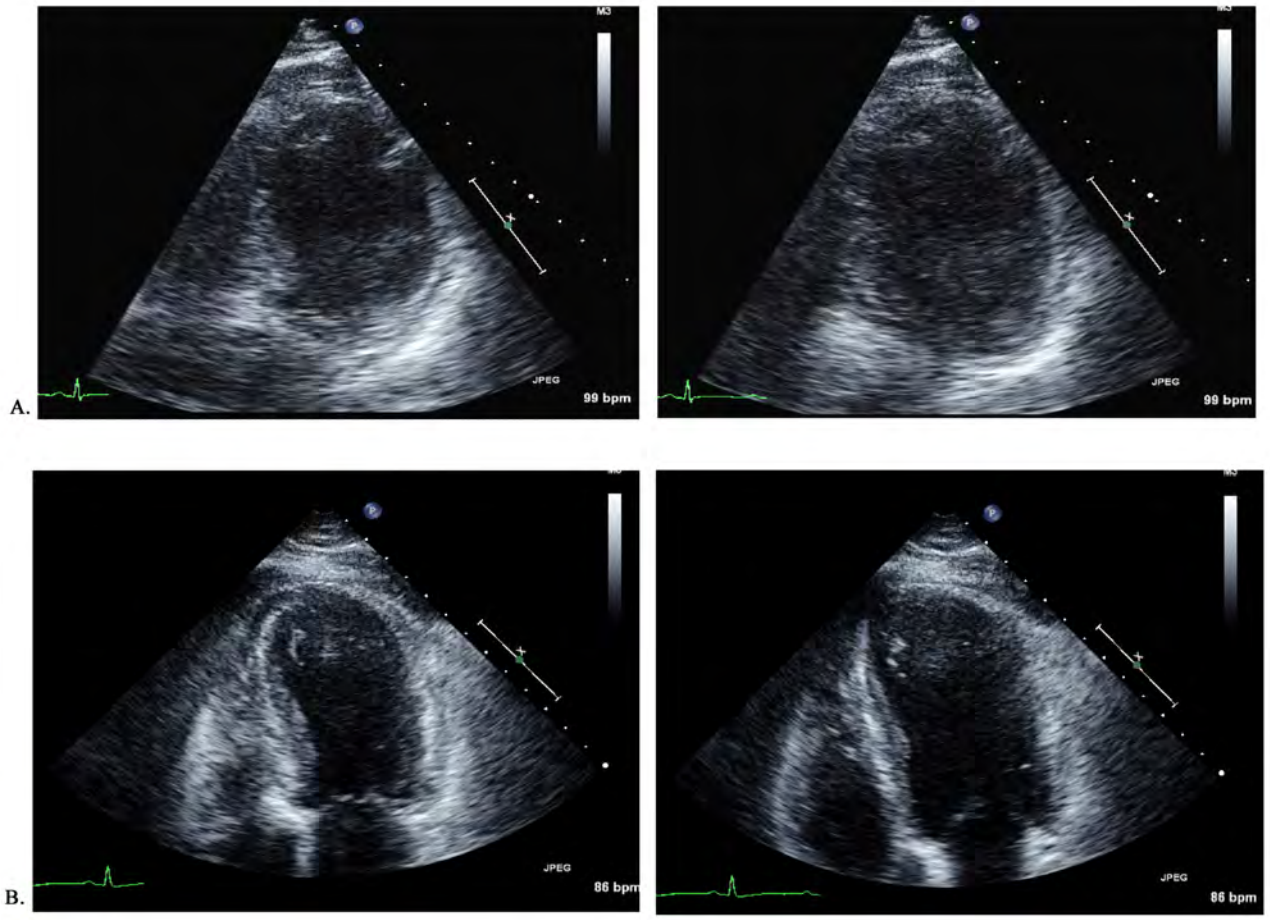

Figure 3. Transthoracsic echocardiogram showing (A), on day one, a severely hypokinetic left ventricular anteroapical wall and mid to distal septum during systole (left) and diastole (right) and (B) on day four, some minor improvements to the wall motion abnormalities during systole (left) and diastole (right).

also shown that approximately 70% of TCM patients had coronary spasm during a provocation test.[3] They suggested that either simultaneous multivessel coronary artery spasm or microvascular spasm may contribute to the onset.

Sadamatsu et al[4] reported two cases of patients with apical wall motion abnormalities with reduced coronary flow reserves not due to coronary stenosis. They postulated that this reduction in reserve may be due to microvascular dysfunction. Kurisu et al[5] and Bybee et al[6] showed that patients with transient LV apical ballooning syndrome have higher thrombolysis in myocardial infarction (TIMI) frame counts during the acute period than control patients. An elevated TIMI count has been associated with increased coronary microvascular resistance.[7] Bybee et al[6] suggested that the dysfunction of coronary microvasculature may play a role in the pathogenesis of transient LV apical ballooning syndrome. Single photon emission computed tomography (SPECT) studies of TCM patients also show impaired myocardial microcirculation.[8] Other studies have also shown that mental stress may cause endothelial dysfunction, further supporting the idea that coronary microcirculation may play a role in the etiology.[9,10] However, at this time, it is unclear whether the microvascular dysfunction is a cause or result of the cardiomyopathy.

The most commonly discussed mechanism for this condition is stress-induced catecholamine release. They may be a direct toxic effect on the myocardium by changing autonomic tone, enhancing lipid mobility, calcium overload, free radical production, or increased sarcolemmal permeability.[11] Akashi et al[12] suggested that neurogenetically mediated myocardial stunning due to autonomic imbalance may be the cause of the LV motion abnormalities. They have shown that patients with TCM had heart rate variability (HRV) parameters consistent with impaired cardiac sympathetic nervous function at time of diagnosis. These patients showed improvement in the HRV parameters at three months follow up. Wittstein et al[13] showed that stress-induced cardiomyopathy patients have statistically higher levels of catecholamines (epinephrine, norepinephrine, and dopamine) than patients with myocardial infarctions. These elevated

levels may cause myocardial stunning in TCM patients. The catecholamine levels however, were not known prior to the episode of TCM or MI.

Ellison et al[14] showed that high doses of isoproterenol, a β1/β2 adrenergic stimulator, cause diffuse myocyte death but spare cardiac stem cells in rats. They postulated that the sparing of stem cells may contribute to the ability of myocardium to rapidly recover from acute hyperadrenergic damage, which may be the case in TCM. Bybee et al[15] reported a decreased myocardial glucose uptake in TCM patients and hypothesize this may be due to myocardial insulin resistance due to catecholamine excess. Akashi et al[16,17] reported an increased myocardial ^{123}I-metaiodobenzlguanidine (^{123}I-MBG) washout rate (WR) in TCM patients. An increased WR indicates increased norepinephrine release from sympathetic nerve endings or increased clearance of ^{123}I-MBG by extraneural tissues. The increased WR also correlated to increased plasma norepinephrine levels in the patients, further supporting the idea of catecholamine-induced myocardial stunning.

Presentation and Clinical Course

Although TCM was first named in the early 1990s in Japan, cases in Europe were not recognized until the late 1990s and not in the United States until 2003.[1,18-20] Patients with TCM often present to the ED complaining of chest pain and a standard approach is prudent. It is important to remember that TCM patients may not have cardiac risk factors, except age. These patients may have had a recent normal stress test or normal cardiac catheterization, as they do not have obstructive coronary lesions. However, more serious presentations of TCM such as ventricular fibrillation or cardiogenic shock have also been reported.[21,22]

The epidemiology of TCM reveals several trends. Patients are typically postmenopausal Asian or Caucasian women, but investigators do not report a racial predilection. In 286 reported cases of TCM, Gianni et al[21] reported 88.8% of patients were female. In 185 cases, Donohue and Movahed[22] reported age 67.7 years. Of the cases that reported race, 57.2% of patients were Asian, 40% Caucasian, and 2.8% other races.

The prevalence of the syndrome is unknown, although various researchers have reported that 1.7-2.2% of patients with suspected ACS at their institutions are diagnosed with TCM.[6,8,17,23] An emotional or physical stressor frequently precedes the development of symptoms (26.8% and 37.8%, respectively).[21] These stressors have included: learning of the death of a loved one, bad financial news, significant arguments, legal problems, car accidents, natural disasters, exacerbation of chronic medical illness, new significant medical diagnosis, surgery and other medical procedures, staying in an intensive care unit, and use of or withdrawal from illicit or narcotic drugs.[21,22,24-28] In some cases no stressor is identifiable.[22] However, patients with traditional ACS could also have proximate stressful events, but these have not been similarly quantified.

All the patients in our series had loss of a family member as a major stressor. Family and social history have not yet shown to be relevant to TCM diagnosis. Physical exam may only be significant for emotional upset. Initial ED testing should include ECG, cardiac markers basic labs, and chest radiography.

The ECG often reveals ST elevation (often precordial) during the acute phase, followed by T wave inversion, QT prolongation, and sometimes Q waves during the subacute phase.[21,29,30] A few studies have shown minor ECG differences between patients with TCM and ACS.[29,31] Cardiac markers are usually elevated. However, the levels tend to be lower and normalize sooner than with ACS patients.[21,22] Angiography is required for diagnosis, as there is no accurate way to reliably distinguish TCM from ACS using ECG or cardiac markers.

Patients should be treated as having ACS until proven otherwise. Aspirin and heparin should be initiated, and a cardiologist consulted. The diagnosis of TCM will be made if PCI is the initial treatment. Although thrombolysis will not benefit these patients, it should not be withheld if PCI is not available and the patient otherwise meets criteria.

Coronary angiography, echocardiography, or ventriculography of TCM patients during the acute phase reveal left mid-ventricular dysfunction and apical dyskinesis or akinesis with apical ballooning. Mean EF for these patients range from 20-49%.[21] LV basal hyperkinesis also occurs, and one systematic review reported 16% with transient LV outflow tract obstruction (LVOT) due to this hyperkinesis.[21] Coronary arteries are either normal or show mild stenosis (<50% luminal stenosis).[21,30] On follow-up studies, patients show rapid improvement in wall motion and restoration of LV function with improved EF of 60-76%.[21]

Acute complications occur in approximately 20% of TCM cases and include cardiogenic shock, left-sided heart failure with or without pulmonary edema, tachy- and bradyarrhythmias, LV thrombus formation or free wall rupture, and death.[21-23,30,32-36] Cardiogenic shock results from pump failure or LVOT obstruction, which can cause severe mitral regurgitation. For hypotensive patients, urgent echocardiography should be performed.[30] If hypotension is due to pump failure, inotropic agents should be initiated. If necessary, intra-aortic balloon counterpulsation has been utilized in TCM patients.[33] It is important to recognize and diagnose TCM-related cardiogenic shock, and treat with the balloon pump because TCM patients fare much better than other causes of cardiogenic shock. If the hypotension is due to LVOT obstruction, inotropic agents will worsen the obstruction. Instead, beta-blockers and fluid resuscitation are the initial treatment.

The long-term therapy of TCM has been that of cardiomyopathy with LV systolic dysfunction: beta-blockers, ACE-inhibitors, and diuretics. Aspirin and calcium channel blockers have also been recommended.[30] However, Fazio et al[37] found that treatment with any of these medications does not make a significant difference in outcome. Prognosis for patients with TCM is good for those who survive the acute episode. Mortality rates have been reported by Gianni et al[21] and Donohue and Movahed[22] at 1% and 3.2%, respectively. Recovery time for TCM patients is generally rapid. Marked improvement in ECG findings, cardiac markers, and EF can be seen within days.[13,17,26,38] Complete recovery of LV usually occurs within 1-4 weeks, although some have taken up to a year.[32] Recurrence appears to be rare at 2-3%. However, as this is a newly described entity, accurate long-term data is not yet available.[21,33]

Diagnostic criteria for TCM have not been established. However both American and Japanese researchers have proposed similar criteria for the diagnosis of TCM. These authorities stress the important findings of: (1) Transient akinesis or dyskinesis of the apical and midventricular segments in association with regional wall motion abnormalities that extend beyond the distribution of a single epicardial vessel; (2) immediate ST segment elevation on ECG followed by T waves that become progressively more negative and a prolongation of the QT interval; (3) only a modest elevation of cardiac markers; and (4) absence on angiography of obstructive coronary artery disease or evidence of acute plaque rupture. The groups also put forth similar exclusion criteria: (1) recent significant head trauma or intracranial bleeding, (2) cerebrovascular disease, (3) pheochromocytoma, and (4) myocarditis.[30,39] These criteria have not been validated, but can be used as guidelines that, when present, should raise suspicion of TCM.

Several ED circumstances may lead to TCM presentation. The ED deals with the consequences of natural disasters, and emergency physicians (EP) should be aware that this syndrome might present during or soon afterward. The syndrome may also occur in younger patients without cardiac risk factors. Some anxious or emotionally distraught patients with chest pain may be more complicated than simple anxiety disorder, and may develop dysrhythmias or shock.

The emotional distress and chest pain of family members after death notification are sometimes dismissed or managed with benzodiazepines and social work consult. While much has been written on the emotional support needed for survivors after death notification in the ED, we found no study that looked at the medical management of physical symptoms of these survivors.[40-44] EPs should consider TCM in the differential diagnosis of chest pain following death notification. Increased awareness of this entity will contribute to timely diagnoses and appropriate treatment.

History

Although TCM was named in the early 1990s the idea of a stress-induced physical disorder or death has been in the literature for decades. In 1942 Walter B. Cannon[45] wrote a remarkable article detailing numerous accounts of so-called "Voodoo death" reported by educated and independent observers. The cases of Voodoo death occurred among many different aboriginal tribes throughout the world. The cases involved people who were "hexed" or condemned to death by medicine men or Voodoo priests. In all cases the condemned person and all his/her family and associated believed there was no escaping the death that was sure to ensue. In most of these cases poison was ruled out or unlikely as the cause of death, and Cannon postulated that intense fear could so over-activate the sympathetic and sympatho-adrenal systems that death ensued.[45]

In 1971 Engel[46] reported on 170 cases of sudden death associated with psychological stress. He identified eight categories in which these deaths could be classified: learning of the death of a loved one, during the acute grief period, threat of loss of a loved one, during mourning or an anniversary, on loss of status or self-esteem, threat of or actual physical danger, and reunion, triumph, or happy ending. All these instances represent a situation in which the person is overwhelmingly distraught or excited and are very similar to the precipitating events reported in TCM. Engel postulated that these deaths may involve activation of both a strong sympathetic and parasympathetic response and lead to lethal cardiac events.[46]

Cebelin and Hirsch[47] reported in 1980 the post-mortem analysis of myocardium from 15 victims who died from physical assault but whose autopsies revealed no internal injuries sufficient to cause death. Many of these patient's (73%) hearts showed myofibrillar degeneration and "contraction band" necrosis. Cebelin and Hirsch[47] postulated that the cause of death could have been a catecholamine mediated "stress cardiomyopathy." Interestingly, Wittstein et al.[13] have reported very similar endomyocardial pathologies in TCM patients. Although the mortality rate in diagnosed TCM is low, it is possible that some of these sudden unexplainable deaths during intense emotional distress represent a more-fatal version of TCM.

CONCLUSION

As a newly recognized disorder, much remains unknown about TCM, especially etiology. Other aspects are also puzzling, such as why postmenopausal women are mostly affected and why the apex of the LV is so impaired while the remainder is relatively spared. TCM is a rare but potentially fatal condition, initially indistinguishable from ACS. The EP should consider the diagnosis in patients with chest pain and a recent stressful event, especially elderly females.

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Chest Swelling and Fever in an Intravenous Drug User

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This case report describes a sternoclavicular infection in an IV drug user. The history and physical exam suggested an abscess. In the emergency department (ED) the patient refused incision and drainage but did consent to simple needle aspiration. Subsequent culture of the aspirate revealed Pseudomonas aeruginosa. He was admitted for IV antibiotics. After admission, a bone scan suggested the presence of osteomyelitis. The patient refused operative débridement, but ultimately did consent to bedside incision and drainage. By day five, the fever had resolved and the patient signed out AMA. He was given a prescription for Ciprofloxacin. The patient had an unscheduled follow up in the ED five months later for an unrelated heroin overdose. Physical examination demonstrated complete resolution of the infection.
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INTRODUCTION

Bacterial septic arthritis is dangerous and destructive. Early intervention is key to prevent irreversible joint damage and hematogenous spread. In the United States, the most common etiology of septic arthritis is *Neisseria gonorrhea*.[1] Sternoclavicular joint and sacral iliac joint infections are associated with intravenous drug use (IVDU). Methicillin-resistant *Staphylococcus aureus* (MRSA) has surpassed *Pseudomonas aeruginosa* as the most common cause of sternoclavicular septic arthritis.[2] While one case of sternoclavicular septic arthritis due to *Staphylococcus aureus* has been published in the emergency medicine literature,[3] we present the first case of septic arthritis of the sternoclavicular joint due to *Pseudomonas aeruginosa.*

CASE REPORT

Chief Complaint

"I'm growing a breast."

HISTORY

A 28-year-old man presented to the emergency department (ED) with a painful fluctuant swelling in the left anterior chest. He first noticed the swelling one week prior when a friend joked he was growing a breast. Since that time, the area has become progressively more swollen. He also reported fevers and chills for the past two days. The pain was worse with arm movement. The patient has used IV heroin for three years, most recently just prior to the ED visit, and most commonly injected his arms. The patient reused his needles after cleaning them in vodka. He denied any IVDU in the neck or chest and had never had similar swellings in the past. He denied skin popping and pocket shooting. Past medical history was unremarkable. The patient had never been tested for hepatitis, HIV, or sexually transmitted diseases. Last tetanus toxoid was two years ago. He denied sharing needles.

PHYSICAL EXAM

The patient appeared in mild discomfort. Vital signs were pulse of 102 beats per minute, blood pressure of 140/72 mm Hg, respiratory rate at 18 breaths per minute and temperature 99.6 °F orally.

The head exam was normal; the neck revealed several small (<1cm) mobile lymph nodes primarily on the left side of the neck anterior to the sternocleidomastoid muscle. Chest exam revealed a 3 x 4 cm tender, fluctuant, nodular mass in the area of the left sternoclavicular joint (Figure). There was no overlying erythema or other skin lesions on the chest. The cardiovascular exam revealed no murmurs; peripheral pulses were strong and equal. The abdominal exam revealed normal bowel sounds; it was soft and non-tender with no organomegaly. The extremities had track marks, but no evidence of abscesses or cellulitis. Movement of the

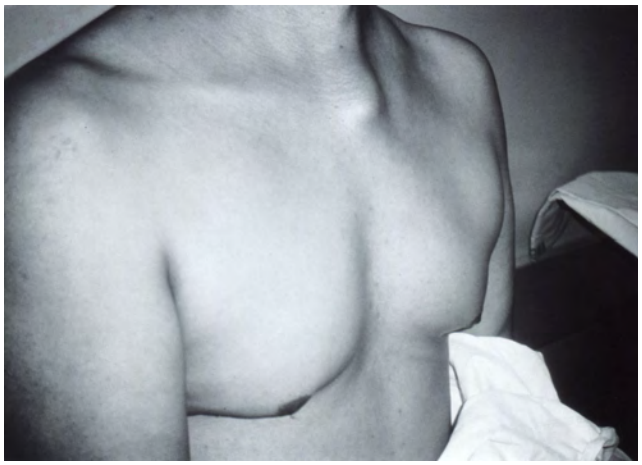

Figure.

left arm caused pain localized to the swelling. There was no physical exam evidence of embolic phenomena (no Osler's nodes, Janeway lesions, splinter hemorrhages, Roth spots, or conjunctival petechiae.)

DIAGNOSTIC TESTING

The CBC revealed a white count of 13, 500/mm³. The hemoglobin was 13.5 g/dl. Platelets were 459,000/mm³. The sedimentation rate was 97 mm/hr. Electrolytes, liver panel, renal function and urinalysis was normal. A chest radiograph was normal with no bony destruction in the area of swelling. The patient refused incision and drainage of the suspected abscess. After a lengthy discussion and education on the increasing frequency of resistant organisms found in abscesses, he consented only to a diagnostic needle aspiration. Using sterile technique, the area was prepped and draped. Two cc's of 1% lidocaine was infiltrated subcutaneously. Then, an 18G needle was easily inserted into the center of the abscess yielding 12 cc of purulent fluid. Diagnostic studies of the aspirate included a gram stain, cell count, and culture and sensitivity. Cell count revealed 78,000 WBCs /mm³ and gram stain showed gram-negative rods. Wound culture and two blood cultures were sent.

DIAGNOSIS

Sternoclavicular septic arthritis and abscess.

TREATMENT

In the ED, the patient received IV vancomycin and ceftazidime. After admission, the orthopedic service incised, drained, and irrigated the abscess at the bedside. The initial aspirate was positive for *Pseudomonas aeruginosa*, sensitive to ceftazidime and ciprofloxacin. Blood cultures were negative. An echocardiogram revealed no valvular abnormalities or vegetations. HIV, hepatitis B and C testing were negative. A bone scan revealed osteomyelitis in the area of the left sternoclavicular joint and clavicle. Orthopedics further recommended surgical irrigation and débridement of the joint space, but the patient refused. The fever subsided on hospital day three and the patient signed out AMA on day five, with a prescription for 30 additional days of oral ciprofloxacin. He did not follow up at the clinic.

Although he missed his clinic follow up, he did return for further care five months later for a subsequent overdose that required treatment with naloxone. When lucid, he reported taking the entire course of Cipro because he did not want to get "shooter's heart." He no longer had pain or swelling in the area, or any fevers, and said he felt fine (except for the acute withdrawal symptoms).

DISCUSSION

This case illustrates the difficulty in the evaluation of some ED patients. Many IVDUs are reluctant to cooperate with a complete medical evaluation for a number of reasons: fear of narcotic withdrawal, drug intoxication, lack of health insurance, social stigma, just to name a few. Signing out against medical advice is a common problem in this population. These patients often have serious medical problems and can present a high liability to the emergency physician. Patient education is critically important. Additionally, infections in this patient population require rapid evaluation and early antibiotic administration due to the high morbidity and mortality.

Sternoclavicular abscess is a potential complication of septic arthritis of the sternoclavicular joint,[4,5] found in 22% of cases. The mean age is 45 years, with 74% male.[2] Patients often present with a painful swelling over the sternoclavicular joint preceded by chest pain or pain referred to the neck or shoulder. Fever and leukocytosis are of little help, as 35% are afebrile and 44% have a normal WBC.[2] An erythrocyte sedimentation rate (ESR) is more sensitive at 91%,[6] but merely indicates inflammation not specific to any organism.[7] Bacteremia occurs in 62% of patients with sternoclavicular septic arthritis, with osteomyelitis occurring 55% of the time, and mediastinitis, 13%. Currently, *Staphylococcus aureus* is the most common bacteria in sternoclavicular abscess formation in IVDU patients. However, this represents a shift, as *Pseudomonas aeruginosa* was responsible for the bulk of cases (82%) prior to 1981, whereas *Staphylococcus aureus* caused most cases (77%) after 1981.[8]

Two factors may help explain why pseudomonas was the most common gram-negative pathogen, and why, more recently, S. *Aureus* has dominated. First, the IVDU drug of choice prior to 1981 was commonly pentazocine, which was frequently injected with the adjuvant, tripelennamine. This first-generation ethylenediamine antihistamine was thought to intensify the "rush" associated with pentazocine injection. One study found that tripelennamine favored the growth and survival of certain serotypes of pseudomonas.[8] This, in combination with the routine use of contaminated water

sources to clean needles,[9,10] made pseudomonas the most common organism causing sternoclavicular septic arthritis in IVDU patients prior to 1981.[2]

Second, a shift in the illicit drug of choice from pentazocine to heroin more recently may have contributed to the observed shift in bacteriology. Prior to the 1980s, the commonly used pentazocine did not require heating prior to injection. Therefore, pseudomonas remained the more common offending organism. Heroin has now surpassed pentazocine use.[11] Because heroin requires heating prior to injection, bacterial contaminants are eliminated. As a result, skin pathogens predominate as the most common organisms. These factors, in our opinion, help to explain both the historical predominance of *Pseudomonas aeruginosa* as the cause of sternoclavicular septic arthritis, as well as its replacement as the predominant organism by staphylococcus.[2] Thus, this case illustrates a declining but still important diagnostic consideration.

In a recent study *Staphylococcus aureus* was responsible for half of all sternoclavicular joint infections, while *Pseudomonas aeruginosa* was responsible for only 10%.[2] The reason for this predilection to the sternoclavicular joint may be the combination of surface proteins, which bind connective tissue and the proximity of the subclavian vein.[11] Intravenous drug use is the most common predisposing risk factor for sternoclavicular septic arthritis (21%), followed by upper extremity infection (15%), diabetes mellitus (13%), trauma (12%) and subclavian catheters (9%). However, almost one quarter of patients with sternoclavicular septic arthritis have no risk factors.[2]

The presentation of septic arthritis does not inform the etiology of the infection. However, *Pseudomonas aeruginosa* can give rise to blue-green pus. Occasionally, a breakdown product of hemoglobin called verdoglobin can be detected in pseudomonas infections by ultraviolet light in suspect wounds.[12] Pseudomonas also has a characteristic "fruity-musty" odor.[13] The most specific lesion for a pseudomonas infection is ecthyma gangrenosum, which is a hemorrhagic necrosis of skin that does not contain pus, and is due to pseudomonas infection.[12]

Treatment includes incision and drainage of a suspected sternoclavicular abscess, as well as wound and blood cultures (at least three if endocarditis is suspected). In this case, the patient would not allow incision and drainage of the abscess. Surgical incision and drainage is considered the definitive treatment of a soft-tissue abscess.[14] Diagnostic needle aspiration is recommended if one is unsure of pus localization. It should not be considered a substitute for incision and drainage.[15]

The evaluation and management of suspected soft-tissue infections and abscesses in febrile IVDUs must be done rapidly. Diagnostic evaluation includes lab tests, blood and wound cultures, and incision and drainage of the abscess. The timely administration of empiric antibiotics should include coverage for MRSA and pseudomonas species (e.g. vancomycin and ceftazidime). Gram stain and culture of the sternoclavicular abscess will help guide antibiotic selection, especially with the increasing frequency of resistant organisms such as methicillin resistant *Staphylococcus aureus* (MRSA). Surgical debridement or resection should be considered if imaging studies detect osteomyelitis or spread into adjacent structures.[1]

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Subchorionic Hemorrhage Appearing as Twin Gestation on Endovaginal Ultrasound

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This case study describes a pregnant patient with vaginal bleeding who had a bedside endovaginal ultrasound in the emergency department (ED). The emergency physician identified a live intra-uterine pregnancy (IUP) with another structure that appeared to be a second gestational sac. The patient subsequently had an endovaginal ultrasound in the radiology department 46 minutes later. The attending radiologist described one live IUP and a subchorionic hemorrhage. Comparison of the ED and radiology ultrasound showed that the second structure, identified as a subchorionic hemorrhage, had significantly decreased in size. Endovaginal ultrasound in the evaluation of possible ectopic pregnancy is a useful bedside tool in the ED. We discuss a pitfall that can occur with endocavitary ultrasound when a twin gestation is presumed.
[*West*JEM. 2008;9:115-117.]

INTRODUCTION

Emergency department (ED) bedside ultrasound is a fast and accessible tool for clinical evaluation of various patient symptoms. This technique allows a more rapid disposition and can aid rapport with patients and improve their education.[1] Pelvic ultrasound is one of the six major emergency ultrasound applications.[2] An endovaginal probe can reveal findings that help to diagnose a possible live intrauterine pregnancy (IUP), ectopic pregnancy, or abnormal IUP. As vaginal bleeding remains one of the 10 most common reasons for visits to the ED[3] and as bleeding occurs in 21% of pregnancies before the twentieth week,[4] bedside endovaginal ultrasound has become a mainstay of ED evaluation.

However, the acquisition of bedside ultrasound skills is not without pitfalls. Even experienced ED sonographers can misinterpret images, as bedside ultrasound is limited by patient physiology, the limited image quality of portable equipment, and time constraints. We describe a misinterpretation of an ED ultrasound in the common scenario of first trimester vaginal bleeding.

CASE REPORT

A 24-year-old $G_3P_1A_1$ Hispanic female presented to the ED at six and one-half weeks by dates with vaginal bleeding of one-day duration, requiring the use of one sanitary pad. She described mild, colicky, bilateral lower quadrant abdominal pain. She denied fever, chills, nausea, vomiting, diarrhea, dysuria, and hematuria. She had a spontaneous abortion one year earlier. The patient had no previous surgeries or medical problems. She was taking no medications and denied alcohol, tobacco, or illicit drugs.

On presentation to the ED, vital signs were: pulse of 86 beats per minute, respiratory rate 20 breaths per minute , blood pressure 129mmHg/62mmHg, temperature 37.5 degrees Celsius and O_2 saturation 99% on room air. Physical exam revealed a nontoxic female in no acute distress. Abdominal examination was non-tender and non-distended, with normal bowel sounds. The uterus was not palpable. There was no rebound tenderness or guarding. On pelvic exam, the cervical os was closed and scant blood was noted in the vaginal vault. No active bleeding was present. The patient had a positive Chadwick's sign. Bimanual exam revealed neither masses nor cervical motion tenderness. The remainder of the physical exam was unremarkable.

Laboratory results revealed: WBC of 9,200 cells/mL, hemoglobin of 13.9 g/dL, hematocrit of 41.2%, and 270,000 platelets/mL. Serum beta human chorionic gonadotropin was 25,845 mIU/mL (normal values for first trimester pregnancy at this institution: 5,000 to 200,000 mIU/mL). Clean-catch urinalysis demonstrated specific gravity of 1.010, pH of 7, no WBCs, 6 RBCs, small leukocyte esterase, large hemoglobin, few bacteria, and negative nitrite, glucose, and ketones. The patient's blood type was O, Rh positive.

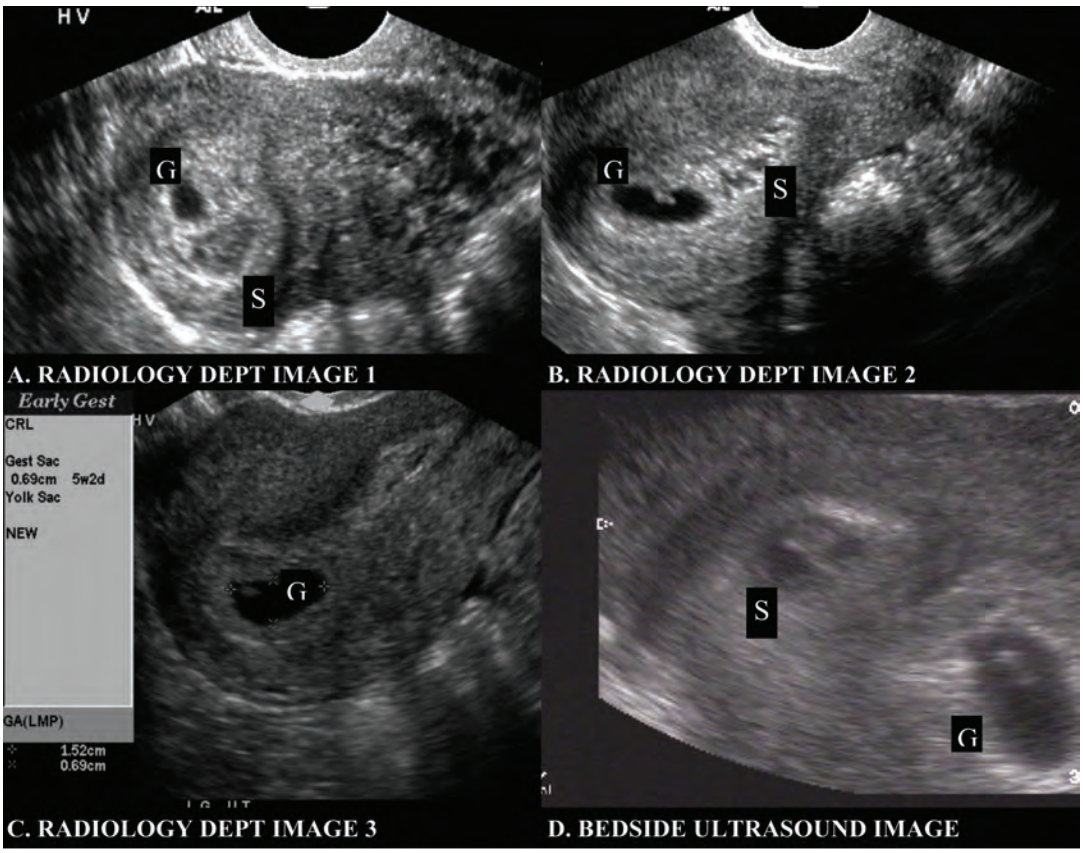

Figure 1. Endovaginal ultrasound with one live intrauterine pregnancy and a separate subchorionic hemorrhage resembling two gestational sacs. (S= subchorionic hemorrhage; G= gestational sac)

A bedside ultrasound was performed using a 7.5 MHz endovaginal probe (BK Hawk 2102 Ultrasound, Copenhagen, Denmark). Sagittal and coronal planes of the uterus and adnexa were obtained. There was no free fluid in the anterior or posterior cul-de-sacs. The adnexa were visualized and appeared normal, with no evidence of extra-uterine gestation. Two fluid-filled oval structures were noted in the patient's uterus (Figure 1D). One of the sacs (measuring approximately 1.5cm x 0.7cm) contained a fetal pole and fetal heart motion (labeled G on image). Power flow Doppler was not used because of questionable risk to the fetus[5]. The second sac (labeled S on image), measured approximately 1.2cm by 0.6cm. In it, a hyperechoic area similar in size and shape to a fetal pole was observed. Fetal heart motion was not visualized in this area. Scanning through sagittal and coronal planes did not uncover any physical connection between these two sacs. The patient tolerated the endovaginal probe without significant pain or complications. The ED bedside ultrasound interpretation was one live IUP with a possible second gestational sac. To better evaluate the sac without cardiac motion, the patient subsequently had an endovaginal ultrasound in the radiology department 46 minutes later (ATL 5500 SonoCT, Bothell, WA). These images are shown in Figure 1 as Radiology Dept Images 1-3 (Figure 1A,B,C). The attending radiologist described one live IUP and a subchorionic hemorrhage. Comparison of the ED and radiology ultrasound showed that the second structure, identified as a subchorionic hemorrhage, had significantly decreased in size. There was no noted increase in vaginal bleeding between ED and radiology scans.

The patient was discharged home on pelvic rest with obstetrical follow-up.

DISCUSSION

First trimester vaginal bleeding is a common chief complaint in the ED. It has been shown that EM residents can diagnose live intrauterine pregnancy with good sensitivity (91%) and specificity (99%) when fetal cardiac activity is present.[6] In this case, a live IUP was diagnosed. However, a separate finding was unable to be definitively explained. This confusion was despite considerable experience in bedside ultrasound, including endocavitary sonography, by the emergency physician. Therefore, the patient was sent for a pelvic ultrasound in the radiology department. This case illustrates the utility of pelvic ultrasound at the bedside and the importance of recognizing limitations when unsure of sonographic findings.

The emergency physician must rule in an IUP, thereby making the diagnosis of ectopic pregnancy highly unlikely. One in 4000

pregnancies in the general population may include simultaneous IUP and ectopic pregnancy, also known as a heterotopic pregnancy.[7] With ovarian hyperstimulation accompanying in-vitro fertilization, heterotopy occurs as often as 1 in 100 pregnancies.[8] This patient was noted to have an IUP and no signs of ectopic pregnancy, such as free fluid or adnexal mass.

In addition to an IUP, a pseudogestational sac must be included in the differential diagnosis. Stimulation from an ectopic pregnancy may cause fluid collection in the endometrium, and the physician may miss an important diagnosis of ectopic pregnancy due to the identification of a pseudogestational sac, which has only one hyperechoic ring. Identification of an IUP is confirmed when a gestational sac greater than five millimeters in diameter with a lucent center is surrounded by two concentric and hyperechoic rings, known as the double decidual sign. This sac must be located in the endometrium and contain a fetal pole or a yolk sac. When the gestational sac is larger than 10 millimeters in diameter, lack of cardiac activity in a definite fetal pole or absence of a fetal pole is a probable abnormal IUP. In this case, the ED bedside ultrasound does not show a double decidual sign. However, a hyperechoic structure inside the subchorionic hematoma appears similar to a fetal pole. The images from the radiology department do not display a distinct echogenicity inside the sac. This case adds another diagnosis to be considered when identifying a structure in the uterus. Subchorionic hemorrhage appears as a collection of black fluid on ultrasound.[9] Lack of a double decidual sign helps to differentiate it from a true IUP.

The subchorionic hemorrhage seen on the bedside ultrasound resembled a hypertrophied endometrium with scant fluid. These findings can mislead novice sonographers into diagnosing an intrauterine pregnancy.[10] The time from ED to radiology sonography allowed the size and shape of the hemorrhage to change, further resembling a subchorionic hemorrhage. Clinical increase in vaginal bleeding was not observed. This is not surprising given that the total volume of the subchorionic hemorrhage first identified in the ED was less than 1cc. The detection of a subchorionic hemorrhage on ultrasound increases the risk for miscarriage, stillbirth, placental abruption, and preterm labor.[11] It is controversial whether hematoma size influences the likelihood for carrying a pregnancy to term.[12,13,14,15]

In this case, an IUP and a subchorionic hemorrhage were both seen on a sonogram performed by the radiology department and initially interpreted as a possible twin gestation on the first ultrasound done by the emergency physician. Correct interpretation of bedside endovaginal ED ultrasound can be challenging and carries high risk. This case highlights the need for cautious interpretation and the importance of pursuing uncertain findings with confirmatory studies and the best available equipment. In other words, in a situation like this one where an IUP and another uncertain finding is present, a formal sonogram must be ordered in order to avoid the pitfall of missing a significant diagnosis when a bedside ultrasound may not be sufficient.

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Point: Diagnostic Radiation: Why Aren't We Stopping (Or at least Slowing Down)?

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Over the past 12 to 18 months I have heard from numerous sources that emergency physicians are using too much diagnostic radiation. The federal government (Biological Effects of Ionizing Radiation VII), the radiology community and several subspecialty groups are all calling for a re-evaluation of the use of medical radiation.[1,2] And yet, we seem to be doling out diagnostic tests that expose our patients to radiation on an hourly basis, with no sign of stopping.

The proposed increase in lifetime risk for developing a solid tumor or leukemia after the radiation of an abdominal/pelvic CT scan is 1:1,000.[3] Not much when you consider that the lifetime risk overall is 45%. However, there are reports of individuals receiving as much as 175 mSv (18 individual CT studies) over the course of therapy for one episode of urolithiasis.[3] It does not seem to dawn on us that our diagnostic CT is going to be followed by several others as the urologist or internist follows the passage of the stone.

Another study, published in the *Journal of Trauma* last year described the typical radiation exposure suffered by a trauma patient. Study patients, described as all trauma patients arriving by emergency medical services (EMS), received an average of five CT scans and 14 plain films during their hospital stay. The expected excess cancer mortality from this exposure was 190 per 100,000. Given 2.6 million trauma patients admitted each year, the public health ramifications are obvious. The authors concluded that unnecessary CT examinations should be avoided and dose-reduction protocols and shielding should become routine practice.[4] It has not been my experience that these recommendations have been broadly adopted.

One argument made by the academic community for so many advanced imaging studies in the setting of renal colic or routine trauma is that the CT allows us to visualize alternate or unexpected diagnoses. A recent collection of several studies detailing some 2600 patients' workup for flank pain consistent with urolithiasis and renal colic showed that there was a 12% rate of alternative findings. Most of these were adnexal masses. Really? As clinicians, can we not distinguish between an adnexal mass and the flank pain of kidney stones? As to the dreaded aneurysm/dissection of that major artery in the belly, there were three of these diagnosed in over 2600 scans (0.1%).[5] That 0.1% is the same as the risk of cancer we are causing by ordering so many studies.

While I realize that it is fuzzy math the truth is that with a focused history and physical we probably could do better, saving the radiation for the older patient, or those with co-morbidities. Doppler ultrasound is available as are Ultra-Low-Dose CT scanners.[6,7] These latter have been described since the turn of the millennium and expose the patient to no more radiation than a KUB (0.69 mSv) with a sensitivity and specificity EXACTLY the same as the typical CT scan. We don't push for a change in technology because it's difficult, expensive and time consuming.

The truth is it's easier to order a CT. We don't need to examine the patient and we don't have to take any risk. But that's not the art of medicine as we were taught, it's the art of lawsuit-aversion and it is irresponsible.

And we don't get any help. The radiologist won't perform a cone-down view or a limited study when you just need a little information because that puts *them* at risk. So even though we only need to see C6-T1, they insist on performing an entire cervical spine CT scan because they are afraid of being found liable for missed injuries in parts of the spine not even imaged. And how many patients with a classic presentation for acute appendicitis go to the OR without first receiving mandatory "radiation therapy"?

Ironically, there was a recent article from the United Kingdom where CT scan was recommended as the "newer and better" technology that should replace intravenous urogram.[8] For some reason, the medical community in Britain has decided to make the same mistake we Yanks did…leave behind a perfectly sound imaging technology and replace it with one that provides limited information about function in favor of relatively meaningless information about the size and composition of the offending stone.[8] The oath that I took in medical school stated that I "will neither give a deadly drug to

anybody who asks for it, nor will I make a suggestion to this effect." We prescribe the deadly drug of unnecessary radiation on a daily basis, so truly, why aren't we stopping?

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Counter-Point: Are We Really Ordering Too Many CT Scans?

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In a recent review published in the *New England Journal of Medicine*, DJ Brenner and EJ Hall, professors of Radiation Biology at Columbia University, analyzed the current trend to increased use of computed tomography (CT) scanning, the attendant radiation exposure, and the long-term potential for induction of fatal malignancies in the population.[1] This article was widely discussed in the popular press and on television news with headlines such as "Unnecessary CT scans exposing patients to excessive radiation: cancer cases could spike as a result," "Doctors may risk overuse of CT scans," and "The doctor says get a CT scan. Should you?"[2-6]

The original journal article and subsequent news coverage stated that one-third of CT scans are medically unnecessary, needlessly exposing millions of patients to high doses of radiation. However, the "evidence" on which this statement was based was insubstantial. In an ad hoc survey conducted during a panel discussion at a meeting of pediatric radiologists, a speaker mentioned that he felt 10% of CT scans were not medically necessary.[7] He then queried the audience, which responded that up to 30% were unnecessary. Aside from the fact that this was merely a casual inquiry, these are not the people making clinical decisions about patient care.

Emergency physicians (EPs) have, to some extent, been singled out with regard to this issue since we are responsible for ordering many CT studies. Dr. Brenner is quoted in *USA Today* as stating: "Virtually anyone who presents in the emergency room with pain in the belly or a chronic headache will automatically get a CT scan. Is that justified?" Dr. Fred Mettler, chief of Radiology at the New Mexico Veterans Administration Hospital, was quoted by MSNBC: "The pressure is greatest for ER doctors who 'are in a bind ... they have all these patients stacked up' and need to make quick decisions." Again, it was not clinicians who made these statements, and they do not reflect the practice of high-quality emergency care.

EPs were also cited for being unaware of the magnitude of the radiation dose associated with CT scanning. In one survey, 91% of EPs did not believe that CT scans were associated with an increased risk of fatal cancer.[8] Clearly this misperception should be remedied, but its impact on clinical care is less certain.

In actuality, there is abundant evidence demonstrating the considerable benefit of CT in managing emergency department (ED) patients, substantially outweighing its risks. In fact, in some circumstances (e.g., patients with acute headache) we may not perform CT scans frequently enough. Nonetheless, the issue of radiation exposure with CT scanning is an important one and needs to be considered in clinical practice.

Increasing CT Usage and Radiation Exposure

In their review, Drs. Brenner and Hall[1] describe the marked increased in the use of CT scanning, from three million scans in the U.S. in 1980, to 20 million in 1995, to over 60 million in 2005. Although the risk for an individual is small, in a few decades up to 2% of all cancers may be due to radiation exposure from CT scans, an increase from the current estimated rate of 0.4%. In addition, CT use is projected to increase even more in the future due to screening of asymptomatic patients, such as chest CT for lung cancer in smokers, virtual colonoscopy and cardiac/coronary artery scans. However, before any screening program is instituted, its benefit over the risks of radiation must clearly be established.

CT, particularly multidetector helical CT, is exceedingly "user-friendly" for the clinician, the patient and the radiologist. It is readily available, very fast, produces high-quality images, and is capable of detecting a wide array of illnesses. The typical CT radiation dose is 10 to 20 millisieverts (mSv), which is associated with a lifetime risk of fatal cancer of approximately one per 2,000 CT scans. The radiation exposure from three or four CT scans is roughly equivalent to that experienced by atomic bomb survivors in Japan who were located one to two miles from "ground zero."[9] Although this startling figure has been questioned due to differences in the type and duration of radiation exposure,[10] it could well serve as a powerful argument to convince a patient to forgo a CT scan that the physician felt was truly unnecessary. Nonetheless, in EM practice, CT scans clearly provide potentially life-saving information for more than one in 2,000 patients.[11]

The radiation dose and risk of malignancy vary

substantially with age of the patient – from one in 10,000 for patients over age 40 years, to roughly one in 500 for a neonate.[1,12] Infants have a more than ten-fold greater risk than middle-aged adults due to their increased radiation sensitivity, smaller size, and longer life span, which provides a greater length of time for induction of malignancy. However, this calculation presumes that the same CT radiation settings are used in children as in adults. One major method to reduce radiation exposure in children is to reduce the radiation settings of the scanner based on the size of the patient. The earlier work of Dr. Brenner was instrumental in bringing attention to this issue.[12-14]

Drs. Brenner and Hall propose three ways to reduce CT radiation exposure in the population. The first is to reduce the radiation dose delivered by the scanner, a strategy especially important in children. CT dose reduction leads to increased image noise, but numerous studies have shown that image quality remains acceptable. Also, newer scanners employ automatic radiation exposure control. EPs should be cognizant of the equipment used in their institution and advocate that dose-reduction methods be used by radiologists, radiology technicians and CT scanner manufacturers.

The second measure to reduce radiation exposure is to use alternative imaging options whenever possible. These include ultrasonography and MRI, although the circumstances in which this approach would be useful are limited. In addition, the availability of MRI for ED patients is not widespread.

Their third suggestion is to reduce the number of CT scans ordered, limiting them to medically necessary situations, which the authors estimate could eliminate up to one-third of CT scans. However, their statement is unwarranted for most ED cases, and, if applied indiscriminately, the omission of CT could potentially be injurious to patients by causing diagnostic delay or misdiagnosis.

CT in Emergency Medicine

The benefits of CT for a multiplicity of medical conditions, including traumatic injuries, neurological emergencies, abdominal pain, and certain thoracic disorders (pulmonary embolism and aortic dissection), are undisputed. At the same time, CT should not be used in place of a carefully performed history and physical examination. Indiscriminate use of CT, or any other diagnostic test, is a recipe for disaster. Such an approach would lead to over-ordering of CT scans, ordering the wrong CT protocol, erroneous use of CT results, and delaying necessary treatment in patients with serious conditions (e.g. ischemic bowel obstruction) while awaiting CT.

CT is now a staple in the diagnosis of appendicitis. It reduces the rate of negative laparotomy from an historical 15-20% to 4%,[15] reduces delays to surgery, and reduces the rate of misdiagnosis. CT is particularly useful when the diagnosis is uncertain based on clinical examination, although it can also be beneficial in cases in which the diagnosis seems highly likely.

A recent prospective randomized trial of 152 patients with suspected appendicitis compared a strategy of mandatory CT in all patients to a selective approach based on clinical judgment.[16] Half the patients had appendicitis. The investigators found that the selective approach reduced CT scanning by one-third, but was associated with an increased rate of negative laparotomy (14% versus 2.6%), and an increased rate of perforation (18.4% versus 10.3%). The same investigators compared outcomes in three hospitals that had different rates of CT use in patients undergoing appendectomy.[17] The negative laparotomy rates varied significantly depending on the rates of CT utilization. CT utilization in the three hospitals was 87%, 66% and 13% and the negative laparotomy rate was, respectively, 2.5%, 17%, and 23%. A third study found that even among patients deemed by a surgeon to "definitely" have appendicitis, CT revealed that appendicitis was not present in a substantial number of cases (five of 18 cases, or 28%).[18]

When a less serious disorder is under consideration, alternative diagnostic strategies should be used. Renal colic is one example. It is not life-threatening and can be predicted with good clinical certainty, particularly when a patient has had prior documented episodes of renal colic. CT should be reserved for circumstances in which there is diagnostic uncertainty. CT is also useful when pain is refractory, and information about ureteral stone size and location would be therapeutically beneficial. An alternative diagnostic approach to a patient with high likelihood of renal colic would be ED discharge with instructions to the patient to strain his or her urine. Eventual retrieval of the stone confirms the diagnosis. A bedside renal ultrasound showing hydronephrosis might also be useful in supporting the clinical diagnosis of renal colic.

The cervical spine is an area where more widespread use of CT could lead to an unwarranted increase in radiation exposure. For high-risk patients (> 5% incidence of cervical spine injury), CT is a substantial advance. For low-risk patients who cannot be clinically cleared, conventional radiography is still used. However, the incidence of cervical spine injury in such patients is exceedingly low (0.2%).[19] Because many radiologists now prefer CT to radiography since it is more comprehensive and results in fewer missed injuries, there may be a tendency to recommend CT even though the patient is at very low risk of injury. Use of CT in the vast majority of these non-high-risk patients is not warranted.

For major trauma victims CT of the head, neck, abdomen, and, in many cases, chest is clearly beneficial despite its high radiation dose.[20] For minor trauma cases, CT is not needed when the clinician is confident there is no serious injury, and the patient can be observed for an appropriate period of time.

Finally, in patients with acute headache, data suggest that we may not be ordering CT scans frequently enough.

Subarachnoid hemorrhage (SAH) is the most consequential headache disorder because a small hemorrhage often precedes a major life-threatening bleed, which, when promptly diagnosed and treated, will prevent a subsequent major hemorrhage. Unfortunately, the initial "sentinel" headache is sometimes missed, with dire consequences. In a recent series of patients with SAH, 19% of those who were initially neurologically intact were missed during a preceding physician visit.[21] The most common reason by far the diagnosis was missed was that CT was not performed – in nearly 75% of missed cases. Misinterpretation of the CT or LP occurred in 15% of the missed cases, and failure to perform an LP when the CT was normal was responsible for fewer than 10%. A similar result was found in an earlier series.[22]

SUMMARY

CT is a tremendous advance in managing a wide array of medical and surgical diseases and, despite its high radiation dose, should be used in many ED patients with potentially serious disorders. CT should not, however, be used indiscriminately or in lieu of a complete history and physical examination.

Measures to reduce radiation exposure to patients from CT should be instituted in accordance with Dr. Brenner's article. First and foremost, CT scanner radiation dose should be reduced as much as possible. While this is especially important in children, it should also be the goal in adults. Alternative diagnostic strategies should be employed whenever possible, including ultrasonography, MRI or watchful waiting, although this is only an option in some circumstances. Finally, ordering medically unnecessary CT scans should be avoided. However, in practice this must be done with caution so as not to risk harming the patient due to misdiagnosis or delayed diagnosis of a potentially serious disorder.

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Images in Emergency Medicine: Traumatic Pneumocephalus

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A six-year-old female was evaluated at the Emergency Department after being struck by an automobile driving 15-20 mph through a pedestrian crosswalk. The initial assessment was significant for a blood pressure at 104/72 mm Hg, heart rate at 124 beats per minute, decreased breath sounds in the left chest with agonal respirations, bleeding from right ear and back of head, open eyes, and non-verbal communication. The patient was intubated and a bedside chest x-ray demonstrated a left lung hemopneumothorax that was immediately drained via chest tube.

A subsequent CT scan of the head exposed skull fractures and massive pneumocephalus with multiple air bubbles surrounding the brain and within the lateral ventricles. Pneumocephalus has been reported in traumatic and non-traumatic instances; however, trauma accounts for most cases (74%), and its incidence has been estimated between 0.5 and 1.0% of all head injuries.[1] The most common traumatic causes include facial bone or cranial fracture with an injury that extends to the dura mater.[2] The prognosis is largely related to the type of injury and the number of air bubbles or pockets, but it has been shown that a pneumocephalus with multiple air bubbles is prognostically unfavorable, regardless of the mechanism of injury.[3]

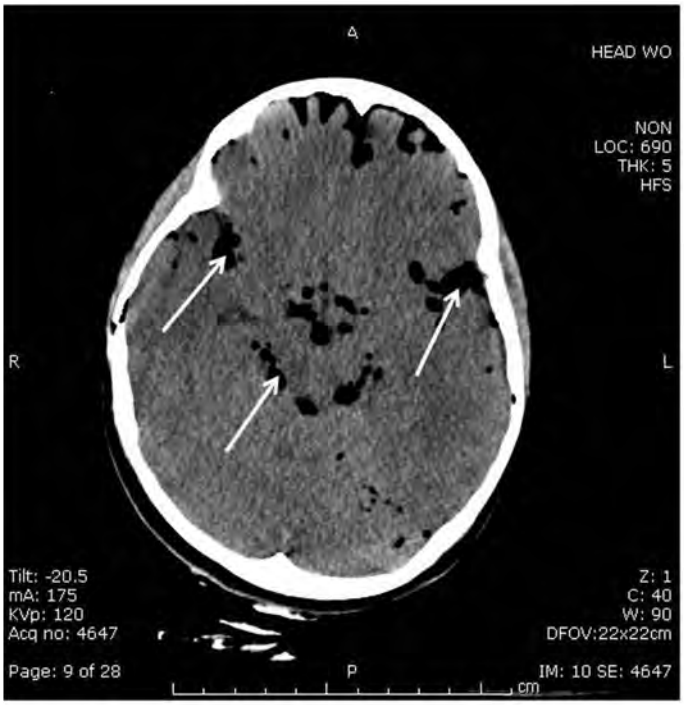

Figure 1. CT image demonstrating severe and widespread air bubbles in the subarachnoid and subdural spaces, as noted by the dark areas and highlighted by the arrows.

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Images in Emergency Medicine: Pediatric Spinal Cord Injury without Radiographic Abnormality

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An 11-month-old Hispanic male was brought to the emergency department (ED) by paramedics after being struck in his stroller by a full-size pickup truck. Witnesses told medics on the scene that the stroller was pushed 20 feet but did not tip over, and the patient was not ejected. The patient's mother was also struck and sustained an ankle fracture. At the scene the patient was alert with stable vital signs and no visible injuries. The stroller was noted by the paramedics to be extensively damaged. On arrival in the ED the patient's vital signs were stable and exam revealed no signs of external trauma or neurologic deficits. CT of the head and neck was normal, but the patient was admitted for observation due to the presumed serious mechanism of injury and uncertain history. Six hours after admission the patient was unable to move his lower extremities, and lower extremity sensation was significantly decreased. MRI revealed significant spinal cord contusion and hemorrhage from C7 to T1, as well as ventral subdural hemorrhage from C2 through C7, and evidence of interspinous ligament rupture between C5 and C6. Neurosurgery treated the injuries non-operatively and the patient remained paraplegic.

Due to the inherent flexibility of the pediatric cervical spine and supporting ligaments, blunt trauma may cause significant spinal cord injury without visible fracture or dislocation. Spinal cord injury without radiographic abnormality (SCIWORA) is defined as any objective signs of myelopathy as a result of trauma in the presence of normal radiographs.[1] This relatively rare but devastating pattern of injury was originally described in children by Pang and colleagues in 1982. Children with SCIWORA experience delayed onset of neurologic deficits, sometimes up to four days after the initial injury, and generally have poor outcomes. Recently the NEXUS study group prospectively identified 27 adult patients with SCIWORA (0.08% of the total 34,069 patients enrolled), but none of the over 3,000 children enrolled had SCIWORA.[2]

Despite its low incidence, SCIWORA remains a concern in any pediatric blunt trauma patient with delayed onset of neurologic signs or symptoms, and urgent MRI is indicated. This case also illustrates the importance of a thorough and accurate history from medics and witnesses in cases of pediatric trauma, where the patient (and often the parents) may provide incomplete or inaccurate information.

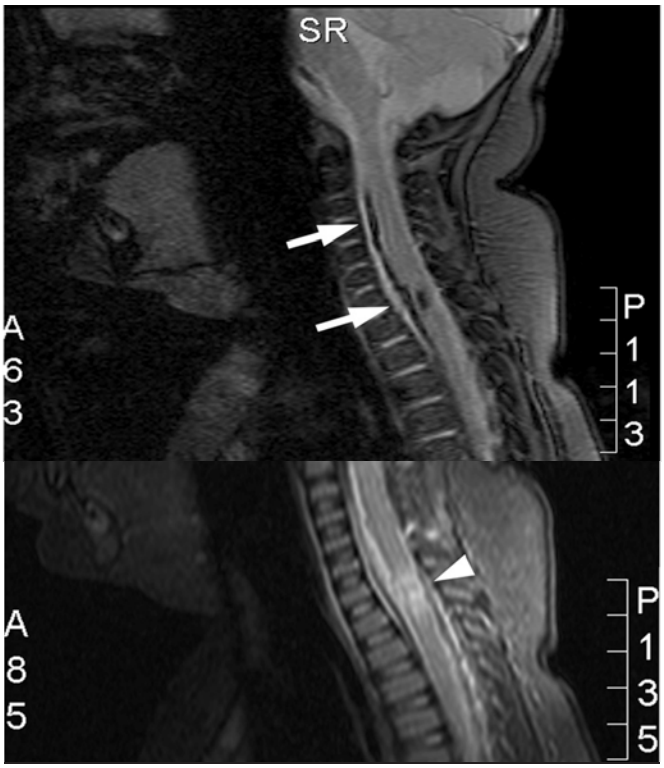

Figure 1. Upper plate, T1-weighted image showing anterior subdural hemorrhage from C2 to C7 (arrows). Lower plate, T2-weighted image showing extensive cord hemorrhage between C7 and T1.

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Images in Emergency Medicine: Left Atrial Myxoma

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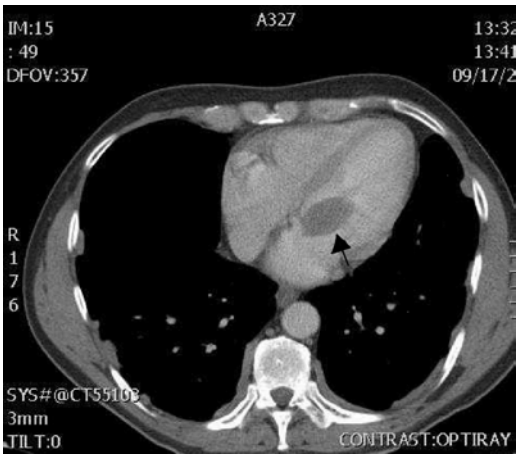

Figure 1.

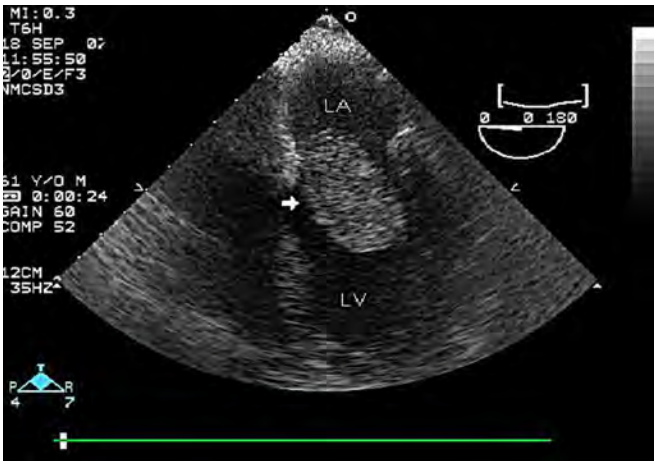

Figure 2.

A 64-year-old white male presented to the Emergency Department with complaints of 24 hours of right lower quadrant abdominal pain. The patient was without past medical or surgical history. Review of systems was remarkable for nausea without vomiting and absence of urinary symptoms or change in bowel habits. Physical examination was significant for a blood pressure of 136/76 mm Hg, a heart rate of 95 beats per minute, respiratory rate of 16 breaths per minute, and temperature of 98.7 degrees Fahrenheit. Pulse oximetry was recorded at 100% on room air. Patient was an alert, well-appearing adult male with exquisite tenderness to palpation over the right lower quadrant without guarding, rebound, or rigidity. The rest of the physical examination and the laboratory evaluation were without significant findings or abnormalities. A CT scan of the abdomen and pelvis (Figure 1) with P.O. and I.V. contrast is shown demonstrating an intra-cardiac soft tissue mass (closed dark arrow). No intra-abdominal/pelvic radiographic findings to explain the patient's abdominal pain were found. Subsequent bedside echocardiography demonstrated a 1.5cm x 2.5 cm left-sided atrial myxoma that extended into left ventricle during systole (Figure 2, white arrow). Atrial myxomas are rare, benign, intra-cardiac tumors with a wide spectrum of symptoms related to location, embolization, and propensity to obstruct blood flow through the heart.[1,2] These are often difficult to diagnose. Most commonly, left-sided atrial myxomas present with dyspnea on exertion, dizziness, syncope, neurological signs and symptoms as a result of systemic embolization.[1,2] Right-sided atrial myxomas present with easy fatigability, peripheral edema and ascites, or pulmonary embolism.[3,4] Additionally, pain resulting from embolic infarction of solid and hollow organs has been reported in the literature.[5,6]

Subsequent to resection of the myxoma, the patient had complete resolution of the abdominal pain. No cause was ever identified.

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Images in Emergency Medicine: Pacemaker Extrusion Causing Chest Pain

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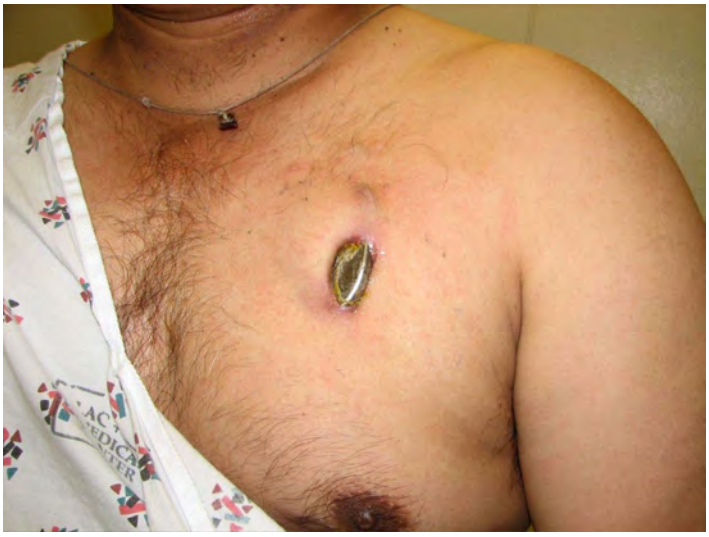

Figure.

A 55-year-old Hispanic male had a pacemaker placed in Mexico approximately one year prior to presenting to the Emergency Department. He noticed minor discomfort in his left chest one month earlier but did not see a physician. The discomfort steadily increased and he saw a small piece of metal poking through the skin. He assumed it was a staple or something minor related to the surgery; however, it gradually increased in size over the next few weeks until he realized it was the pacemaker itself eroding though his chest wall. A cardiology consultation was called, and an EKG showed that the pacemaker was still functioning normally. He was admitted with a plan for operative repair.

Pacemaker erosion or extrusion has been reported in 0.9% of patients receiving the device.[1] The two main causes are infection and pressure necrosis.[1,2,3] Infection has been shown to be reduced by antibiotic treatment during the peri-placement period, and pressure necrosis appears to be influenced largely by the size of the device, complexity of the connections and technical skill with which the pocket is created.[1,2] After extrusion, the pacemaker should be considered contaminated and removed.

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Images in Emergency Medicine: Dermatomyositis

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A 41-year-old Guatemalan woman with a history of remote uterine cancer presented to an urban community Emergency Department complaining of a pruritic, initially painful rash on her chest, eyelids, thighs, and elbows for three weeks. She also stated she had weakness in her thighs and shoulders with difficulty standing. She denied any new exposures (ex. foods, clothes, soaps, fragrances, medicines, or illicit drugs), recent travel, bites, or recent trauma. She denied fever, night sweats, weight loss, recent illnesses, or sick contacts. Her vital signs were normal and stable. On exam she was found to have circumferential, violaceous patches on her eyelids and proximal chest, and erythematous patches on the extensor surfaces of her elbows and in a malar distribution on her face. She was noted to have proximal thigh weakness and was unable to get up from a chair without assistance. The remainder of her exam, including sensation, cranial nerves, and distal motor function, was otherwise unremarkable. Laboratory studies were remarkable for an AST of 336, ALT of 158, LDH of 1948, CK of 9031, and ANA +. Her CBC and remaining metabolic panel were within normal limits. She was presumptively diagnosed with dermatomyositis and referred to general medicine and rheumatology clinics where she was started on a course of Prednisone with marked resolution of her symptoms. She recovered without sequelae.

Dermatomyositis (DM) is part of spectrum of idiopathic inflammatory myopathies. It has a prevalence rate estimated at approximately one per 100,000 in the general population with a 2:1 female predominance, and peak incidence between the ages of 40 and 50. Diagnosis is largely clinical and includes symmetric, proximal muscle weakness, characteristic rash, and elevated muscle enzymes. Common skin findings include Gottron's sign (symmetric, roughened, erythematous skin changes over the extensor surfaces of the metacarpophalangeal and interphalangeal joints, elbows, or knees), heliotrope rash (a violaceous eruption on the upper eyelids sometimes accompanied by edema), and shawl sign (a diffuse flat erythema in a shawl-like distribution over the chest and shoulder, or a V-shaped pattern over the anterior neck and chest).

Both measurement of serum muscle enzyme levels (CK, LDH, aldolase, AST, ALT) and testing for the presence of

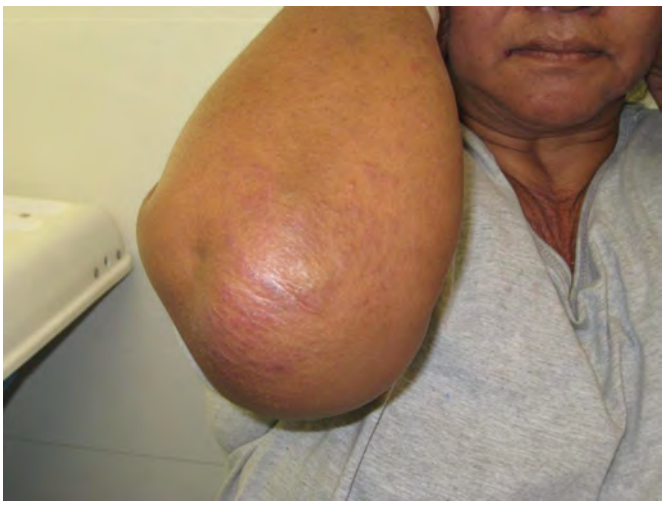

Figure 1. Gottron's Sign

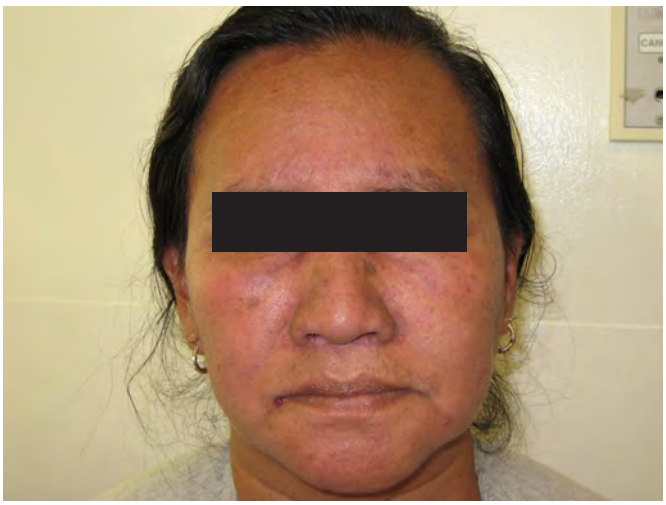

Figure 2. Hiliotrope Rash and Erythroderm

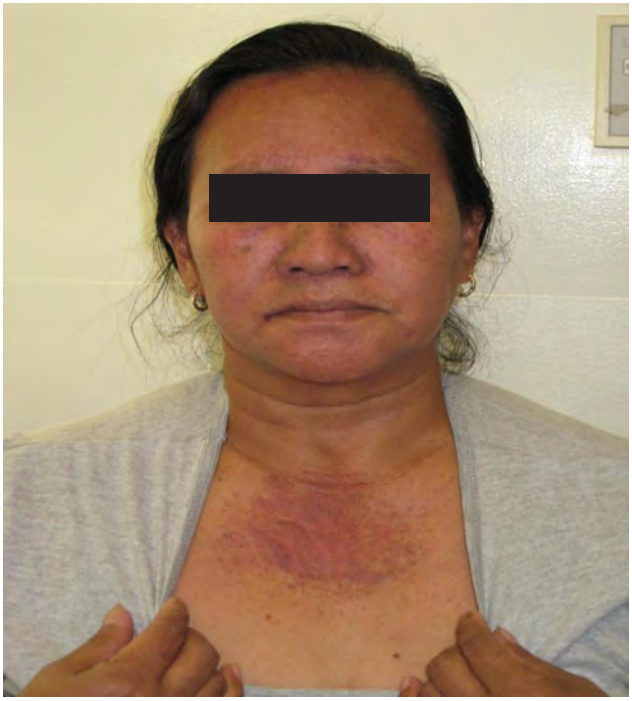

Figure 3. Shawl sign

myositis-specific autoantibodies play important diagnostic and prognostic roles in the assessment of patients with suspected DM. Other diagnostic and prognostic modalities include electromyography, MRI, and tissue biopsy. Prognosis is related to the severity of symptoms. Significant associated complications are not uncommon and can include interstitial lung disease and malignancy, and to a lesser extent esophageal disease and myocarditis. Therapy should be guided primarily by the degree of motor involvement and consists primarily of glucocorticoids and supportive therapy.

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Erratum

Author Jarrod Larson was missing from the list of authors on page 1, Volume IX, Issue 1 of the *Western Journal of Emergency Medicine*. The correct list of authors for "Accuracy of ED Bedside Ultrasound for Identification of Gallstones: Retrospective Analysis of 575 Studies" is as follows:

William Scruggs, MD
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Alexander Zlidenny, MD
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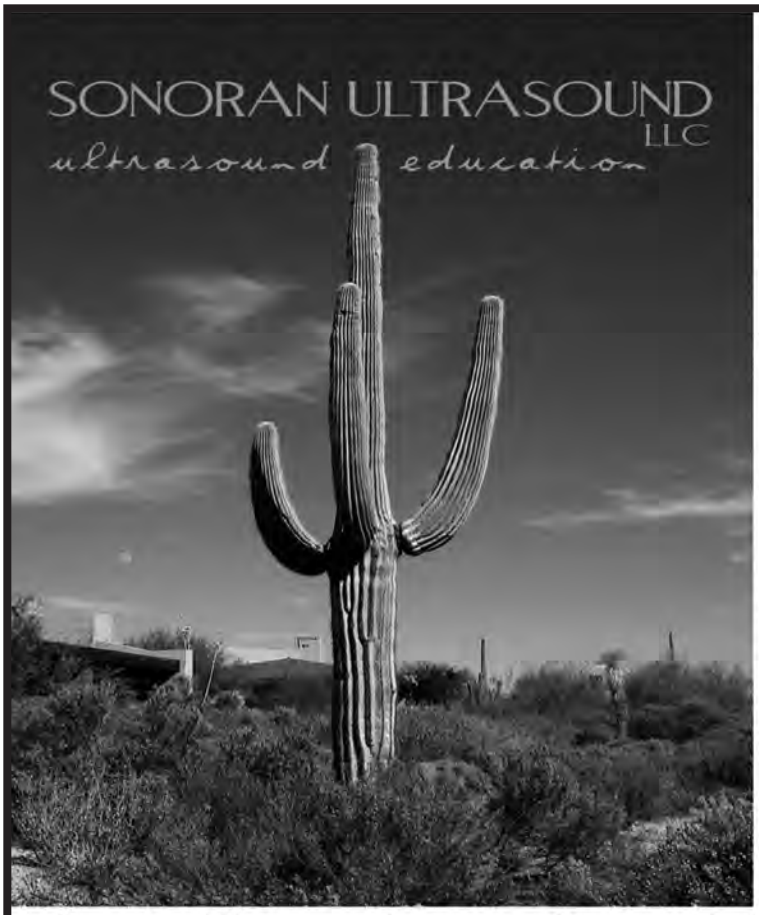

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