RESEARCH

MRI features of myelin oligodendrocyte glycoprotein antibody disease: a descriptive study—how it differs from neuromyelitis optica spectrum disorders and multiple sclerosis

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Abstract

Background Myelin oligodendrocyte glycoprotein antibody disease (MOGAD) is a novel inflammatory demyelinating disease of the central nervous system. This study aims to characterize the MRI features of MOGAD and contrast our results with the findings previously described in the literature and its close differential diagnoses.

Results Most of the abnormal findings are in the brainstem followed by supratentorial deep/subcortical white matter and optic nerves. Brain lesions in MOGAD tend to show a pattern that is different from multiple sclerosis (MS) and neuromyelitis optica spectrum disorders (NMOSD). Orbital MRIs show features of bilateral longitudinally extensive optic neuritis predominantly involving the anterior segments. The spinal cord is the least affected and mostly shows longitudinally extensive lesions in the dorsal spine.

Conclusions We could identify numerous characteristic radiological features that could help distinguish MOGAD from NMOSD and MS. We hope this study helps clinicians systematically evaluate and manage patients with clinical features of neuroinflammatory diseases.

Keywords MOG antibody disease, Neuromyelitis optica, Multiple sclerosis, Demyelination, MRI

Background

Myelin oligodendrocyte glycoprotein antibody disease (MOGAD) is a novel inflammatory demyelinating disease of the central nervous system. Myelin oligodendrocyte glycoprotein (MOG) is a cellular adhesive molecule located on the myelin surface and controls the stability of oligodendrocyte microtubules and regulates complement cascade [1]. Over the past few years, MOG antibodies have been researched, with some early experimental investigations speculating on their involvement in neuroinflammatory diseases. MOG antibodies were also found in the cerebrospinal fluid and sera of individuals who were then diagnosed with neuromyelitis optica spectrum disorders (NMOSD) and multiple sclerosis (MS) which supported this notion [2]. But more recently, a subset of patients with antibodies to MOG on cell-based assay who exhibit clinical phenotype and MRI features distinct from NMOSD and MS have been identified and classified [3].

It was found that almost 40% of patients with NMOSD-like presentations test negative for aquaporin-4 antibody (AQP4-IgG), even though the majority of NMOSD cases (a close differential diagnosis of MOGAD) are linked to aquaporin-4 antibodies. Those patients were found to be positive for MOG antibodies. It is now clear that the histopathology of inflammatory CNS lesions in MOG antibody-positive and AQP4 antibody-positive

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patients differ. The myelin oligodendrocyte glycoprotein, a membrane protein expressed on the outermost surface of myelin sheaths and the cell surfaces of oligodendrocytes, is damaged in MOGAD, whereas NMOSD is linked to antibodies that target aquaporin-4 (AQP4), the most prevalent water channel in the central nervous system and one that is particularly expressed in astrocytic processes at the blood–brain barrier [4–6]. Therefore, MOGAD is now understood to be a unique condition with specific management and therapeutic needs.

Methods

Brain, spine, and/or orbital MRI of MOGAD patients, stored in the Picture Archiving and Communication System (PACS) of the Department of Radiology at St. John's Medical College Hospital from November 2020 to November 2022, were evaluated in this study. The approval of the institutional ethics committee (IEC Study Reference No. 349/2020) of St. John's Medical College was obtained prior to the study. Patients who tested positive for MOG antibody IgG by cell-based indirect immunofluorescence assay who underwent MRI brain, spine, and/or orbits at St. John's Medical College Hospital, Bangalore, during the period of study were included in the study. Patients for whom MRI is contraindicated such as patients with cardiac pacemakers, cochlear implants, and metallic implants as well as patients who are known cases of other diseases of the central nervous system unrelated to MOGAD but are likely to have MRI brain and spine abnormalities were excluded from the study.

The MR scans were performed with either 3 or 1.5 T MRI machines (GE Healthcare, USA) at St. John's Medical College Hospital. For MRI brain, axial fast spin-echo T2-weighted images, axial/ sagittal fast spin-echo FLAIR, sagittal T1 weighted images, axial diffusion-weighted images (DWI) with ADC map, axial susceptibility-weighted images, and T1 post-contrast images (if available) were analysed. For orbits, axial and coronal T2-weighted and T1 fat-saturated post-contrast images were taken in a small field of view. Sagittal and axial T2-weighted images and T1 pre- and post-contrast images (if available) were obtained for the spine. MR contrast study is not warranted for every patient as contrast studies are done only if they are requested by the referring physician.

The lesions in the brain were described based on their signal intensity, site, and pattern of enhancement. Lesions showing post-contrast enhancement or diffusion restriction were considered acute lesions. Spinal lesions were described according to their location, length, cord expansion, and enhancement pattern. Longitudinally extensive transverse myelitis was defined as T2 hyperintense or enhancing spinal cord lesions extending three or more

segments of vertebrae [7]. Lesions in the optic nerve were described based on their site, post-contrast enhancement pattern, length of the segment that is involved, and signal abnormality. A T2 hyperintense lesion or an enhanced segment of length 17.6 mm or more was described as long-segment optic neuritis [8].

Results

A total of 124 MRIs (76 brain, 32 spinal cord, and 16 orbital MRIs) were evaluated in this study. Patient details (clinical and demographic) are briefed in Table 1. The proportion of males to females in the study was 16 as opposed to 14, with a ratio of 1.14:1. The mean age of the cohort was 33.3 years. The mean age of symptom onset was 26.1 years. The most frequent initial presentation was encephalopathy (predominantly in paediatric patients and young adults of less than 25 years of age) (Fig. 1), reported in 12 patients (40% of all cases) followed by optic neuritis, experienced by nine patients (30% of all cases), and transverse myelitis, seen in seven patients (23.3% of all cases). Encephalopathy was also the most common presentation on relapse, which was followed by optic neuritis and transverse myelitis.
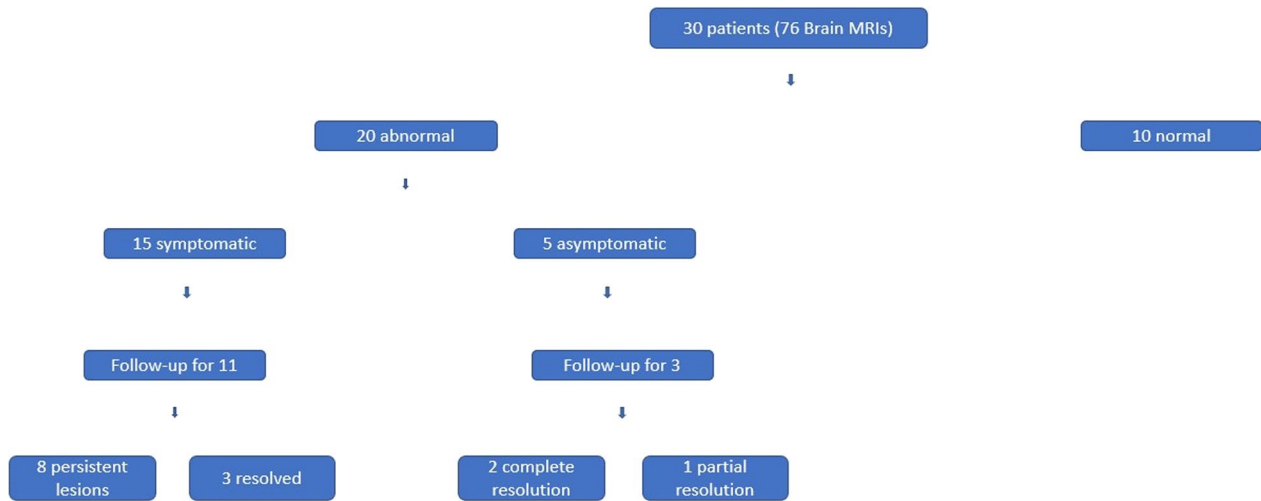
MRI brain

In our study, 76 brain MRIs were evaluated (Fig. 2). Patients were divided into groups depending on whether they had consistently normal brain MR scans throughout the duration of their illness and whether they ever had abnormalities in their MR brain. According to the contribution of the brain lesions to their clinical symptomatology, those patients with abnormal brain MRIs were then split into symptomatic and asymptomatic groups. Ten of the 30 MOGAD patients who obtained multiple

Table 1 Epidemiological and clinical parameters of the MOGAD patients

Parameters	MOGAD patients [*n* = 30 (%)]
Sex	
Male	16 (53%)
Female	14 (47%)
Age	
Mean	33.3 years
Range	5–65 years
Mean age at the onset of disease	26.1 years
Initial presentation	
Encephalopathy	12 (40%)
Optic neuritis	9 (30%)
Transverse myelitis	7 (23%)

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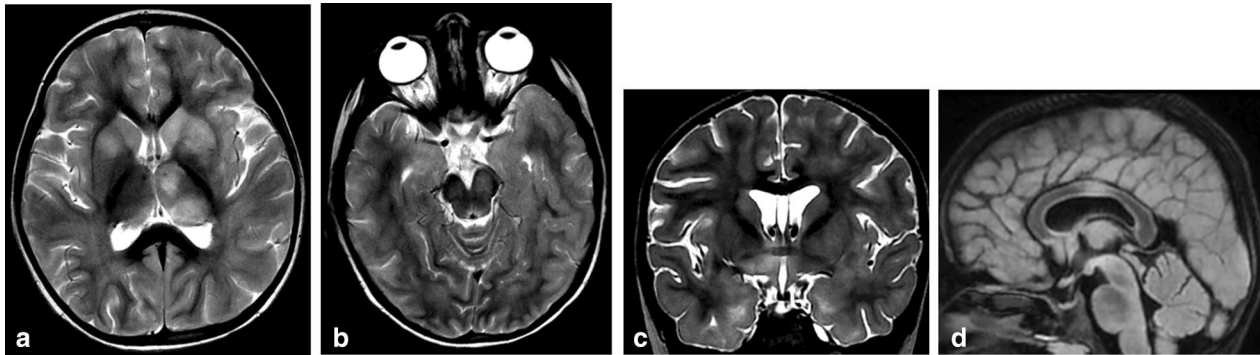
Fig. 1 The case illustrates paediatric MOGAD-related encephalitis in a 6-year-old girl who presented with fever and features of raised intracranial pressure. The brain lesions show typical characteristics of MOGAD—bilateral, asymmetrical, T2 hyperintense, and poorly demarcated with a fluffy appearance. **a** Axial T2-weighted MR image shows bilaterally enlarged thalami and basal ganglia (more on the left side) with hyperintense signal. **b** Axial T2-weighted MR image shows hyperintense signal in the left hemi-midbrain. **c** Coronal T2-weighted MR image shows bilateral hyperintense areas with surrounding oedema in the subcortical and deep white matter of fronto-parietal lobes, basal ganglia, thalami, and hypothalamic region. **d** Sagittal T2 FLAIR MR image shows hyperintense signal in the body of corpus callosum

Fig. 2 Flowchart of MOGAD patients who underwent MRI brain

brain MRIs throughout the course of their disease were normal. Twenty MOGAD patients (67%) had MRI brain abnormalities, of which 5 were asymptomatic and 15 were symptomatic.

Brain lesions in the asymptomatic group were dispersed, punctate, few in number, and non-enhancing in four patients. One patient showed localized leptomeningeal enhancement. Three out of those 5 asymptomatic patients obtained follow-up MRI scans, of which two patients had complete resolution of all lesions, and 1 showed partial resolution. Elevan of the 15 MOGAD patients who were symptomatic as well as exhibited abnormal brain MRIs obtained follow-up MRI scans. On the final MRI taken during remission, 8 exhibited residual lesions with varying degrees of recovery.
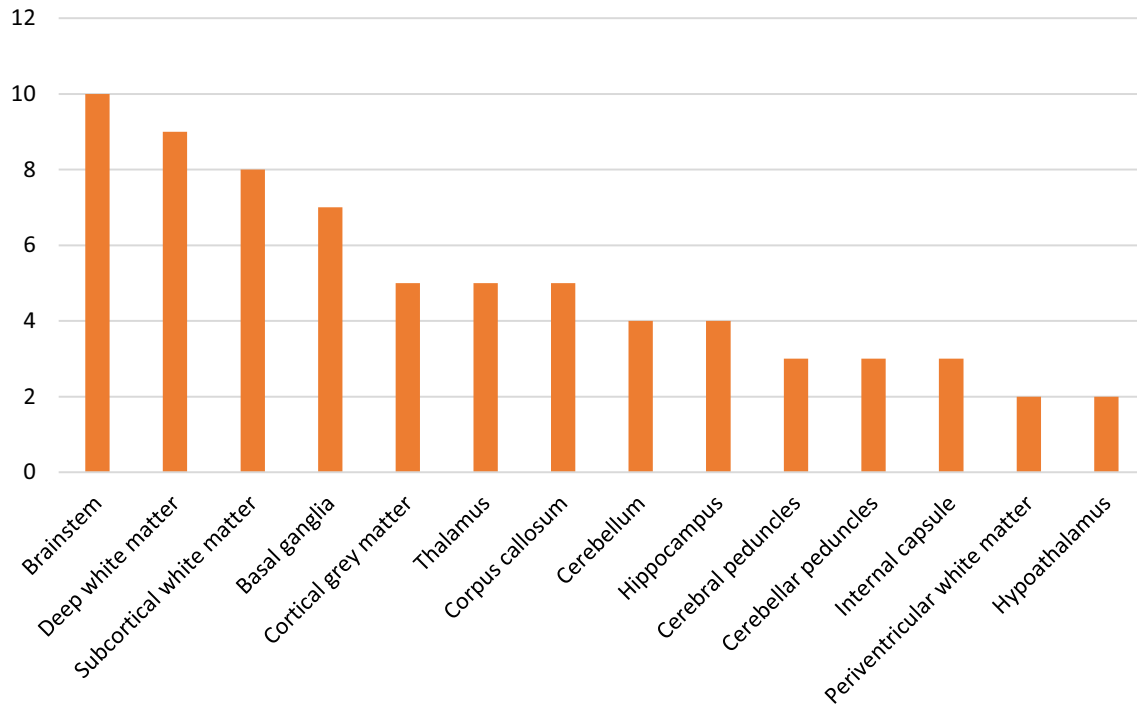
Most of the brain lesions in MOGAD patients showed few characteristic features. They were bilateral, asymmetrical, T2 hyperintense, and poorly demarcated with a fluffy or cloud-like appearance. Bilateral, large, brainstem, and deep grey nuclei lesions with involvement of cerebellar peduncles were more common in children.

Overall, 10 patients (50%) showed brainstem lesions out of which pontine lesions were found in six patients (30%), and midbrain and medullary lesions in four patients each (20%). However, the single most common lesion location overall (out of 20 patients with abnormal MRI brain) was supratentorial deep white matter involved in nine patients (45%), followed by juxta-cortical/subcortical white matter noted in eight patients (40%). Basal ganglia lesions were seen in seven patients

(35%), whereas lesions in cortical grey matter, corpus callosum, and thalamus were seen in five patients each (25%). Cerebellum (20%), hippocampus (20%), cerebral peduncles, cerebellar peduncles, and internal capsule (15% each) were other regions that were involved (Fig. 3). Only two patients (10%) had periventricular white matter and hypothalamic involvement (Fig. 4). Periventricular lesions seemed to extend from

surrounding cortical and subcortical lesions in one of those cases. McDonald's criteria for multiple sclerosis were not met by any of the MOGAD patients.

Only 4 out of 19 patients who received contrast scans had gadolinium enhancement of brain lesions, with patchy enhancement being the most common pattern (in three patients). Leptomeningeal enhancement was present in 3 individuals (Fig. 5a). Eight out of 14 patients


Fig. 3 Location of brain lesions in MOGAD
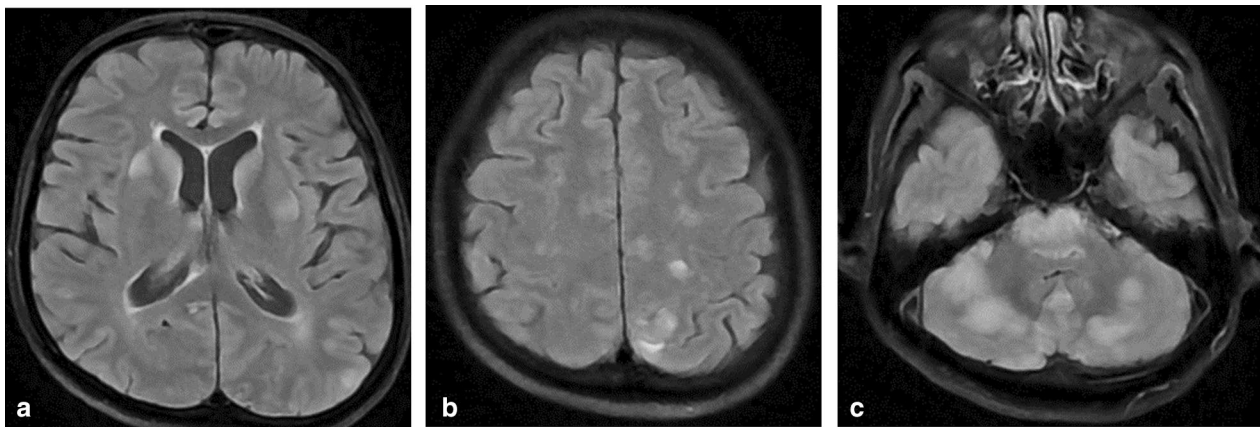
Fig. 4 The case illustrates MOGAD-related encephalitis in a 48-year-old male who presented with generalized tonic–clonic seizures. The brain lesions show typical characteristics of MOGAD—bilateral, asymmetrical, T2 hyperintense, and poorly demarcated with a fluffy appearance. **a** Axial T2 FLAIR MR image shows hyperintense signal in bilateral basal ganglia, cortex, and subcortical and deep white matter in parieto-temporal lobes. **b** Axial T2 FLAIR MR image shows hyperintense signal in bilateral cortex and subcortical and deep white matter in fronto-parietal lobes (more on left side). **c** Axial T2 FLAIR MR image shows hyperintense signal in pons and cerebellum
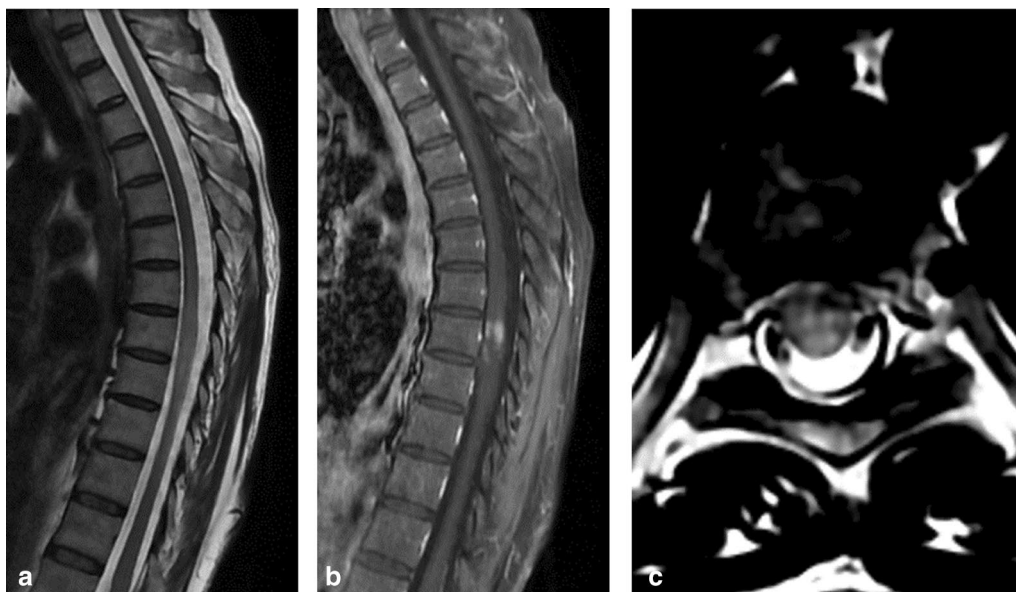
Fig. 5 The case illustrates long-segment dorsal cord involvement in MOGAD where a 47-year-old female presented with back pain and paresthesia in bilateral lower limbs. **a** Sagittal T2-weighted MR image of the dorsal spine shows cord signal hyperintensity in D7-D10 vertebral levels associated with cord expansion. **b** Sagittal T1 post-contrast MR image of the dorsal spine shows patchy contrast enhancement at D8 vertebral level. **c** Axial T2-weighted MR image at D8-D9 inter-vertebral level shows central T2 hyperintensity with a weak surrounding T2 hyperintense signal predominantly confined to cord grey matter suggestive of 'H sign'

(57%) who underwent follow-up scans were noted to have partial or complete resolution of their brain lesions.

MRI spinal cord

A spinal cord MRI was performed on 21 of the 30 patients in this cohort, either routinely or to evaluate potential relapses. This study examined 32 spinal MRIs in total (Fig. 6). Despite a clinical suspicion of myelitis in 3 participants, which was ruled out by negative MRI, 12 patients (57%) had normal spinal cord MRIs throughout the course of their illness. Nine patients (43%) had abnormalities in spinal cord MRIs, and 5 of those patients had repeated scans done either during remission or recurrence. Three of the patients had persistent lesions or new lesions on follow-up, while two out of five patients (40%) showed normalization of the T2 hyperintense MRI signal. One of those patients also displayed mild cord atrophy.

The locations of lesions in the spinal cord were as follows: One patient had cervical (12%), four had thoracic (44%), five had cervicothoracic (56%), and one had lumbar spine and conus involvement (12%) (Fig. 7).

Depending on the extent of the involvement, spinal cord lesions can be classified into two types: short-segment lesions affecting two or less than two vertebral segments and longitudinally extensive transverse myelitis (LETM) involving three or more vertebral segments [7]. In our cohort, seven patients (78%) had longitudinally extensive transverse myelitis (LETM), while six patients

(67%) had short-segment spinal cord lesions (SSTM). Three (33%) of the aforementioned patients had concurrent long and short-segment lesions.

In the axial plane, six patients (67%) had involvement of the central cord while holocord involvement was noted in two patients (22%). Three out of six patients with central cord involvement showed a peripheral hazy T2 hyperintense signal (Fig. 5). Peripheral cord involvement was seen in one patient (11%). Five patients (56%) had cord expansion.

Enhancement on post-contrast T1 images was noted in three patients (33%), in two of which, acute clinical relapse localizing to the spinal cord also occurred at the same time (66%). Despite the enhancement on the spinal MRI, the third patient was in clinical remission. The enhancement pattern was heterogeneous with blurred margins (cloud-like enhancement). A few areas of well-defined nodular enhancement were also found.

Orbital MRI

Sixteen orbital MRIs belonging to 12 patients were evaluated in this study. In the optic pathway, T2 hyperintense lesions were classified based on their location in these four segments: anterior optic nerve (intra-ocular and intra-orbital segments), posterior optic nerve (intra-canalicular and intracranial/pre-chiasmal segments), chiasma and optic tracts. Any lesion in the optic pathway extending for more than 17.6 mm was considered a
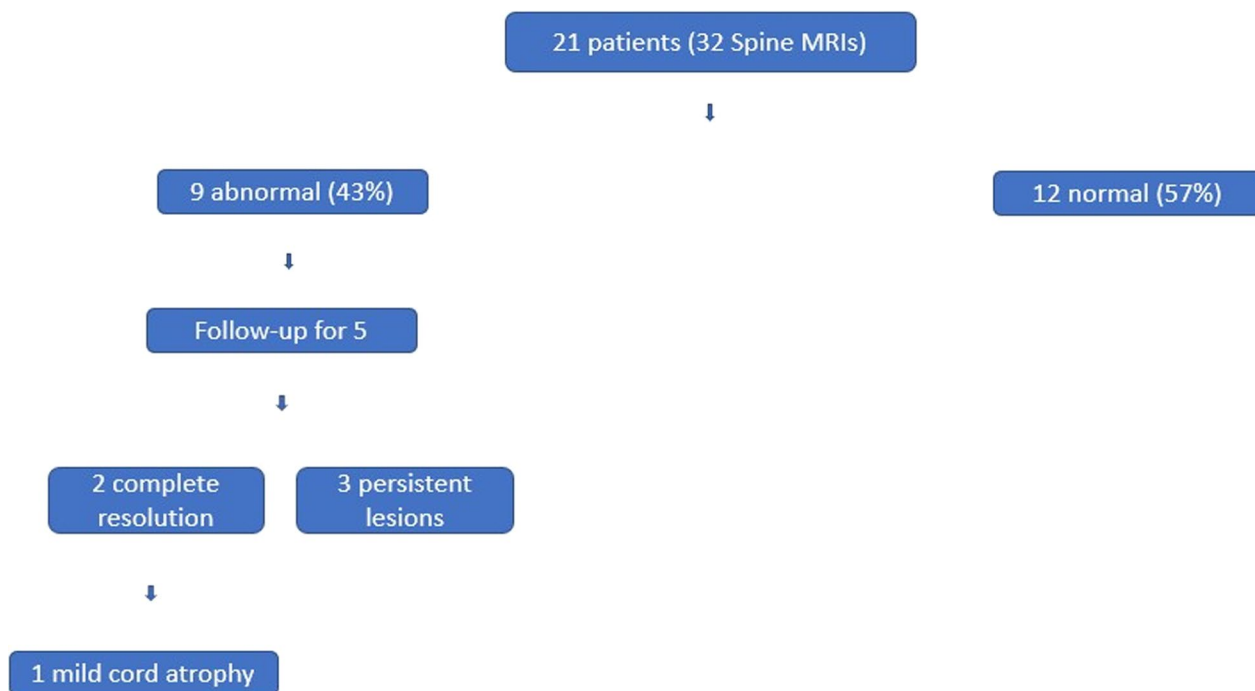
**Fig. 6** Flowchart of MOGAD patients who underwent MRI spine
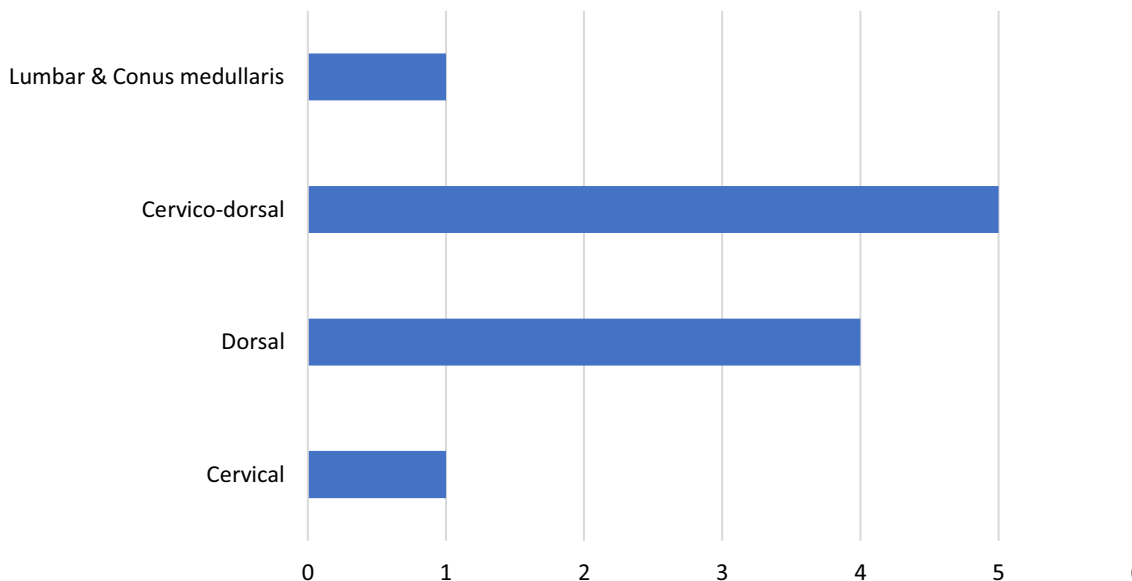
Fig. 7 Location of spinal cord lesions in MOGAD

longitudinally extensive lesion [8]. 5 out of eight patients (63%) with lesions along the optic pathway showed anterior optic nerve lesions out of which four were isolated lesions with no evidence of concurrent lesions in the brain or spine. One patient (12%) showed a posterior optic nerve lesion which was an isolated lesion. Two patients (25%) showed involvement of both the anterior and posterior optic nerve. No lesions were found involving optic chiasma or optic tracts (Fig. 8).

Gadolinium enhancement was evident in four out of these six patients (67%) who underwent contrast study. Out of those six patients, five patients had anterior optic

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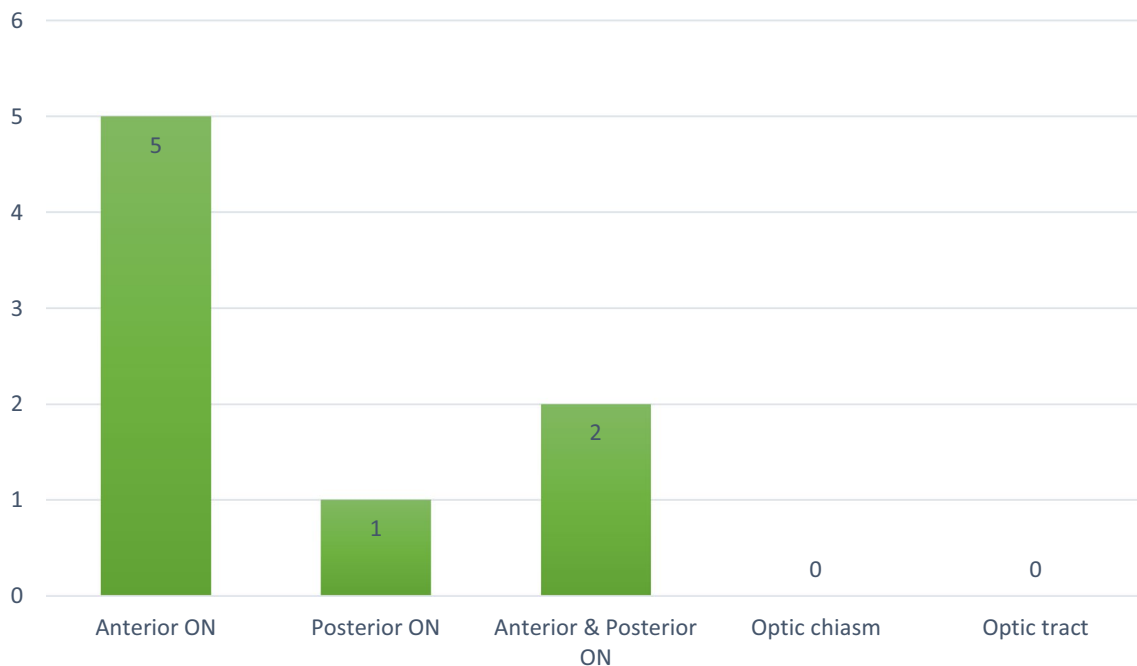
Fig. 8 Location of optic nerve lesions in MOGAD

nerve lesions and three of them showed post-contrast enhancement (60%), whereas one patient had posterior optic nerve lesions which showed post-contrast enhancement (100%). Peri-optic nerve sheath enhancement was found in three (50%) patients.

In our cohort, the average length of the optic nerve lesion was 19.1 mm. Five out of eight patients (63%) had longitudinally extensive lesions (Fig. 9), whereas the rest of the three (37%) showed short-segment optic neuritis.

Five patients underwent sequential MRI of the optic nerves, and two of them had lesions completely disappear. While two patients had persistent T2 hyperintense optic nerve lesions on follow-up, one patient showed partial resolution of T2 hyperintense lesions. Two patients showed mild optic nerve atrophy on follow-up. Six out of

eight patients (75%) experienced at least one instance of both optic nerve involvement during the course of their illness, while two (25%) had unilateral optic neuritis. None of the patients had alternating optic neuritis.

Discussion

In our study, MOGAD did not show significant male or female preponderance. The age of the patient showed a correlation with the clinical phenomenology of MOGAD, with paediatric patients and young adults frequently presenting with encephalopathy and the rest of the patients experiencing relatively more optico-spinal presentations. This was consistent with prior research, where younger patients frequently presented with encephalopathy while older patients were more prone to optic neuritis [9, 10].

Fig. 9 The case illustrates bilateral long-segment optic neuritis involving the anterior segments of optic nerves in a 37-year-old girl with MOGAD who presented with diplopia and blurring of vision. **a** Coronal T2 fat-saturated MR image of orbits shows thickening of optic nerves with hyperintense signal. **b** Sagittal T2 fat-saturated MR image of right orbit shows longitudinally extensive hyperintense signal in the anterior segment of the right optic nerve associated with optic nerve thickening. **c** Sagittal T2 fat-saturated MR image of the left orbit shows longitudinally extensive hyperintense signal in the anterior segment of the left optic nerve associated with optic nerve thickening

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An infectious prodrome was usually present before the beginning of the initial symptoms in MOGAD (47% of patients), although this is much less prevalent in NMOSD (10%) [11].

Overall, the location of lesions in our population of MOGAD patients was in line with previous studies with few differences. Unlike most of the previous studies where the majority of MRI findings were localized to the optic nerves [7, 12–14], brain parenchymal lesions were found to be more common in our cohort where the brainstem was the most frequently affected location, followed by supratentorial deep white matter. In the brainstem, most lesions were seen in the pons (60% of brainstem lesions). This is in keeping with the study of Cobo-Calvo et al. [13]. Surprisingly, basal ganglia lesions were found in 35% of patients with brain abnormalities. Other commonly affected locations include subcortical/ juxta-cortical white matter, cortical grey matter, thalamus, corpus callosum, cerebellum, hippocampus, cerebral and cerebellar peduncles, and internal capsule. Our MOGAD cohort's callosal lesions were different from Dawson's fingers of multiple sclerosis and the arch of bridge sign seen in NMOSD. The callosal lesions were focal, discrete, and had no specific periventricular orientation. Similar findings were also documented by Jurynczyk et al. [15].

The brain lesions were typically bilateral, asymmetric, T2 hyperintense, and poorly demarcated. Leptomeningeal enhancement was observed in three patients in one study [16] and one patient in another [7] as compared to four patients in our study. Amongst those four patients, three presented with encephalopathy and one was asymptomatic (Fig. 10a). A rare presentation of isolated unilateral limbic encephalitis was also noted in a patient with relapsing MOGAD in our study (Fig. 10b, c).

MOGAD was observed to be more frequently linked to an encephalopathic phenotype and seizure presentation than NMOSD and MS. Diffuse signal abnormalities in T2-weighted images in deep white matter, subcortical white matter, deep grey matter, cortical grey matter, or in a combination of these regions were identified to represent MRI findings of MOGAD encephalomyelitis or encephalitis. In a German study, like our study, supratentorial deep white matter was the most affected site, seen in almost half the patients with brain lesions [7]. Active lesions had restricted diffusion and/ or post-contrast enhancement. Partial or complete resolution of brain lesions on a subsequent MRI, which was found in 57% of patients who got follow-up MRI brain suggests MOGAD rather than NMOSD or MS where lesions are more likely to persist. This may point to an underlying MOGAD-specific healing mechanism.

Typical spinal MRI findings in our cohort of MOGAD patients include centrally located, T2 hyperintense, longitudinally extensive lesions involving both grey and white matter, with involvement of more than 50% of the axial section of the cord, and cord swelling. Our findings supported the observations of van Pelt et al. in a study conducted in the Netherlands, where the dorsal cord was the most commonly affected location [14]. Most other studies across the globe also showed a similar trend. However, cervical cord involvement was reported to be the most affected segment in MOGAD by Jarius et al., in contrast to our study, where isolated cervical cord lesions were the least common lesions. 72.4% of patients in the same study had longitudinally extensive lesions, at least once, while 41.3% of patients had short-segment myelitis [7]. Longitudinally extensive lesions were found to be more frequent (78%) than short-segment lesions in our
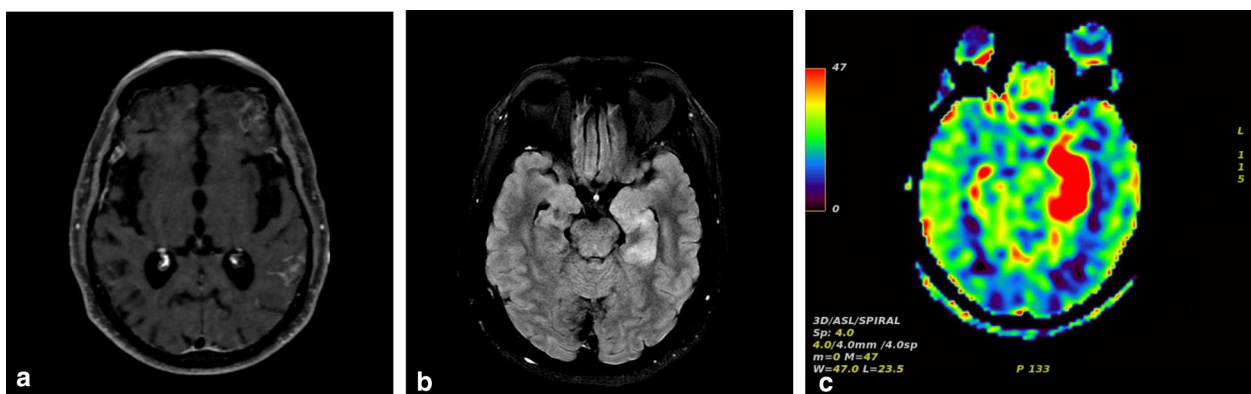
Fig. 10 Atypical presentations of MOGAD. **a** The case illustrates isolated aseptic meningitis in a 20-year-old man with MOG antibody disease. Axial post-contrast T1 weighted MR image shows contrast enhancement in the left frontal and parietal lobes. **b** The case illustrates isolated unilateral limbic encephalitis in a 54-year-old man with MOG antibody disease on relapse. Axial T2 FLAIR MR image shows hyperintensity in the left hippocampal region. **c** Axial ASL image of the aforementioned case of isolated unilateral limbic encephalitis in a 54-year-old man with MOG antibody disease shows increased cerebral blood flow in the left mesial temporal lobe

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cohort as well. Thirty-three per cent of our patients with spinal lesions showed concurrent long and short-segment lesions, whereas, in the aforementioned German study, 20 out of 28 patients showed two simultaneous spinal cord lesions. Two-thirds of our patients with abnormal MRI spine still had short-segment spinal cord involvement, even though only a small percentage of MOGAD patients presented with short-segment myelitis in various studies [17, 18]. This finding is probably connected to when the MR spine study was conducted in relation to when the symptoms first appeared [19]. In our dataset, one patient had involvement of conus medullaris—a lesion that was previously believed to be a salient feature of MOG myelitis [20].

Lesions in NMOSD, in contrast to those in MOGAD, are more frequently longitudinally extensive, commonly affect the cervical or dorsal spine, can be centrally or peripherally located, involve more than 50% of the cord, and typically demonstrate greater post-contrast enhancement and cord oedema. Contrary to MOGAD, NMOSD spinal lesions are more likely to have T2 signal intensity greater than CSF and T1 signal intensity similar to or lower than CSF, sometimes known as 'bright spotty lesions' and 'T1 dark spots' respectively. Bright spotty lesions, especially when paired with longitudinally extensive lesions and T1 dark spots, are a distinguishing imaging finding between NMOSD and MS [19, 21]. Demyelinating lesions in MS are predominantly short-segment peripherally located lesions involving the dorsolateral tract [22].

Involvement of the spinal cord grey matter, described as 'pseudo-dilatation of the central canal', was reported by Dubey et al. in MOGAD. This is a distinctive sagittal T2 hyperintense line surrounded by a hazy T2 hyperintense signal of the anterior and posterior grey matter horns. The 'H sign' on the axial plane indicates central T2 hyperintensity with a weak surrounding T2 hyperintense signal confined to cord grey matter. This sign was found to be present in 28% of MOGAD cases, but only in 8% of NMOSD cases and in none of MS cases [6, 11]. Although a typical 'H sign' could not be elicited in most patients in our cohort, 50% of patients with central cord hyperintensity were found to have a peripheral hazy T2 hyperintense signal (Fig. 5c).

Most MOG antibody-positive patients showed remission of spinal cord lesions on follow-up in a UK study [23]. A similar trend was also noted in a study conducted in USA [24]. In our cohort, the resolution of T2 hyperintense spinal lesions on follow-up was observed in 40% of patients. However, one of those patients had mild cord atrophy post-resolution of abnormal cord signal.

Patients with MOGAD who underwent orbital MRI revealed oedematous, enlarged, and tortuous optic nerves, with bilateral long or short segments of abnormal T2 hyperintense signal that typically involved anterior segments of the optic nerves while sparing the optic chiasm and retro-chiasmatic pathways. Restricted diffusion may or may not be visible in the optic nerve. This finding may help to distinguish MOGAD optic neuritis from NMOSD, the latter of which more typically affects posterior structures [8, 25]. Like how bilateral optic neuritis used to set NMOSD apart from multiple sclerosis, it now seems that MOGAD is much more likely to have bilateral optic neuritis than NMOSD. Our findings are consistent with a similar discovery made by Kitley et al., who found that more than 60% of MOGAD patients had bilateral optic neuritis [23]. However, in our study, the total number of patients with imaging features of optic neuritis was significantly lower than in most other studies.

17.6 mm was determined to be a cut-off point discriminating NMO from MS optic nerve lesions based on a prior study conducted by Mealy et al. in NMOSD. The average length of the optic nerve lesion in our cohort of MOGAD patients (19.1 mm) was even longer than the optic nerve lesions described in that study group of NMOSD patients [8]. Enhancement of the peri-optic nerve sheath was another striking feature that was observed in 50% of patients who underwent contrast study. Similar findings were also described in MOGAD in various studies performed across the globe [26, 27].

This study has potential limitations. Firstly, the sample size of this study is relatively small as MOGAD is a rare and newly described neuroinflammatory disease. Therefore, further research with a larger sample size is necessary to substantiate the findings described in this study. Secondly, contrast-enhanced MRI was not warranted in every patient as the contrast studies were performed only if requested by the treating team. Due to the same reason, orbital and spinal cord MRIs were not performed for all patients. As a result, we may have overlooked some abnormal findings in patients who did not undergo contrast studies as well as in asymptomatic patients without orbital and spinal cord MRIs. Finally, all patients who did not come for review imaging might not necessarily be in remission. Some of them might have been lost in follow-up.

Conclusions

We could identify numerous characteristic radiological features that could help distinguish MOGAD from NMOSD and MS. Firstly, in contrast to MS and NMOSD, lesions in the brain typically affect the brainstem and deep white matter. Additionally, the resolution of CNS lesions shall point to a MOGAD-specific healing mechanism. Furthermore, longitudinally extensive optic neuritis in MOGAD is usually bilateral,

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affects the anterior structures, and may have peri-optic nerve sheath enhancement. Lastly, spinal cord lesions tend to be longitudinally extensive, mostly involve the dorsal spine, and may have central cord hyperintensity with a peripheral hazy T2 hyperintense signal. We hope our findings help clinicians systematically evaluate and manage patients with suspicious neuroinflammatory diseases.

Abbreviations

ADC	Apparent diffusion coefficient
ADEM	Acute disseminated encephalitis
ASL	Arterial spin labelling
AQP4	Aquaporin-4
CT	Computed tomography
DWI	Diffusion weighted imaging
FLAIR	Fluid-attenuated inversion recovery
LETM	Longitudinally extensive transverse myelitis
MOG	Myelin oligodendrocyte glycoprotein
MOGAD	Myelin oligodendrocyte glycoprotein antibody disease
MRI	Magnetic resonance imaging
MS	Multiple sclerosis
NMOSD	Neuromyelitis optica spectrum disorders

Availability of data and materials
All the images used and analysed in the current study are MRI images of brain, spine, and orbit of MOGAD patients available in the PACS of the Department of Radiology at St. John's Medical College. Rest of the datasets analysed in this study are attached as supplementary files.

Declarations

Ethics approval and consent to participate
The study was conducted in St. John's Medical College, Bangalore, and was approved by the institutional ethics committee (IEC Study Reference No. 349/2020). Written consent was taken from all the participants of the study as per the requirements of the institutional ethics committee.

Consent for publication
Consent for publication of this scientific study was obtained from the participants as well as from the Head of the Department of Radiology and the Dean of St. John's Medical College, Bangalore.

Competing interests
The authors hereby declare that they do not have any competing interests.

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