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ORIGINAL COMMUNICATION

Unilateral cortical FLAIR-hyperintense Lesions in Anti-MOG-associated Encephalitis with Seizures (FLAMES): characterization of a distinct clinico-radiographic syndrome

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Abstract

Objective To characterize the clinical symptoms and magnetic resonance imaging (MRI) findings of unilateral cortical FLAIR-hyperintense Lesions in Anti-MOG-associated Encephalitis with Seizures (FLAMES).

Methods This is a case report and systematic review of the literature to identify cases of unilateral cortical FLAMES. Cases were reviewed to determine the frequency of clinical symptoms (seizures, headache, fever and cortical symptoms referable to FLAMES location), and to determine whether MRI abnormalities are restricted to the unilateral cortex in this syndrome.

Results We identified 20 cases of unilateral cortical FLAMES for review. Among them, 17/20 (85%) had seizures, 14/20 (70%) had headache, 13/20 (65%) had fever, 11/20 (55%) reported cortical symptoms referable to the FLAMES location, and 19/20 (95%) reported at least two of these four findings. On MRI 4/20 (20%) had some contralateral hemispheric cortical signal abnormality, and 6/20 (30%) had MRI findings concerning for meningeal inflammation.

Conclusions In patients with unilateral cortical FLAMES, the clinical symptoms of seizures, headache, fever and cortical symptoms referable to the FLAMES location are frequent. Although initially described as a unilateral cortical encephalitis, bilateral cortical involvement and possible meningeal inflammation could indicate a broader disease spectrum. Recognition of this distinct clinico-radiographic syndrome may facilitate prompt diagnosis and treatment.

Keywords MOG · Myelin oligodendrocyte glycoprotein · Encephalitis · Autoimmune · Seizures · MRI

Introduction

Anti-myelin oligodendrocyte glycoprotein (MOG) antibody positivity defines a subset of patients with inflammatory demyelinating diseases of the central nervous system (CNS), most commonly optic neuritis and/or transverse myelitis [1]. Unilateral cortical encephalitis is a rare anti-MOG phenotype that was first reported by Ogawa et al. [1–12] in 2017. All four patients described by Ogawa et al. [2] had seizures, unilateral cortical hyperintensities best seen on magnetic resonance imaging (MRI) T2-weighted fluid-attenuated inversion recovery (T2-FLAIR) sequences, and MOG-IgG positivity. After their index case, Ogawa et al. [2] identified three more patients by testing for MOG-IgG in a cohort of 24 consecutive adult patients with steroid-responsive encephalitis of unknown etiology; these three MOG-IgG-positive patients had similar distinct unilateral cortical T2-FLAIR hyperintensities in contrast to none of the MOG-IgG-negative patients, suggesting this neuroimaging finding is characteristic of anti-MOG-associated encephalitis. Since this initial report by Ogawa et al. less than 20 cases have been published in the literature, and the clinico-radiographic features of this rare entity remain incompletely described. We present an exemplary case of a unilateral cortical FLAIR-hyperintense lesion in anti-MOG-associated encephalitis with seizures, which we refer to as FLAMES, and systematically review the literature to better characterize this unique clinico-radiographic syndrome.

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Case report

A 23-year-old right-handed man presented to hospital with fever, acute-onset aphasia and right-sided weakness, preceded by 10 days of headache. Brain MRI showed T2-FLAIR hyperintensity and swelling of the left frontal cortex, as well as adjacent sulcal T2-FLAIR hyperintensity and leptomeningeal enhancement (see Fig. 1). On T2-weighted imaging the abnormality was not as clearly visualized, but there was cortical brightness on diffusion-weight imaging (DWI) that spared the subarachnoid space (see Fig. 1). There was subtle corresponding darkness on the apparent diffusion coefficient (ADC) map, suggesting true diffusion restriction. Routine electroencephalogram (EEG) showed left hemispheric slowing, in keeping with a lesion or a post-ictal state. He was suspected to have infectious encephalitis and started on empiric antimicrobial therapy, as well as phenytoin in case of seizures. Lumbar puncture revealed a normal opening pressure of 19 cm H_2O, 85 white blood cells (WBC)/μL (64% lymphocytes), elevated protein and normal cerebrospinal fluid (CSF)/serum glucose ratio. Extensive testing for infectious, autoimmune or malignant causes of encephalitis were non-diagnostic (see Table 1), but MOG-IgG was not sent due to absence of demyelinating lesions on brain MRI.

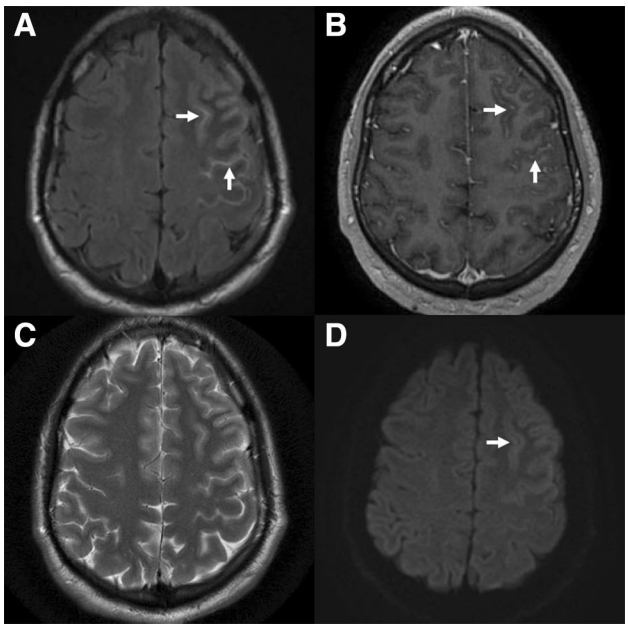

Fig. 1 Brain magnetic resonance imaging of our patient with a unilateral cortical FLAIR-hyperintense Lesion in Anti-MOG-associated Encephalitis with Seizures (FLAMES). On axial T2-weighted fluid-attenuated inversion recovery (T2-FLAIR) image pre-gadolinium, cortical swelling and hyperintensity of both the left frontal cortex and adjacent sulci is seen (**a**, arrows). On axial T1-weighted image post-gadolinium, corresponding leptomeningeal enhancement is also seen (**b**, arrows). The cortical swelling is not as well visualized on axial T2-weighted image (**c**). On axial diffusion-weighted image there is brightness of the cortex with sparing of the subarachnoid space (**d**, arrow). Corresponding subtle darkness on apparent diffusion coefficient map was seen, compatible with true diffusion restriction (not shown)

On day 3 he developed left hemispheric-onset seizures that were captured on continuous EEG and clinically characterized by aphasia, head deviation to the right and right arm jerking, so levetiracetam was added for seizure control. On day 4 of admission he suffered intractable high-pressure headache, and therapeutic lumbar puncture revealed an elevated opening pressure of 31 cm H_20, 599 WBC/μL (34% lymphocytes, 49% neutrophils), elevated protein, a normal CSF/serum glucose ratio and the absence of oligoclonal bands. On day 10 severe high-pressure headache mandated another therapeutic lumbar puncture that again revealed an elevated opening pressure of 39 cm H_20, 182 WBCs/μL (71% lymphocytes), elevated protein and normal CSF/serum glucose ratio. Intravenous methylprednisolone 1 g daily was started for possible autoimmune encephalitis and by day 17 he had no further headache, seizures or neurologic deficits. He was discharged on a 4-week prednisone taper and levetiracetam for his seizure tendency. On follow-up clinical assessment 6 months later he was now asymptomatic. Repeat brain MRI 4 months after initial presentation showed near-resolution of the initial abnormalities, with only subtle residual left frontal cortical and sulcal hyperintensity on T2-FLAIR images. There were, however, new T2-hyperintensities in the pons and subcortical white matter (see Fig. 2). Anti-MOG-associated disease was suspected, and serum testing for MOG-IgG was positive by EUROIMMUN cell-based assay (CBA). Serum and CSF from initial presentation were retrospectively tested for MOG-IgG and were also positive by EUROIMMUN CBA (titres not performed).

Systematic review of the literature for unilateral cortical FLAMES

Two authors (AB and AM) reviewed the literature for cases of unilateral cortical FLAMES. We searched PubMed and EMBASE for '[unilateral] AND [MOG]', '[unilateral] AND [encephalitis]', and '[MOG] AND [encephalitis]'. All relevant published articles dating back 10 years from April 18 2019 were reviewed for potential study inclusion. Cases were included if they (a) had predominantly unilateral cortical T2-FLAIR hyperintensity at presentation, without the involvement of the adjacent juxta-cortical white matter on MRI and (b) MOG-IgG antibodies were identified by CBA in serum and/or CSF. Patients were excluded if insufficient patient data were provided. A total of 938 search results were screening for potential inclusion in this review. Any

Table 1 List of investigations which were normal or negative in our patient

Malignancy and toxic/metabolic testing
CSF cytology and flow cytometry
Computed tomography of thorax/abdomen/pelvis (incidental pancreatic cystic lesion found but unlikely malignancy, only routine radiographic follow-up recommended)
Ultrasound of scrotum
Serum and CSF lactate
Toxin screen for opioids, cannabinoids, cocaine, amphetamines, barbiturates, benzodiazepines
Infectious testing
Serology for HIV, syphilis, hepatitis B and C, HTLV, CMV, EBV, arboviruses, toxoplasma, brucella (indeterminate result, testing laboratory stated likely not relevant to clinical presentation), bartonella, toxocara, tularensis, lyme, histoplasmosis, blastomycosis, coccidioidomycosis, Q fever (IgG reactive but IgM non-reactive), leptospirosis (IgM positive but gold standard confirmatory MAT negative × 2)
CSF testing for HSV-1/HSV-2 PCR, VZV PCR, enterovirus PCR, AFB culture, bacterial gram stain and culture, cryptococcal antigen, fungal culture, bacterial 16S ribosomal RNA
TB skin test
Rheumatologic and autoimmune neurologic testing
Serum antiphospholipid antibodies, lupus anticoagulant, p-ANCA, c-ANCA, rheumatoid factor, ANA, IgG4, immunoglobulins (IGAM)
CSF and serum ACE
Serum for anti-Hu, Yo, Ri, Ma2, CV2 and amphiphysin antibodies
Serum and CSF for anti-NMDAR, AMPAR, $GABA_BR$, LGI1, CASPR2, DPPX, GAD65 and IgLON5 antibodies
Serum and CSF for anti-aquaporin-4 antibodies

ACE angiotensin-converting enzyme, *AFB* acid fast bacillus, *AMPAR* alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor, *ANA* anti-nuclear antibody, *c-ANCA* cytoplasmic anti-neutrophilic cytoplasmic antibody, *ASPR2* contactin-associated protein-like 2, *CMV* cytomegalovirus, *CSF* cerebrospinal fluid, *DPPX* dipeptidyl-peptidase-like protein 6, *EBV* Ebstein-Barr virus, $GABA_BR$ gamma-aminobutyric acid B receptor, *GAD65* glutamic acid decarboxylase-65, *HIV* human immunodeficiency virus, *HSV* herpes simplex virus, *HTLV-1* human T-lymphotropic virus type 1, *LGI-1* leucine-rich glioma inactivated 1, *MAT* microscopic agglutination test, *NMDARN*-methyl-D-aspartate receptor, *p-ANCA* perinuclear anti-neutrophilic cytoplasmic antibody, *PCR* polymerase chain reaction, *RNA* ribonucleic acid, *TB* tuberculosis, *VZV* varicella zoster virus

discrepancy between reviewers regarding inclusion was resolved by discussion to achieve consensus. Given our patient's clinical features of seizures, headache, fever and cortical symptoms referable to his FLAMES location, we categorically searched for the presence or absence of these symptoms in reported cases.

We identified 24 patients, including our own, for potential study inclusion. Four patients were ultimately excluded (1 reported as unilateral cortical T2-FLAIR hyperintensity but juxta-cortical white matter also involved on neuroimaging; 1 unilateral meningeal enhancement without cortical T2-FLAIR hyperintensity; 1unilateral cortical T2-FLAIR hyperintensity but anti-MOG antibodies not tested; 1 insufficient patient data provided), resulting in 20 patients with unilateral cortical FLAMES for this review. The mean age was 29 years (range 11–46 years) and 12/20 (60%) were male. The frequency of symptoms among cases were as follows: 17/20 (85%) reported seizures, 14/20 (70%) reported headache, 13/20 (65%) reported fever, 11/20 (55%) reported cortical symptoms referable to the FLAMES location, and 19/20 (95%) reported at least two of these four findings (see Table 2). Although first described as a strictly unilateral phenomenon, 4/20 (20%) showed some contralateral hemispheric cortical signal abnormality on MRI either at presentation or with disease progression. Furthermore, despite initial characterization as a cortical encephalitis, 6/20 (30%) reported adjacent T2-FLAIR sulcal hyperintensity and/or post-gadolinium leptomeningeal enhancement on MRI. Clinical and radiographic improvement was seen in all patients after the administration of corticosteroids for whom this outcome was reported, although relapses occurred when steroids were rapidly discontinued (see Table 3).

Discussion

In this case report and systematic literature review focusing on clinical symptoms and MRI findings of unilateral cortical FLAMES, we have determined distinctive features of this syndrome. Seizures were highly prevalent as described in the initial description by Ogawa et al [2]. In addition to seizures, however, the presence of headache, fever and cortical symptoms referable to the FLAMES location were each present in over half of cases published in the literature. The high prevalence of these findings is in spite of probable under-reporting due to the retrospective nature of data collection. Fever and headache, particularly severe headache with elevated intracranial pressure as was seen in our patient, may lead to diagnostic confusion with CNS infection despite MRI findings typical of unilateral cortical FLAMES.

Although unilateral cortical T2-FLAIR hyperintensity is characteristic of this disease, contralateral hemispheric

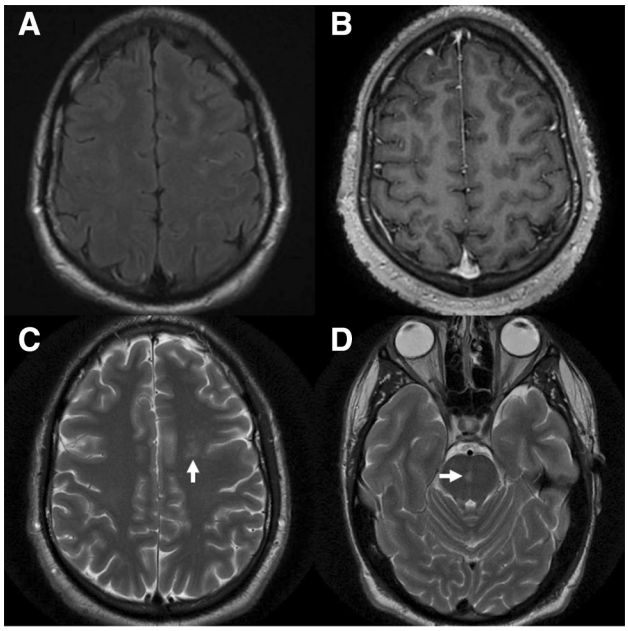

Fig. 2 Follow-up brain magnetic resonance imaging of our patient with a unilateral cortical FLAIR-hyperintense Lesion in Anti-MOG-associated Encephalitis with Seizures (FLAMES). On axial T2-weighted fluid-attenuated inversion recovery (T2-FLAIR) image pre-gadolinium, near-complete resolution of the previously demonstrated left frontal cortical and sulcal hyperintensity is seen (**a**). On axial T1-weighted image post-gadolinium, complete resolution of the previously demonstrated leptomeningeal enhancement is also seen (**b**). On axial T2-weighted image, however, there are now hyperintense lesions in the left frontal subcortical white matter (**c**, arrow) and pons (**d**, arrow)

cortical signal abnormality was reported in 4/20 cases; unilateral cortical FLAMES may therefore exist on a spectrum of anti-MOG-associated encephalitis that includes more fulminant bilateral cortical lesions [11]. Importantly, the T2-FLAIR hyperintensities seen in this condition are distinct but not pathognomonic, and other diseases involving either the cortex (e.g., seizures, Creutzfeldt-Jakob disease) or the subarachnoid space (e.g., subarachnoid hemorrhage, carcinomatous meningitis) merit consideration [13]. This is because in addition to cortical T2-FLAIR hyperintensity, adjacent sulcal T2-FLAIR hyperintensity and/or leptomeningeal enhancement was reported in 6/20 cases and has led some authors to posit a primary meningeal process [4, 6]. Corresponding DWI hyperintensity of the swollen cortex in our case clearly spared the subarachnoid space, arguing against primary meningeal pathology despite sulcal T2-FLAIR hyperintensity and leptomeningeal enhancement. Local cortical swelling causing mass effect may itself lead to these sulcal abnormalities on MRI through the alteration of regional flow dynamics and compression of leptomeningeal vessels, so in isolation they are not definite evidence of leptomeningeal inflammation [14]. Interestingly, however, a patient with asymptomatic unilateral hemispheric leptomeningeal enhancement and anti-MOG antibodies has been described [15]. No cortical T2-FLAIR hyperintensity or swelling was reported in this case, and so the possibility of primarily meningeal inflammation extending to the cortex and causing symptoms in unilateral cortical FLAMES cannot be excluded. Pathologic data in this syndrome are sparse but lymphocytic infiltration of both the subarachnoid space and brain parenchyma with perivascular involvement has been reported, potentially indicating both meningeal and cortical inflammation [3, 7]. Of note, pathologic evidence of demyelination is largely absent, calling into question whether anti-MOG antibodies are pathogenic or an epiphenomenon of another immune-mediated disease mechanism [3, 7]. Anti-MOG antibodies have been found to overlap with other autoimmune encephalitides, including anti-*N*-methyl-D-aspartate receptor (NMDAR) encephalitis [9]; although neither anti-NMDAR antibodies nor other established antibodies against onconeural, cell-surface or synaptic proteins have consistently been identified in unilateral cortical FLAMES, the possibility of an as of yet uncharacterized antibody causing this syndrome warrants further investigation.

Conclusion

Unilateral cortical FLAMES has emerged as a radiographic marker of a distinct anti-MOG-associated disease. Despite the small number of cases and retrospective nature of data collection, this study determines the typical clinical findings (seizures, headache, fever and focal cortical symptoms) of this unique anti-MOG phenotype. Although initially described as a unilateral cortical encephalitis, bilateral cortical involvement and possible meningeal inflammation in some reports may reflect a broader disease spectrum. The syndrome is highly steroid-responsive so identifying the typical clinical symptoms and neuroimaging features of unilateral cortical FLAMES is essential for prompt treatment. Improved recognition of these patients may also facilitate larger cohort studies to better determine the pathophysiology, optimal treatment regimen and long-term prognosis of this condition.

Table 2 Clinical and MRI features of patients with unilateral cortical FLAMES

Publication	Age/Sex	Fever reported?	Headache reported?	Seizures reported?	Symptoms referable to FLAMES location reported?	Other encephalitic symptoms reported?[a]	Unilateral cortical FLAIR hyperintensity?	Sulcal FLAIR hyperintensity and/or meningeal enhancement?
Budhram 2019	23/M	Yes	Yes	Yes	Yes, aphasia, hemiparesis	Yes, irritability	Yes	Yes
Patterson 2019	39/F	Yes	Yes	No	Yes, aphasia	Yes, agitation	Yes	Yes
Cobo-Calvo 2018	37/M	No	No	Yes	Yes, hemiparesis and hemianopia	No	Yes	No
Adachi 2018	27/M	Yes	Yes	Yes	Yes, aphasia	Yes, confused, irritated	Yes, also lesser involvement of contralateral cortex	No
Sugimoto 2018	19/F	Yes	Yes	Yes	No	No	Yes, considered secondary to meningeal process; also subtle contralateral cortical DWI hyperintensity	Yes
Ikeda 2018	29/F	Yes	No	Yes	No	No	Yes, with progression to involvement of contralateral cortex	Yes
Wang 2018 (1)[b]	20/F	Yes	Yes	Yes	No	Yes, decreased level of consciousness	Yes	No
Wang 2018 (2)[b]	31/M	Yes	Yes	Yes	No	Yes, lethargy	Yes	Yes
Wang 2018 (3)[b]	11/F	Yes	Yes	Yes	Yes, hemiparesis	Yes, lethargy	Yes	No
Wang 2018 (4)[b]	19/M	No	Yes	Yes	No	Yes, memory impairment	Yes	No
Wang 2018 (5)[b]	23/M	Yes	No	Yes	No	Yes, cognitive decline	Yes	No
Zhou 2017	31/M	Yes	Yes	Yes	No	No	Yes	No
Fukushima 2017	17/F	No	No	Yes	Yes, aphasia	No	Yes	No
Fujimori 2017	46/M	Yes	Yes	Yes	Yes, focal-onset seizures clinically	Yes, dizziness then memory decline, lethargy, paraparesis	Yes, with progression to bi-frontal cortical involvement	No, not at initial presentation but later bi-frontal enhancement
Ogawa 2017 (1)[b]	39/M	No	No	Yes	Yes, post-ictal hemiparesis	Yes, paranoia, hallucinations	Yes	No
Ogawa 2017 (2)[b]	36/M	No	No	Yes	No	No	Yes	No (enhancement interpreted as cortical)
Ogawa 2017 (3)[b]	23/M	No	Yes	Yes	Yes, focal-onset seizures clinically	No	Yes	No (only equivocal enhancement reported)
Ogawa 2017 (4)[b]	38/M	No	Yes	Yes	Yes, aphasia, hemiparesis	Yes, agitation and violent behaviour	Yes	No
Yamamoto 2017	29/F	Yes	Yes	No	Yes, right arm tremor	No	Yes	No
Numa 2016	37/F	Yes	Yes	No	No	No	Yes, considered secondary to meningeal process	Yes

DWI diffusion-weighted imaging, *FLAIR* fluid-attenuated inversion recovery, *MRI* magnetic resonance imaging

[a]Other encephalitic symptoms defined as any decreased or altered level of consciousness, lethargy, cognitive decline, psychiatric symptoms or alteration in personality/behaviour not reported to be directly related to seizures

[b]Multiple patients in a single publication who met inclusion criteria for this study have been numbered in the order they appeared in the original publication

Table 3 CSF findings, response to steroids and history of typical CNS demyelinating syndromes in patients with unilateral cortical FLAMES

Publication	CSF pleocytosis?[a]	CSF OCB?	Clinical response to steroids?	Imaging response to steroids?	History of typical CNS demyelinating syndrome?
Budhram 2019	Yes, 599 WBC/μL	No	Yes	Yes	No
Patterson 2019	Yes, 146 WBC/μL	Not reported	Yes	Yes	No
Cobo-Calvo 2018	Yes, 120 WBC/μL	No	Yes[b]	Not done	No
Adachi 2018	Yes, 205 WBC/μL	Not measured	Yes	Yes	No
Sugimoto 2018	Yes, 200 WBC/μL	No	Yes, but relapsed after no steroid taper	Yes	Yes, LETM
Ikeda 2018	Yes, 73 WBC/μL	No	Yes	Yes	Yes, bilateral ON
Wang 2018 (1)[d]	Yes, 15 WBC/ μL	Not reported	Yes[c]	Yes	Yes, subsequent ADEM and right ON
Wang 2018 (2)[d]	Yes, 142 WBC/μL	Not reported	Yes[c]	Yes	Yes, subsequent left ON and ADEM
Wang 2018 (3)[d]	Yes, 76 WBC/μL	Not reported	Yes[c]	Yes	Yes, subsequent right ON and then left ON
Wang 2018 (4)[d]	No, 3 WBC/μL	Not reported	Yes[c]	Yes	Yes, previous bilateral ON
Wang 2018 (5)[d]	Yes, 500 WBC/μL	Not reported	Yes[c]	Yes	Yes, bilateral ON
Zhou 2017	Yes, 142 WBC/μL	No	Yes, but relapsed after rapid taper	Yes	Yes, subsequent left ON and ADEM
Fukushima 2017	Yes, 137 WBC/μL	Yes	Yes	Not reported	Yes, multiple ADEM presentations
Fujimori 2017	Yes, 56 WBC/μL	Yes	Yes	Yes	Yes, subsequent right ON
Ogawa 2017 (1)[d]	Yes, 29 WBC/μL	Not measured	Yes	Yes	Yes, previous right ON
Ogawa 2017 (2)[d]	Yes, 63 WBC/μL	No	Yes	Yes	Yes, right ON
Ogawa 2017 (3)[d]	Yes, 101 WBC/μL	No	Yes	Yes	No
Ogawa 2017 (4)[d]	Yes, 311 WBC/μL	No	Yes	Yes	No
Yamamoto 2017	Yes, 349 WBC/μL	Not reported	Yes, but relapsed after steroid taper	Not reported	No
Numa 2016	Yes, 17 WBCs/ μL	No	Yes, but relapsed after no steroid taper	Yes	Yes, right ON, previous ADEM

ADEM acute disseminated encephalomyelitis, *CNS* central nervous sytem, *CSF* cerebrospinal fluid, *LETM* longitudinally extensive transverse myelitis, *OCB* oligoclonal bands, *ON* optic neuritis, *WBC* white blood cell count

[a]If patient had multiple CSF analyses during clinical assessment, highest CSF WBC count selected for table

[b]Clinical response to steroids based on initial presentation compared to outcome Disability Status Scale (DSS)

[c]Clinical response to steroids based on initial presentation compared to modified Rankin Scale (mRS) at last follow-up

[d]Multiple patients in a single publication who met inclusion criteria for this study have been numbered in the order they appeared in the original publication

Author contributions AB assessed the patient, performed the review of the literature, and drafted the manuscript, figure and tables. AM assessed the patient, performed the review of the literature, and edited the manuscript and tables for important intellectual content. CL assessed the patient, drafted the tables and edited the manuscript for important intellectual content. SMH-M assessed the patient and edited the manuscript for important intellectual content. MS reviewed the neuroimaging and edited the manuscript for important intellectual content. MWN assessed the patient and edited the manuscript for important intellectual content.

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Compliance with ethical standards

Conflicts of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

Ethical standards The authors hereby declare that the research documented in the submitted manuscript has been carried out in accordance with ethical standards.

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