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A burning encephalitis: Fluid-attenuated inversion recovery-hyperintense lesions in Anti-myelin oligodendrocyte glycoprotein-associated encephalitis with seizures in anti-myelin oligodendrocyte glycoprotein-associated encephalitis with seizures—A case report and review of the literature

Ibtissam El Ouali[1,2], Amine Naggar[1,2], Kenza Berrada[1,2], Mohamed Jiddane[1,2] and Firdaous Touarsa[1,3]

Abstract

FLAMES, or fluid-attenuated inversion recovery-hyperintense lesions in anti-myelin oligodendrocyte glycoprotein (anti-myelin oligodendrocyte glycoprotein)-associated encephalitis with seizures, represents a rarely documented syndrome characterized by ambiguous features. Positioned within the spectrum of inflammatory demyelinating diseases of the central nervous system, it is regarded as a distinct subset of myelin oligodendrocyte glycoprotein antibody-associated disease, the latest classification in this domain. Myelin oligodendrocyte glycoprotein antibody-associated disease exhibits a diverse clinical spectrum, spanning from solitary optic neuritis or myelitis to multifocal central nervous system demyelination, manifesting as acute disseminated encephalomyelitis, or cortical encephalitis accompanied by seizures, delineating the fluid-attenuated inversion recovery-hyperintense lesions in anti-myelin oligodendrocyte glycoprotein-associated encephalitis with seizures syndrome. We present a compelling case study of a 30-year-old individual with a history of recurrent seizures initially diagnosed with mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes. However, the disease's progression more closely resembled self-resolving cerebral cortical encephalitis linked with myelin oligodendrocyte glycoprotein antibodies. In addition, we undertake a systematic review of literature cases to explore the diagnostic significance of magnetic resonance angiography, fluid-attenuated inversion recovery, and specialized markers such as diffusion-weighted imaging and perfusion in discerning fluid-attenuated inversion recovery-hyperintense lesions in anti-myelin oligodendrocyte glycoprotein-associated encephalitis with seizures syndrome and elucidating its distinctive characteristics.

Keywords

Focal encephalitis, MOGAD, demyelination, ASL hyper-perfusion, MRI FLAIR, differential diagnosis

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Introduction

Since Ogawa et al.[1] first described FLAMES, FLAIR-hyperintense lesions in anti-MOG associated encephalitis with seizures, less than 20 cases have been identified in the literature, without a wholesome description of its clinical and radiographic features.

[1]Ibn Sina Hospital, Salé, Morocco
[2]Neuroradiology Department, Specialty Hospital of Rabat, Rabat, Morocco
[3]Department of Internal Medicine, Ibn Sina Hospital, Salé, Morocco

Corresponding Author:
Ibtissam El Ouali, Lalla Asmaa Avenue, Salé 11010, Morocco.
Email: Ibtissam.elouali94@gmail.com

Henceforth, FLAMES is identified as a subtype of the MOG antibody-associated disease (MOGAD). This demyelinating entity is commonly affecting more males than females with an age profile ranging from 11 to 46 years.[2]

Physical signs include seizures with additional symptoms such as fever and headache, and specific cortical symptoms such as aphasia. Magnetic resonance imaging (MRI) shows unilateral cortical signal anomalies distinctive of FLAMES syndrome with FLAIR-variable unilateral enhancement of the leptomeninges (FUEL).[3]

Autoantibodies against myelin oligodendrocyte glycoprotein (MOG), an exclusive surface protein of central nervous system oligodendrocytes and myelin sheaths, result in variable phenotypes such as acute disseminated encephalomyelitis (ADEM), optic neuritis, transverse myelitis, and recently added, cerebral cortical encephalitis.[4]

Case presentation

A 30-year-old North African male was admitted to our hospital due to seizures, two similar episodes of headache, memory dysfunction, sensory symptoms, decreased visual acuity, and seizures.

During the first episode, the patient presented left-sided paresthesia and transcortical aphasia. The first cerebral MRI with gadolinium showed significant right hemispheric hyperintensity on T2-fluid-attenuated inversion recovery (FLAIR), cortical hyperintensity on diffusion-weighted imaging (DWI) with overlying leptomeningeal enhancement, perfusion image obtained by arterial spin labeling (ASL) technique revealed hyperperfused areas in the right cerebral hemisphere (Figure 1) raising suspicions of MELAS (mitochondrial encephalomyopathy, lactic acidosis, stroke-like episodes). Serologic testing for human immunodeficiency virus, syphilis, and anti-nuclear antibodies was negative. Body computed tomography showed no evidence of malignancy or granulomatosis such as sarcoidosis. Cerebrospinal fluid (CSF) presented a normal opening pressure, normal protein level, normal glucose, normal cytology, normal CSF/serum IgG index, and no CSF-specific oligoclonal bands. Consequently, the patient was given anti-epileptic drugs. Clinical symptomatology resolved spontaneously and the patient remained under the same symptomatic treatment. One-month follow-up brain MRI showed complete resolution of previously described abnormalities (Figure 2).

Two months later, the patient was readmitted to the hospital due to seizures, memory dysfunction, and right upper limb paresis. MRI found new contralateral temporal and occipital lesions, hyperintense on T2-FLAIR with cortical swelling. On axial DWI, there is the brightness of the cortex (d, arrow), with corresponding subtle darkness on the apparent diffusion coefficient map compatible with true diffusion restriction associated with overlying leptomeningeal enhancement. Perfusion image obtained by ASL technique revealed focal hyperperfusion in the left occipital areas, magnetic resonance spectroscopy found a high choline peak, reverse Chol/Cr ratio, decreased NAA peak, and also prominent lactate (Lac) and lipids (Lip) resonance, represented as a doublet peak in the MR spectra at 1.31 ppm, consistent with an inflammatory spectral profile. The unaffected parenchyma evaluated by the second voxel showed normal spectra (Figure 3). This retrospective clinicoradiographic presentation is typical of fleeting, unilateral cortical FLAIR-hyperintense lesions in anti-MOG-associated encephalitis with seizures (FLAMES). Testing for MOG abs in serum (MOG)-IgG (fixed cell-based assay, EUROIMMUN) was positive. The most recent CSF was positive for anti-MOG antibodies. The patient continued to receive antiepileptic medications and was given 500 mg intravenous methylprednisolone daily for a week, which has been gradually tapered down over 14 weeks.

After this episode, the patient remained symptom-free at 1-month follow-up.

Discussion

Epidemiology

FLAMES is a rare, newly described phenotype of MOGAD, which clinically overlaps with multiple inflammatory demyelinating diseases of the CNS, like aquaporin 4 antibody-positive neuromyelitis optica spectrum disorders (NMOSD), ADEM, and multiple sclerosis. However, it is considered to be an independent entity.[5]

Certain epidemiology data for FLAMES are not available since myelin oligodendrocyte glycoprotein immunoglobulin G was first discovered in 2007 and antibody testing was only widespread a decade later.[6]

According to previous studies, less than 20 cases of MOG antibody-positive cortical cerebral encephalitis (CCE) have been reported, indicating a slightly high prevalence of this disease in young males with a mean age of 29 years. The most frequent presentation is reported to be a decrease in visual acuity in almost 70% of cases.[7]

Pathophysiology

MOG stands as a markedly immunogenic protein intrinsic to myelin, primarily localized on the surface of oligodendrocytes and forming the outermost layer of myelin sheaths within the CNS.

It plays a role in cell adhesion and stability and can participate in modulating the immune response of myelin.[8]

Human pathology of biopsy or autopsy specimen revealed coexisting perivenous and confluent destruction of oligodendrocytes and myelin sheaths in cortical gray matter by MOG antibodies.[8,9]

Studies by Glasser and Van Essen,[10] based on the measured ratio of T1w and T2w on MRI-generated maps of cortical myelin content, show a high degree of cortical myelination

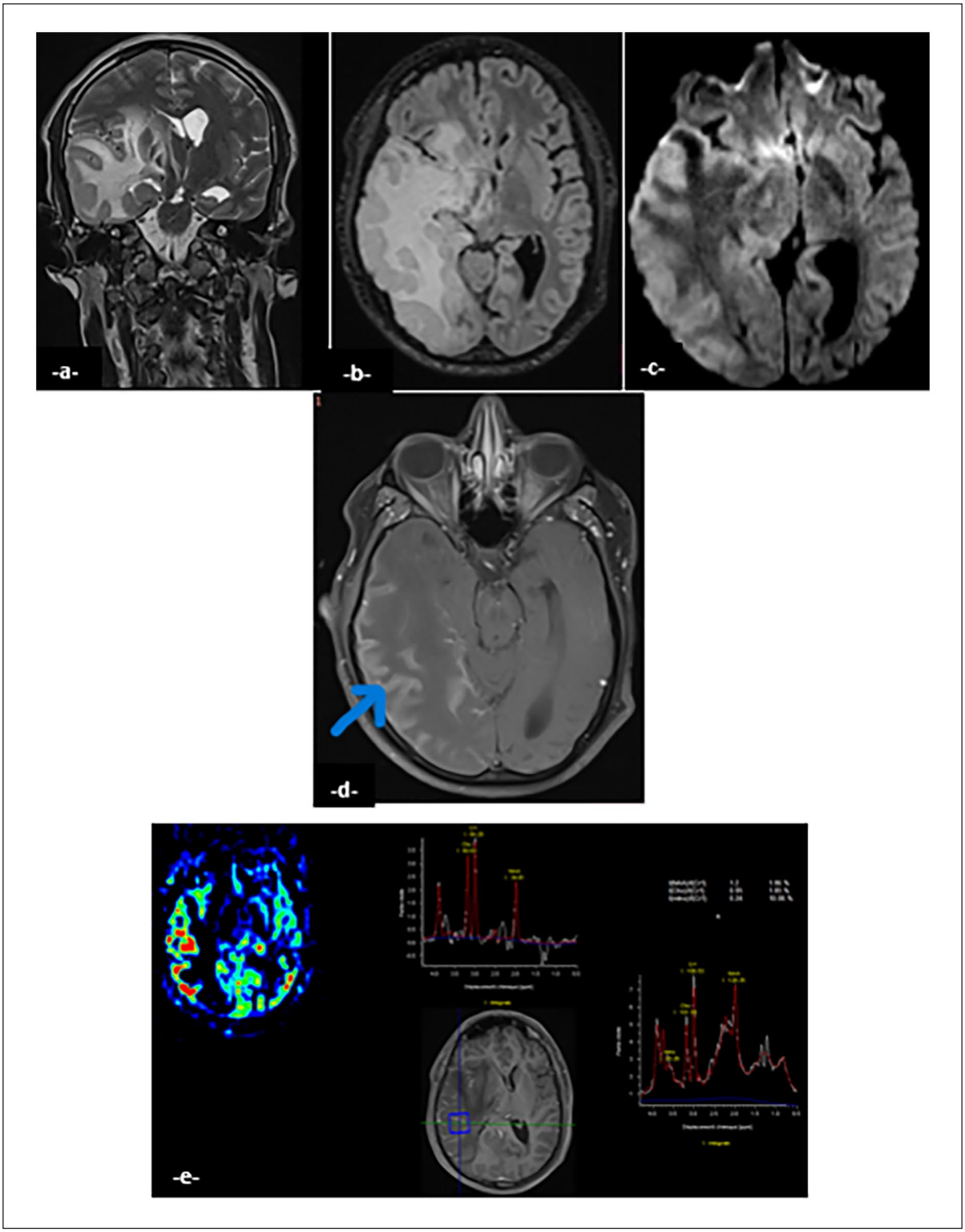

Figure 1. Brain magnetic resonance imaging of the first episode showed on coronal T2: (a) Axial T2-fluid-attenuated inversion recovery. (b) Images of hyperintense lesions of the right hemispheric cortex, subcortical, and periventricular white matter (WM) responsible for a significant mass effect on median structures associated with subtle subcortical hypointensity of corona radiata. Cortical hypersignal on axial diffusion-weighted imaging (c) and an overlying leptomeningeal enhancement (arrows) on axial T1-weighted post-gadolinium (d) Images. Perfusion image obtained by arterial spin labeling shows hyperperfused areas in the right cerebral hemisphere with an inflammatory spectrum on spectroscopy (e): high choline peak, reverse Chol/Cr ratio, decreased NAA (N-acetyl aniline) peak and also prominent lactate (Lac) and lipids (Lip) resonance, represented as a doublet peak in the magnetic resonance spectra at 1.31 ppm.

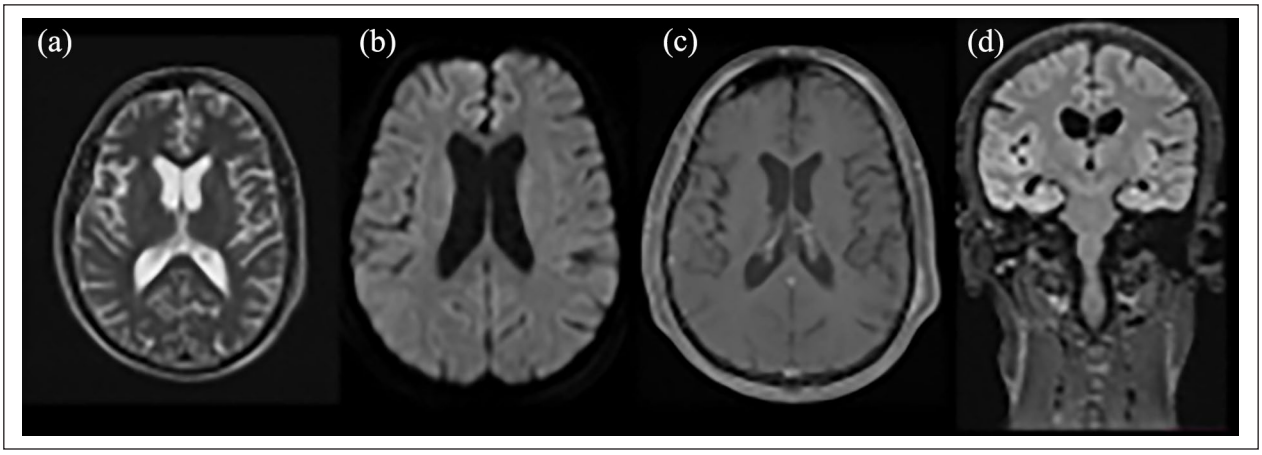

Figure 2. One-month follow-up magnetic resonance imaging on axial T2: (a) Diffusion-weighted imaging, (b) T1 post-gadolinium injection, (c) axial images and coronal T2 fluid-attenuated inversion recovery, and (d) image showed no abnormality.

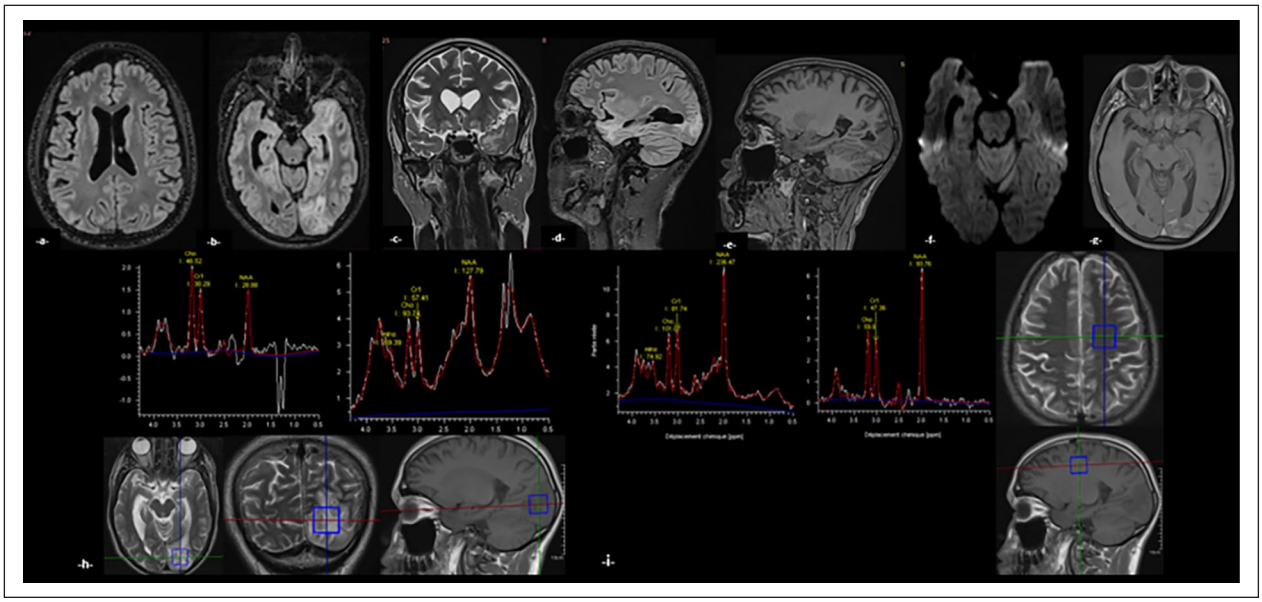

Figure 3. Magnetic resonance imaging after the second admission of the patient showed resolution of right temporal cortical abnormalities, extension of left occipital lesions, and persistence of leptomeningeal enhancement on all sequences (a to e); normal spectroscopy on an unaffected region of cerebral parenchyma (h and i).

in primary cortical areas (motor, somatosensory, visual, and auditory cortices) in comparison with association areas. This cortical demyelination causes profound neural activity alteration, which explains motor deficiency, the associated cognitive decline, and memory dysfunction presented by many patients.[11]

MOG antibodies are detected in several inflammatory demyelinating diseases of CNS, including ADEM, AQP4 seronegative NMOSD, optic neuritis, and transverse myelitis.

The presence of meningeal and cortical inflammation is implied by the documented lymphocytic infiltration occurring in both the subarachnoid space and brain parenchyma, with associated perivascular involvement.[12,13]

Clinical manifestation

The most prevalent neurological presentation was epileptic seizures, followed by optic neuritis, fever, and headache.[14]

As initially described, seizures are the most prevalent presentation in FLAMES.[1]

The presence of severe headache with intracranial hypertension and fever with MRI findings of unilateral cortical lesions is suggestive of the diagnosis of FLAMES. However, CNS infection should be first ruled out.

In addition, cerebral cortical symptoms, such as aphasia, memory dysfunction, and paralysis were frequently reported.[15]

Imaging features

The neuroradiological findings are very characteristic and distinctive of this disease. MRI assessments commonly display the presence of multifocal or diffuse lesions throughout the brain in individuals with MOG-associated encephalitis. These lesions encompass cortical and subcortical regions, white matter tracts, and deep gray matter structures, and occasionally extend to involve the spinal cord.[2]

MRI manifestations of FLAMES often manifest diverse features, including hyperintensity on T2/FLAIR sequences, enhancement post-gadolinium administration indicative of blood–brain barrier disruption, and diffusion restriction suggestive of acute inflammatory or demyelinating processes. The studies have not yet reached conclusive results.[3,4]

Reports in the literature described essentially unilateral cortical hyperintensities, most evident on T2-FLAIR, while T2W sequences show subtle to no abnormalities. However, the existence of bilateral cortical signs abnormalities was documented in the most fulminant forms.[16] This abnormal hyperintensity on FLAIR is indicative of cortical aggression caused by MOGAD-related CCE.

According to prior research, the presence of both cortical T2 FLAIR hyperintense lesions and subcortical white matter T2 FLAIR hypointense lesions is widely regarded as a notably specific indicator of FLAMES syndrome.[17]

However, the additional hyperintensity of juxta cortical WM makes our case very unique, probably reflecting a fulminant form with manifested myelin destruction involving both gray and white matter.

Hyperintensity on DWI is also found as a result of cortical dysfunction by epilepsy.[18]

Leptomeningeal enhancement adjacent to sulcal T2-FLAIR hyperintensity, FUEL, is noted and studies suggest a primary meningeal inflammation included in a broader spectrum of meningo-cortical manifestations as a result of lymphocytic infiltration of the subarachnoid space and the brain parenchyma along with perivascular involvement.[3,19]

Hyperperfusion is seen on single photon emission computed tomography corresponding to the cortical FLAIR hyperintensity areas.

These findings are unique to unilateral cerebral cortical encephalitis (UCCE) and distinct from those of other MOG antibody-associated diseases including ADEM.[1] According to the time course of the diseases, perfusion can detect some focal hypo-perfusion areas in subacute-to-chronic cortical lesions.

Differential diagnosis

Similar neuroimaging features involving the cortex can be encountered in other diseases. Hence, alternative diagnoses relevant to the clinical presentation should be taken into account (Table 1).[20]

Subarachnoid space affections including subarachnoid hemorrhage, infectious, or carcinomatous meningitides are also to be considered as differential diagnoses.

Based on the similarity between these diseases and MOGAD-mediated UCCE, the detection of MOG antibodies is crucial to confirm the diagnosis.

Management guidelines

The prevailing directives for treatment regimens primarily derive from FLAMES case reports, as well as prospective and retrospective studies. There exists a crucial need for tailoring treatment regimens to individual cases. Existing literature delineates instances wherein patients exhibit abrupt symptom amelioration subsequent to corticosteroid administration.

Upon scrutinizing the therapeutic dimension, it becomes apparent that all previously documented cases underwent immunotherapy.

Immunotherapy as an initial treatment often involves high-dose corticosteroids followed by other immunomodulatory agents like intravenous immunoglobulin, plasma exchange (PLEX), or rituximab (RTX) for refractory cases. Notably, in many cases, patients exhibited a favorable response to steroid therapy, a treatment modality recognized for its efficacy in alleviating seizures and associated manifestations of FLAMES syndrome,[26] as also indicated by reports from Ogawa et al.[1].

Nevertheless, a subgroup of patients exhibited complications, for instance, irreversible damage to nerve cells and their connections, leading to permanent neurological deficits, vision loss, and severe cognitive impairment, as highlighted in reports by Fujimori et al.[16] and Sugimoto et al.[19] Consequently, maintenance immunosuppression with medications like azathioprine (AZA), mycophenolate mofetil (MMF), or RTX may be necessary as well for patients with chronic or relapsing autoimmune encephalitis in general.[27]

During maintenance therapy, first-line treatment consists of using immunosuppressive therapy like AZA, MMF, and RTX.[28]

Furthermore, PLEX offers a greater possibility of complete recovery.

Patients may need a combination of antiepileptic drugs (AEDs) to control seizures.[29]

Given the complexity of FLAMES syndrome, individualized care plans involving collaboration among various specialists are crucial to optimize patient outcomes. For the latest and most comprehensive information on FLAMES disease management, consulting recent peer-reviewed literature and guidelines is recommended.[30]

Follow-up MRI scans in most cases show improvement in lesions despite the potential persistence of elevated levels of anti-MOG antibodies.[8]

Table 1. Most important differentials for FLAMES.

Disease	Clinical features	Characteristic MRI features	Keys to diagnosis
Creutzfeldt–Jakob disease[21]	Isolated visual symptoms Isolated cerebellar ataxia Presenile dementia and sleep disturbances	Focal or diffuse, symmetric or asymmetric occipitoparietal hyperintensity at DWI, and/or FLAIR perirolandic area usually spared Symmetric or asymmetric involvement of basal ganglia	clinical features combined with results of at least one paraclinical test (EEG, CSF analysis, and/or MR imaging abnormalities)
Seizure-induced reversible MRI abnormalities (SRMA)[22]	Seizure	Hyperintensity on T2W involving the cortex, hippocampus, and claustrum Cortical laminar necrosis	EEG Reversible abnormalities
Anti-gamma-aminobutyric acid (B) receptor encephalitis[23]	Symptoms of limbic encephalitis often encompass severe epilepsy, typically resistant to multiple antiepileptic medications and unresponsive to immunotherapy interventions.	High T2/FLAIR signal was present in unilateral or bilateral middle temporal lobes Progressive hippocampal atrophy T1w low signal in the hippocampal horn	The presence of anti-GABABR antibodies in CSF and serum
Mitochondrial encephalopathy with lactic acidosis and stroke-like episodes (MELAS)[24]	Stroke-like episodes lactic acidosis encephalopathy, including seizures and migraine-like headaches dementia myopathy sensorineural hearing loss diabetes mellitus	multifocal stroke-like cortical lesions in different stages, crossing the cerebral vascular territories, predilection to the posterior parietal and occipital lobes. Elevated lactate on MR spectroscopy	Identification of a mutation in mtDNA (mtDNA variant) on peripheral blood samples or muscle biopsy
Stroke-like migraine attacks after radiation therapy (SMART)[25]	Following cranial radiation without evidence of residual or recurrent malignancy, individuals may experience prolonged and reversible deficits localized to specific cortical regions, manifesting years after the radiation therapy.	Unilateral gyral hyperintensities appreciated on T2 and FLAIR-weighted imaging with corresponding gadolinium enhancement and cortical thickening, preferentially involving the posterior aspect of the brain, typically sparing the subcortical and deep white matter structures	Clinicoradiological diagnostic criterion
Reperfusion injury	Post-treatment of long-standing severe carotid stenosis	Cerebral white matter edema hypointense on T1 and hyperintense on T2-FLAIR. T2-FLAIR Hyperintense swollen cortex, leptomeningeal enhancement.	Clinical history

CSF: cerebrospinal fluid; DWI: diffusion-weighted imaging; EEG: electroencephalogram; FLAIR: fluid-attenuated inversion recovery; FLAMES: FLAIR-hyperintense lesions in anti-MOG associated encephalitis with seizures; MRI: magnetic resonance imaging; mtDNA: mitochondrial deoxyribonucleic acid.

It might reveal alterations in lesion characteristics and distribution patterns over time. While certain lesions may exhibit resolution or reduction in size, the emergence of new lesions may indicate persistent inflammatory activity. Dynamic changes in MRI findings throughout the treatment course might mirror the response to therapeutic interventions, including immunotherapy. Diminished lesion activity, or resolution evident on MRI may coincide with amelioration in clinical symptoms, whereas sustained or deteriorating MRI findings may signify resistance to treatment or progression of the disease.[15]

In contrast to NMOSD and multiple sclerosis, complete resolution of lesions is significantly observed in most reported cases, with possible relapse in some patients, probably related to higher antibody titer and longer duration of positivity of MOG antibodies.[31]

Also, long use of oral anti-epileptic (AED) has not shown any effect on seizures.[32] But researchers are yet to study the effect of AEDs in long-term treatment on recurrence, duration, and frequency of seizures.

Conclusion

Every sudden-onset, persistent headache or seizure should raise suspicions of a range of differential diagnoses including FLAMES. The typical clinicoradiological presentation is highly suggestive of this unique entity and prompt therapeutical management is required to improve the patient's prognosis.

Management of FLAMES syndrome requires a multifaceted approach tailored to individual patient needs. Since FLAMES syndrome is a complex and relatively newly recognized disorder, treatment strategies continue to evolve.

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Author contributions

I.E.O. Conception of the work, design of the work, and acquisition of data; A.N. Acquisition of data; K.B. Acquisition of data; M.J. Revising the work critically for important intellectual content; F.T. Revising the work critically for important intellectual content and final approval of the version to be published.

Data availability

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

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ORCID iDs

Ibtissam El Ouali https://orcid.org/0000-0001-6501-6888
Amine Naggar https://orcid.org/0000-0003-4841-0423
Kenza Berrada https://orcid.org/0000-0002-2322-618X

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