mainly the optic nerves and spinal cord. Here we reported two atypical cases of MOGAD.

Methods

Case report

Results

We present two patients a male (P1) and a female (P2) aged respectively 25 and 33 years-old. They developed a sub-acute onset headache, along with vomiting. P1 had no medical history, whereas, P2 had a history of ankylosing spondylitis, endometriosis, and V neuralgia. On examination, P1 had a bilateral decreased visual acuity and a bilateral stage II papilledema, however, P2 showed a static and kinetic cerebellar syndrome and a left pyramidal syndrome. Both had an elevated CSF opening pressure. Brain and spine MRI was normal in both cases. The etiological investigation was negative except for anti-MOG antibodies. The diagnosis of intracranial hypertension related to MOGAD was confirmed. Both patients were treated with high-dose corticosteroids and acetazolamide. At follow-up, P1 showed a significative improvement, while, P2 reported an initial improvement of headache then recrudescence of the same symptomatology. Eventually, she had plasmapheresis and steroids were associated to azathioprine with good outcome.

Conclusions

MOGAD is often an underdiagnosed disease characterized by different clinical presentations. Clinicians should consider the diagnosis of MOGAD when patients develop intracranial hypertension.

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Unlikely outcome? A case of acute ischemic stroke in a multiple sclerosis patient on teriflunomide

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Background and aims

Multiple Sclerosis (MS) is a chronic inflammatory disease that affects the central nervous system and can lead to axonal dysfunction. Vascular dysfunction has been suggested as a contributing factor in MS, although the mechanisms are not fully understood. Teriflunomide is an oral medication used to treat Multiple Sclerosis (MS), but there have been reports of rare complications such as thrombosis and vascular accidents.

Methods

Here, we present a case of a middle-aged woman with RRMS who developed an acute ischemic stroke shortly after starting treatment with Teriflunomide.

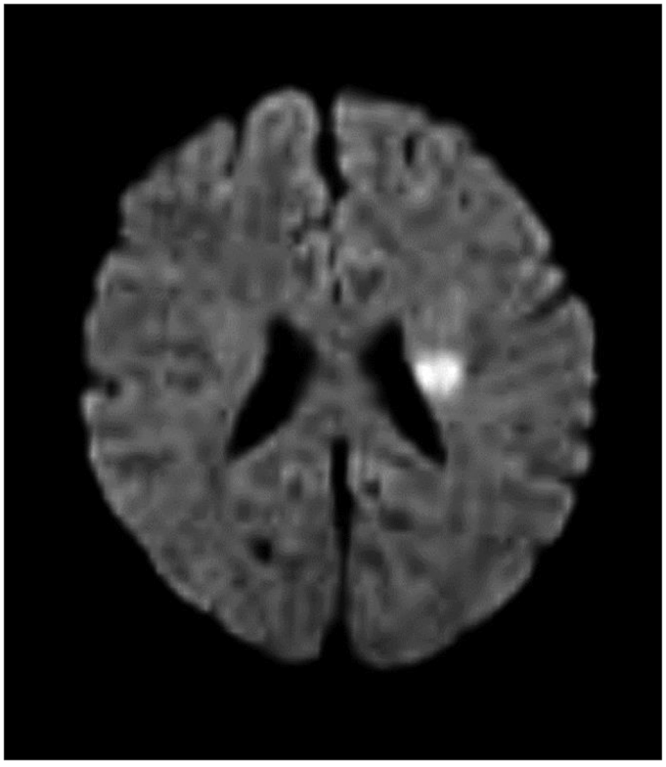
Results

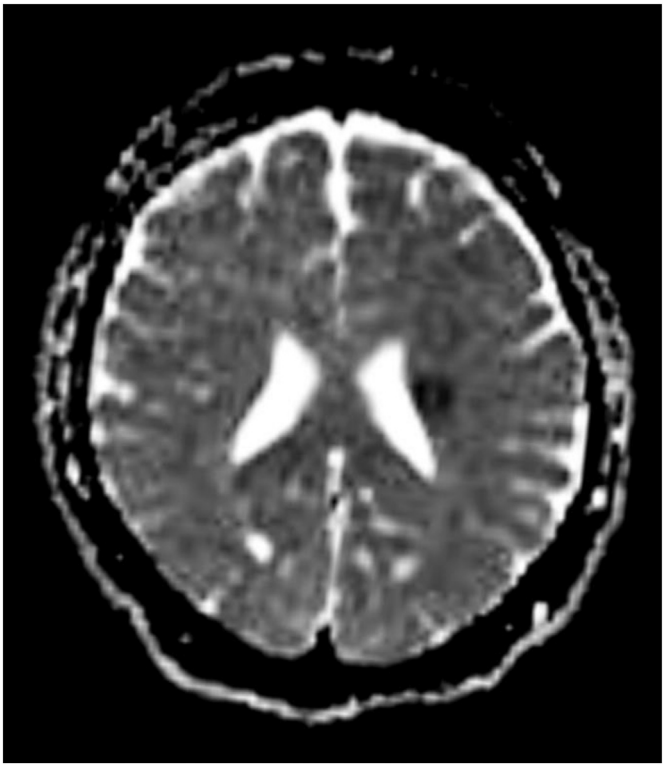
The patient was a 48-year-old woman with RRMS who had been on interferon beta 1a for six years without relapse. Due to side effects, her therapy was changed to 14 mg of Teriflunomide daily. After 35 days, the patient presented to the hospital with sudden right limb weakness. MRI revealed a lacunar infarction restricted to the left basal ganglia.

Further testing ruled out other potential causes of stroke, and the only significant change to her treatment regimen was the addition of Teriflunomide. The medication was discontinued, and the patient was switched to Ocrelizumab.

Conclusions

While rare, thrombotic complications have been reported in patients with MS treated with Teriflunomide. This case highlights the





importance of monitoring patients closely for potential side effects and considering alternative treatments if necessary. Further research is needed to fully understand the mechanisms of vascular dysfunction in MS and the role of Teriflunomide in contributing to these complications.

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Clinicopathological findings of patient with MOG antibodies detected only in cerebrospinal fluid

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Background and aims

Anti-myelin oligodendrocyte glycoprotein antibodies (MOG-Abs) are generally produced in blood, though only detected in cerebro spinal fluid (CSF) in some cases. Thus, the relationship of CSF MOG-Abs with disease pathology is unclear.

Methods

Here, we report clinicopathological findings of a patient with increased MOG-Abs in only the CSF.

Results

A 31-year-old female was presented with T10 level sensory loss and urinary retention. Spinal cord MRI findings showed long T2 hyperintense lesions from the medulla oblongata to the T2 level and from T8 to 12. Brain FLAIR MRI revealed multiple high intensity lesions in the periventricular area, corpus callosum, basal ganglia, and brainstem. CSF analysis indicated increased lymphocytic pleocytosis, protein, IgG index, myelin basic protein, and oligoclonal bands. On autoimmune screening, MOG-Abs only in the CSF were detected with a live cell-based assay (titer 1:1024). The patient rapidly developed tetraplegia, a comatose state, and respiratory failure requiring a respirator. Despite treatments with steroids, intravenous immunoglobulin, and plasma exchange, death due to sepsis occurred six months after onset. A pathological microscopic examination revealed demyelination, macrophage infiltration, and gliosis in the cerebrum (corpus callosum, white matter, periventricular, etc.), brain stem, and spinal cord, with activated complement deposition and CD4-T-cell infiltration around intrameningeal vessels also recognized. Notably, extensive necrosis from the medulla oblongata to cervical spinal cord indicated poor prognosis and marked demyelination adjacent to the CSF cavity suggested a relationship with sustained intrathecal antibody production.

Conclusions

These findings indicate a pathogenic role of CSF MOG-Abs and need for measurement in highly probable cases.

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Mitochondrial MS mimickers: A literature review and a novel form associated to variants in the TUFM gene

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Background and aims

Due to their genetic and clinical heterogeneity, diagnosing mitochondrial disorders (MD) is often challenging. Specifically, when presenting with multifocal white matter abnormalities and acute neurological deterioration, MD may mimic multiple sclerosis (MS). We aim to provide an overview of MD mimicking MS and describe a novel form caused by TUFM variants.

Methods

We conducted a literature review using Ovid and PubMed databases to find articles describing genetic, clinical and/or neuroimaging findings from 1990 to 2023. We also retrospectively analysed the clinical and radiological data of a 37-year-old patient with TUFM variants who presented with MS-like white matter abnormalities.

Results

We identified 10 MD caused by variants in 28 genes presenting with white matter abnormalities mimicking MS. 19 of them are not included in the next generation sequencing panels for genetic white matter disorders. Our index case had a recent onset of ataxia and balance problems in the context of multifocal white matter lesions resembling MS. The whole exome sequencing revealed two pathogenic heterozygous TUFM variants. The diagnosis of mitochondrial disorder was supported by the presence of multisystemic involvement.

Conclusions

MD are a relatively frequent cause of MS mimics, and their growing list emphasizes the need to integrate clinical, genetic, and imaging findings for the differentiation of these conditions. To increase the rate of diagnosis, the genes associated with mitochondrial MS mimickers should be included in clinical panels for genetic leukoencephalopathies. Additionally, our case description expands on the list of mitochondrial MS mimickers by including TUFM-related disorders.

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Mangement of NMOSD and MOGAD in sub-Saharan Africa

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Background and aims

Background: Neuromyelitis Optica spectrum disorders (NMOSD) are a group of inflam- matory disorders of the CNS, characterized by severe attacks of optic neuritis and myelitis. Aims: To focus on the mangement Aspect of NMOSD& MOGAD in Sub-Saharan Africa