Autoimmune encephalitis associated with MOG antibodies

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Background

MOG antibody-associated disease (MOGAD) describes a group of inflammatory disorders, where the immune system produces antibodies against myelin oligodendrocyte glycoprotein (MOG), a protein found on the outer surface of myelin sheaths in the central nervous system (CNS).

The clinical manifestations of MOGAD vary from optic neuritis or myelitis to forms of encephalitis which differ slightly from each other. These include acute disseminated encephalomyelitis (ADEM), FLAIR-hyperintense Lesions in Anti-MOG-associated Encephalitis with Seizures (FLAMES), or cerebral cortical encephalitis (CCE).

What are the symptoms?

The most common symptoms are headaches, seizures, fever, altered mental status (with confusion, behavioural changes, memory problems), speech difficulties, weakness or paralysis, nausea and vomiting, and, in severe cases, coma.

Often, these occur shortly after a transient infection such as a cold or gastroenteritis, and occasionally after vaccination.

Symptoms can develop rapidly, typically over just a few days to weeks.

What are the causes?

MOG-antibodies are likely causative. However, this conclusion awaits formal experimental proof. Yet, the reasons why the immune system raises a response to MOG, a self-protein, likely underlie the mechanisms of causation. These may include immune genes and environmental triggers which activate the immune system.

How is it diagnosed?

Diagnosis is usually confirmed by detecting MOG antibodies in the blood (serum), or sometimes, in cerebrospinal fluid (CSF).

The brain magnetic resonance imaging (MRI) can also be very helpful to identify characteristic lesions and patterns.

How is it treated?

MOGAD is treated with high-dose corticosteroids, often methylprednisolone as a first-line therapy to reduce inflammation.

If steroids are insufficient, second-line treatments include plasma exchange to remove circulating antibodies and intravenous immunoglobulin (IVIG) to modulate the immune system.

Treatment with additional immunosuppressive medications, such as mycophenolate, tocilizumab, rituximab and cyclophosphamide may be associated with reduced relapse rates – further evidence is required to understand this better.

Supportive care includes seizure management with anticonvulsants as well as intensive care admission for raised intracranial pressures. After hospital, Rehabilitation services are often needed including physical and speech therapy.

What are the outcomes?

Compared to other types of autoimmune encephalitis, most people affected have a favourable outlook. Acute symptoms typically begin improving within days to weeks of initiating treatment.

Due to the possibility of relapse, long-term monitoring is important. However, about ~50% of those with MOGAD only experience one episode of symptoms. Some studies have shown that individuals who continue to test positive for the MOG antibody appear to have a higher chance of relapse. The occurrence of relapse can involve the development of a new neurological symptom (typically affecting the eye (causing optic neuritis) or spinal cord (transverse myelitis)) or worsening of old symptom.

In children, complete recovery from the initial onset of symptoms is common. Children who present with ADEM appear more likely to experience MOGAD only once.

Overall, this condition is a rare but treatable condition that requires prompt recognition and intervention.

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