Medical Xpress

Researchers make strides in understanding little-known autoimmune myelin-impairing disorder

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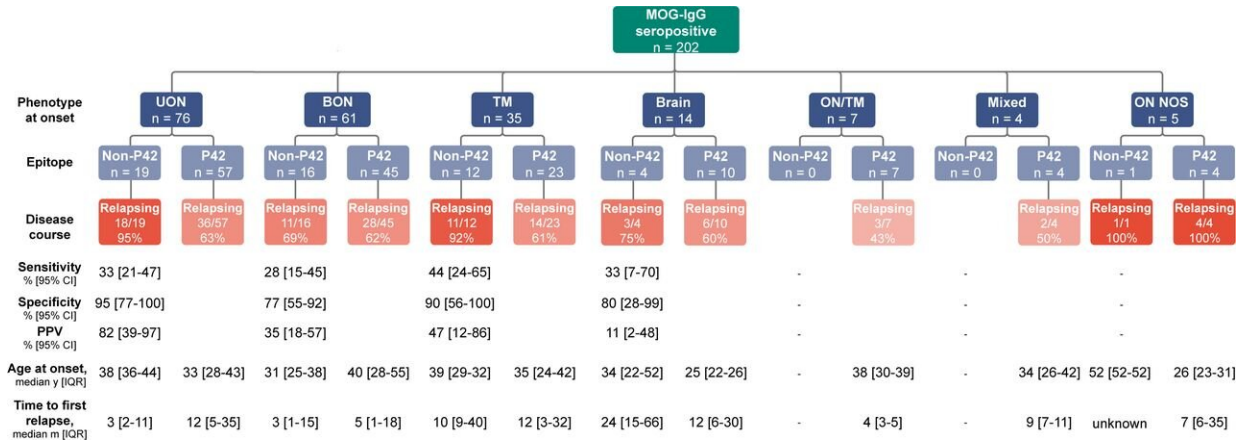

Distribution of non-P42 MOG-IgG in myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) at onset. Credit: *Journal of Neurology, Neurosurgery & Psychiatry* (2024). DOI: 10.1136/jnnp-2023-332851

Blindness and paralysis are often the devastating consequences of little-known disease myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD). An Australian research collaboration is looking to change this, making huge strides in understanding the condition which could lead to better outcomes in the future.

Often referred to as a cousin of MS due to shared symptoms, MOGAD

is an autoimmune condition where the body attacks a protein in the brain, resulting in a swollen central nervous system. The disease affects adults and children but is much less common, and less understood than MS.

One of the biggest challenges with the condition is that some people will experience only one attack in their lifetime while around 40% have recurring attacks, each leading to additional impairment and disability. Strong immunosuppressants can help prevent relapses but drug side effects mean it is vital to identify which patients need this ongoing treatment.

A team of over 50 Australian scientists, headed by Professor Fabienne Brilot from the University of Sydney and the Kids Neuroscience Center at Sydney Children's Hospitals Network have been able to achieve just that.

In a new study published in the *Journal of Neurology, Neurosurgery & Psychiatry*, the researchers detail the discovery of a way to predict which adult patients will experience a relapsing course by examining where the antibody binds on the MOG protein (the epitope) in patients who have tested positive for the MOG antibody.

They found that in around 25% of patients, the antibody was not binding at the dominant epitope (called non-p42 epitope) and these were the patients experiencing a relapse. They also found this was most common in patients who at the initial onset of the disease were affected by vision impairment (lesions on the optic nerve) as opposed to those experiencing issues with movement (lesions on the spinal cord).

Professor Brilot said the clinical translation of the finding is hugely significant given this is the first and only 'test' available.

"The prediction of a relapsing course in MOGAD means neurologists can make more informed treatment decisions. They can initiate maintenance immunosuppression early rather than waiting for relapse to occur, as has happened with multiple sclerosis."

"It will help prioritize patients in busy neuroimmunology clinics and will also help recruitment into clinical trials which are vital to undercover the best treatments for this disease."

The discovery builds on Professor Brilot's previous work in the field, including as a key member of the global consortium that published the first consensus on diagnostic criteria for MOGAD in 2023.

The disease is diagnosed by the detection of the MOG autoantibody. Professor Brilot's lab at Westmead has been the Australia-wide testing point for the antibody for the last 10 years, testing serum or blood of 4000 Australian patients a year.

"The disease is so new, and so newly described that there is a lot we still don't know. But whether or not patients will relapse is the number one question asked of neurologists. One we can now potentially answer," she said.

Based on her experience overseeing MOGAD antibody testing, Professor Brilot aims to have testing for early identification of those at risk of relapsing disease available for clinical purposes in adults soon.

Simultaneously her team is carrying out the same research with children with MOGAD, in the hope that the discovery could also help young people impacted by the disease.

More information: Ganesha Liyanage et al, The MOG antibody non-P42 epitope is predictive of a relapsing course in MOG antibody-

associated disease, *Journal of Neurology, Neurosurgery & Psychiatry* (2024). DOI: 10.1136/jnnp-2023-332851