

SIMPLIFIED ABSTRACT SAMPLE WORK

**Autoimmune nodopathy with anti-NF186 antibodies
following SARS-CoV-2 infection: A case report**

Autoimmune nodopathy with anti-NF186 antibodies following SARS-CoV-2 infection: A case report

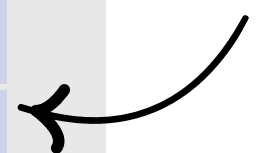
Lai, J., Zhang, X., Wang, Y., Cheng, Y., Wang, H., & Zhu, W. (2025)

Background

Autoimmune nodopathy is a rare disease in which autoantibodies target proteins critical for nerve conduction, such as neurofascin-186 (NF186), leading to neurological dysfunction. This report describes an unusual case of autoimmune nodopathy triggered by SARS-CoV-2 infection.

specific background

Autoimmune monopathy is a rare disorder of the nervous system that occurs when antibodies are produced that target nodal paranodal proteins (like neurofascin-186 (NF186)), thereby creating conduction defects. This report describes an unusual case of autoimmune nonopathy triggered by infection with SARS-CoV-2.

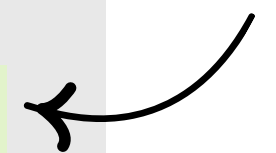


knowledge gap

While autoimmune monopathies have been recognized, the association with viral infections, especially COVID-19, remains to be more established; resulting in limited understanding and treatment protocols for neurological complications delivered at post-COVID.

Here We Show

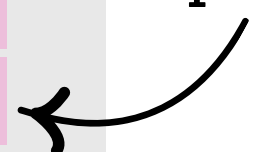
This report is the first with evidence of autoimmune neuropathy with anti-NF186 antibodies associated to SARS-CoV-2 infection, as it relates to potential virally mediated autoimmune neurological disorders.



The clinical examination involved a thorough assessment of neurological function. Electromyography (EMG) was utilized to evaluate conduction defects. Cell-based assays were performed to confirm the presence of anti-NF186 antibodies. Treatment included the use of intravenous immunoglobulin, steroids, plasma exchange, and rituximab.

Results & Interpretation

The patient presented with severe neurological symptoms, with progression to respiratory muscle paralysis. Initiating therapy resulted in minimal improvement; however, the use of plasma exchange led to a partial recovery and full stability once rituximab was instituted. The onset of SARS-CoV-2 did not contribute to worsening symptoms, and muscle strength gradually improved over the treatment course.



This case report reinforces the importance of characterizing autoimmune neuropathies in the post-COVID neurological literature. This case also reinforces the importance of testing for anti-NF186 antibodies in patients with patterns typical of autoimmune mononeuropathy in the post-COVID world. Plasma exchange or therapies that induce B-cell depletion such as rituximab, tend to demonstrate better outcomes than conventional management approaches for autoimmune mononeuropathies associated with viral infections.

Methodological

Implications