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50 Years Ago in *THE JOURNAL OF PEDIATRICS*

Changes in Miller Fisher Landscape

Qaqundah BY, Taylor WF. Miller Fisher Syndrome in a 22-Month-Old Child. J Pediatr 1970;77:868-70.

Miller Fisher syndrome was first characterized in 1956 by its classical triad of ataxia, ophthalmoplegia, and areflexia. We now describe Miller Fisher syndrome as a spectrum of symptoms manifesting as isolated ophthalmoplegia or ataxia, or combined ascending limb weakness, ptosis, mydriasis, or altered mentation (owing to overlap with Bickerstaff brainstem encephalitis). Miller Fisher syndrome can mimic a myriad of pediatric conditions, including acute cerebellar ataxia, infectious or postviral encephalitis, heavy metal poisoning, botulism, and myasthenia gravis. This 22-month-old girl presented with ataxia, nystagmus, and subsequent change in mentation. Cerebrospinal fluid was initially normal, and Miller Fisher syndrome was only recognized after a subsequent spinal tap revealed elevated protein with persistently normal cell count (cytoalbuminologic dissociation) in association with development of areflexia and ophthalmoplegia.

Anti-GQ1b antibodies were subsequently shown to be a more sensitive indicator of Miller Fisher syndrome than cytoalbuminologic dissociation. Cerebrospinal fluid protein slowly increases over 3 weeks, but anti-GQ1b is found in 81% of patients within the first week of symptoms.[1] This antibody targets ganglioside epitopes concentrated in cranial nerves III, IV, and VI, and the dorsal root ganglia, corresponding with Miller Fisher syndrome symptoms.[2] Anti-GQ1b is particular toward Miller Fisher syndrome and Miller Fisher syndrome-like variants, less in classic Guillain-Barre syndrome.

The patient fully recovered at 6 months after a course of prednisone in 1970, but Miller Fisher syndrome is now considered a self-limited condition. The majority of patients show full recovery at 5 months without treatment, and steroids do not shorten the course.[3] Intravenous immunoglobulin or plasmapheresis can be considered in severe Miller Fisher syndrome, but demonstrate limited benefit in outcome, with full recovery at 6 months in 89% and 66%-96% of patients, respectively.

Over the past 50 years, a clearer picture of the Miller Fisher syndrome spectrum has evolved, with a better serologic marker for diagnosis and improved treatment options.

Ava Yun Lin, MD, PhD
Stanford Neuromuscular Program
Stanford University
Palo Alto, California

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