

## The Miller-Fisher Variant of Guillain-Barré Syndrome

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# The Miller-Fisher Variant of Guillain-Barré Syndrome

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## INTRODUCTION

Guillain-Barré syndrome (GBS) is a serious and possibly fatal acute immune-mediated polyneuropathy that typically presents with ascending muscle paralysis and can progress to respiratory failure and death. Miller Fisher Syndrome was named after neurologist Dr. C. Miller Fisher who first described it in 1956 as a limited variant of ascending paralysis, GBS. Miller Fisher syndrome (MFS) is a rare variant of GBS that is observed in only about 1% to 5% of all cases of GBS worldwide. The syndrome begins with the rapid development, over days, of weak eye muscles, with double or blurred vision, and often drooping eyelids with facial weakness; poor balance and coordination with sloppy or clumsy walking; and loss of deep tendon reflexes – the triad of ophthalmoplegia, ataxia, and areflexia.

## CASE DESCRIPTION

Here we present a case of MFS in a young female. A 30-year-old right handed Caucasian female with type 2 diabetes, rheumatoid arthritis, hypothyroidism, and recent influenza vaccination presented with 1 week of double vision and extremity numbness followed by 1 day of dysarthria, dysphagia, and orthopnea. Physical exam demonstrated absent upward, downward, and lateral gaze; hyporeflexia; decreased extremity sensation; and ataxic gait. Labs demonstrated ESR at 65. Lumbar puncture revealed a normal opening pressure with an albuminocytologic dissociation. Nerve conduction study in the upper and lower extremities demonstrated normal motor F-waves with decreased sensory H-reflexes.

## RESULTS

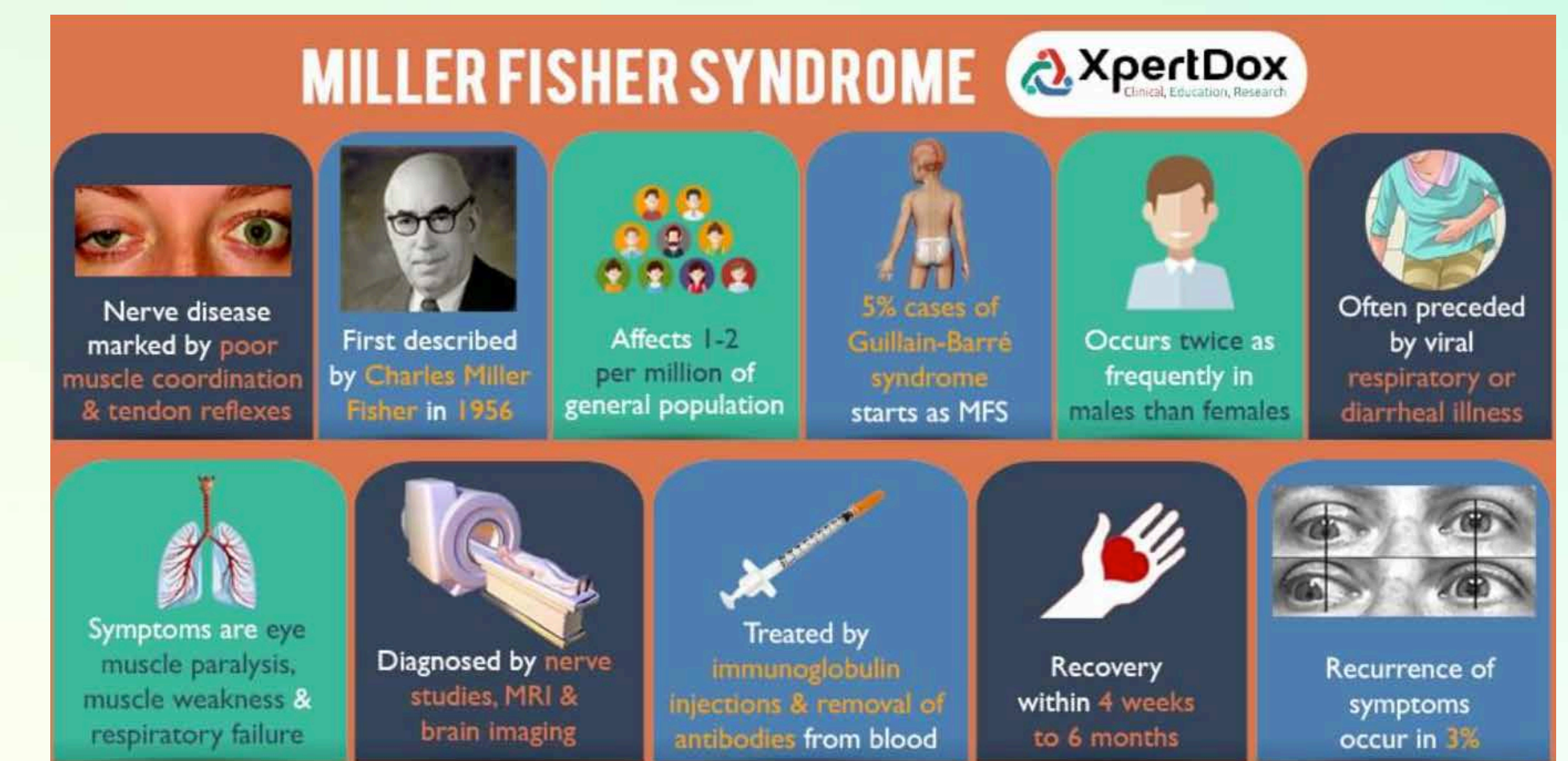
Anti-ganglioside antibody panel demonstrated increased levels of anti-GQ1b antibodies. The patient was subsequently started on intravenous immunoglobulin with improvement after 5 days.

## DISCUSSION

Although the cause is not known, MFS is thought to be an autoimmune process in which a preceding infection stimulates production of an antibody that reacts to a glycolipid found on neuronal membranes, causing demyelination and loss of function of the peripheral nerves. In most people with Miller Fisher Syndrome (96%) an antibody called anti-GQ1b is identified. This is especially important as the GQ1b antigen is located on the Schwann cells of the ocular cranial nerves. The presence of these autoantibodies confirms the diagnosis of the syndrome.

## CONCLUSION

The Miller Fisher variant occurs in less than 5% of GBS cases. Treatment, just as typical GBS, consists of intravenous immunoglobulin or plasmapheresis. IVIG results in an influx of new antibodies which competitively bind to the antigen site, thereby reducing the ability of harmful autoantibodies to bind; whereas, plasmapheresis removes the antibodies from the blood entirely. In most cases, there is almost complete recovery within 6 months. In rare cases, the syndrome may progress and permanent neurological deficits may be present.



<https://www.xpertdox.com/disease-description/Miller%20Fisher%20Syndrome>



RIGHT GAZE

STRAIGHT AHEAD GAZE

LEFT GAZE

<https://pn.bmj.com/content/9/5/289>

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