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Case Report

Understanding the complexity of Miller-Fisher syndrome: a case report

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ABSTRACT

Miller-Fisher syndrome is a rare neurological disorder characterized by a clinical trial of ophthalmoplegia, ataxia and areflexia. It is a variant of Gullian Barrie syndrome. A variety of other symptoms and signs beyond the triad has been reported. Here we report a case of a 58-year-old male patient with atypical presentation of Miller-Fisher syndrome. He had a history of upper respiratory tract infection 3 weeks prior. After hospital admission, he abruptly developed unsteadiness while walking, diplopia and dysphagia. Clinical evaluation along with antiganglioside antibody made the diagnosis of Miller-Fisher syndrome. After immunotherapy was initiated, the patient improved gradually and later discharged to home. To diagnose a case of Miller-Fisher syndrome, a high index of clinical suspicion and ruling out other aetiologies should be made. Timely effective intervention either plasmapheresis or IV Immunoglobulin can completely reverse the symptoms.

Keywords: Antiganglioside antibody, Immunotherapy, Miller-Fisher syndrome

INTRODUCTION

Guillain-Barre syndrome (GBS) is an autoimmune disorder of the peripheral nervous system characterized by weakness, usually symmetrical and evolving over several days or more.[1] After the possible elimination of poliomyelitis, GBS has become the most common cause of symmetrical limb weakness.

The Miller-Fisher syndrome subtype has been seen to have rare descending paralysis (not the usual ascending paralysis) and is especially associated with antibodies against ganglioside GQ1b and has similar cross-reactivity with ganglioside structures in the wall of *Campylobacter jejuni*.[2] Miller-Fisher syndrome (MFS) is characterized by weakness of eye muscles (ophthalmoplegia), loss of deep tendon reflexes (areflexia) and lack of muscle coordination (ataxia).[3] MFS is commonly associated with the involvement of the lower cranial and facial nerves and does not usually involve motor weakness of limbs. However, Miller-Fisher variants including weakness of the respiratory system and limbs have been described. Another variant of MFS is Bickerstaff brainstem encephalitis (BBE) which involves altered consciousness, ataxia, ophthalmoparesis and paradoxical hyperreflexia. The antibody to ganglioside GQ1b is well known as a biomarker of MFS.[4]

The GQ1b epitope is strongly expressed in ocular motor nerves, dorsal root ganglion neurons and muscle spindles. MFS is due to an aberrant acute autoimmune response to a preceding infection (e.g., *Campylobacter jejuni*, Cytomegalovirus, Epstein-Barr virus or human immunodeficiency virus (HIV).

A cross-reaction between peripheral nerve antigens and microbial/viral components through molecular mimicry is thought to drive the inflammatory process of this illness.[5,6]

CASE REPORT

A 58-year-old male patient with a known case of systemic hypertension came with a history of fever for 5 days duration, 3 weeks back, which was associated with rhinitis and cough. He took oral antibiotics from a nearby hospital and his fever and rhinitis resolved but the cough persisted. After 2 weeks of first symptoms, he developed a headache and back pain of 2 days duration along with a cough. Then he came to our hospital for further management. On evaluation, his vitals were stable. On clinical examination, he had features of right lower lobe consolidation and elevated total count with neutrophil predominance. Other system examinations were within normal limits.

Hence, he was admitted. On the next day, he developed swaying while walking to either side and an intentional tremor. On neurological examination, he had ataxic gait, intention tremor and, sluggish deep tendon reflex with normal motor power and cranial nerves. A provisional diagnosis of cerebellar stroke was made and taken up for an MRI brain. However, the MRI brain done was normal.

Later he developed slurring of speech, dysphagia and ptosis of the right eye. On evaluation, he had normal horizontal gaze, gaze-evoked nystagmus, mydriasis of both eyes, absent gag reflex and other signs of bulbar palsy, areflexia and cerebellar symptoms. On the background of a recent respiratory infection, a provisional diagnosis of immune-mediated CNS disorder was made.

He was taken up for NCS study, Lumbar puncture was done and serum anti ganglioside antibody was sent. To rule out other etiologies, Serum and CSF Autoimmune panel and Paraneoplastic workup were done. NCS showed mildly prolonged distal latencies from left median CMAPs and prolonged peak latencies from left median SNAPs. Bilateral tibial F waves showed borderline latencies. LP CSF study was normal including bio fire and viral panel. In terms of clinical background, a diagnosis of atypical presentation of Miller-Fischer syndrome was made. The need for immunotherapy explained to bystanders. They opted for plasmapheresis. After 2 sessions of plasmapheresis, he improved gradually. By that time, his serum anti-ganglioside antibody reports came back positive for anti-GM1 and GQ1b antibodies.

He underwent 5 sessions of plasmapheresis. His symptoms improved gradually. Laryngology evaluation was done for swallowing and showed adequate response. After 2 weeks of plasmapheresis, he became symptomatically better; he was able to walk without any support and was able to take feeds orally. Then, he was discharged to home and kept under follow-up.

DISCUSSION

Miller Fischer syndrome is a rare, acquired nerve disease, a variant of Gullian Barrie syndrome. It is an immune-mediated neuropathy, acute in onset characterized by the classic triad of ataxia, areflexia and opthalmoparesis. Its incidence is higher in the Asian population, especially in male gender. Most of the time it will not present with a typical triad of symptoms. On the background of a recent viral/bacterial infection, if a patient presented with neurological features, GBS is one of our differential diagnoses. In addition to clinical features, radiological imaging, nerve conduction study and Lumbar puncture will give a pathway to final diagnosis. We can use all these investigation tools to exclude other diagnoses.

Nearly 90% of the patients with MFS had a positive test for the IgG anti-GQ1b antibody in their sera.[7,8] Treatment of MFS is mainly immunotherapy- either plasmapheresis or IV immunoglobulin therapy. During plasmapheresis, extraction of all the antibodies that attack Schwann cells and cause peripheral demyelination. It is time-consuming. The other option is IV immunoglobulin therapy. These immunoglobulins competitively inhibit the autoreactive antibodies from destroying the schwann cells.

Other complications of MFS include respiratory paralysis and lower cranial nerve involvement. If respiratory paralysis develops due to phrenic nerve involvement, ICU admission and ventilator support are required. Full recovery usually takes 2-4 weeks and relapses are rarely reported clinically. Our patient had features of cerebellar symptoms, areflexia and lower cranial nerve palsy. Timely diagnosis and early therapy prevented further progression of the disease and gave better outcomes.

CONCLUSION

Miller-Fischer syndrome is a neurological disorder which can present in unusual manner. Appropriate history of preceding viral infection followed by development of neurologic symptoms should raise the suspicion of the disease. Early therapy and multidisciplinary approach in the management will give better prognosis.

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