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Case Report

Miller Fisher Syndrome: a case report

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ABSTRACT

Miller Fisher Syndrome (MFS) is an uncommon variant of Guillain Barre syndrome (GBS). It is largely a clinical diagnosis based on the cardinal clinical features of ophthalmoplegia, ataxia and areflexia. The following case report is that of a patient who presented with an acute history of reeling of head, difficulty in opening the eyelids and swaying while walking, on clinical examination revealed the above-mentioned cardinal features and on investigation showed positive serum anti GQ1b antibody titer. The patient started recovering with conservative management and was discharged in the recovery phase.

Keywords: Areflexia, Anti GQ1b antibody, Ataxia, GBS, MFS, Ophthalmoplegia

INTRODUCTION

MFS is an uncommon variant of GBS. It involves both adults and children. It is largely a clinical diagnosis and this distinctive syndrome comprises ophthalmoplegia, ataxia and areflexia which all develop over a period of few days without significant limb weakness.[1-3]

The above triad was first described by James Collier in 1932 and it was subsequently reported as a variant of GBS by Charles Miller Fisher in three clinical cases in 1956.[4] MFS is a geographically variable variant of GBS observed in about 1% to 5% of all GBS cases in Western countries, yet up to 19% and 25% in Taiwan and Japan respectively.[5]

There is an established male predominance at a ratio of 2:1 and a mean age of 43.6 years, although cases of MFS have been reported in all age groups.[6-7] Preceding infection with Campylobacter jejuni of the HS2 or HS4 serotypes is usual.[1] Upper respiratory infection is the most commonly described prodromic entity followed by gastrointestinal illness.[5-6]

The most common presenting symptom of MFS is diplopia caused by external ophthalmoplegia.[5,7] The spinal fluid protein is elevated after the first week and patient recovers over a matter of weeks.[3] The serum of over 90% of patients contain antibodies against the GQ1b and GT1a gangliosides of both peripheral and central nervous system. Brighton criteria is used for the diagnosis of MFS. MFS is usually having a self-limiting course.[1,3] The treatment options are same as that of GBS, i.e. intravenous immunoglobulin and plasmapheresis but the overall impact of these treatment on eventual recovery is questioned.[1,3] The recovery period is marked by gradual improvement and resolution of symptoms, although rarely serious complications such as respiratory failure or cardiac arrhythmia have been reported.[6] Ataxia and ophthalmoplegia resolve in 1 to 3 months after onset and near complete recovery is expected over several months to a year.[5]

CASE REPORT

A 60-year-old female presented to casualty with a history of vomiting, reeling of head, difficulty in opening the

eyelids and swaying while walking since last four days. There was no history of fever, convulsion, loss of consciousness, slurring of speech, nasal regurgitation of fluids, urinary incontinence, head trauma or unknown bite. She had a history of upper respiratory infection seven days prior to the onset of recent symptoms for which she took some treatment and got relieved.

Figure 1: Total external ophthalmoplegia on both sides.

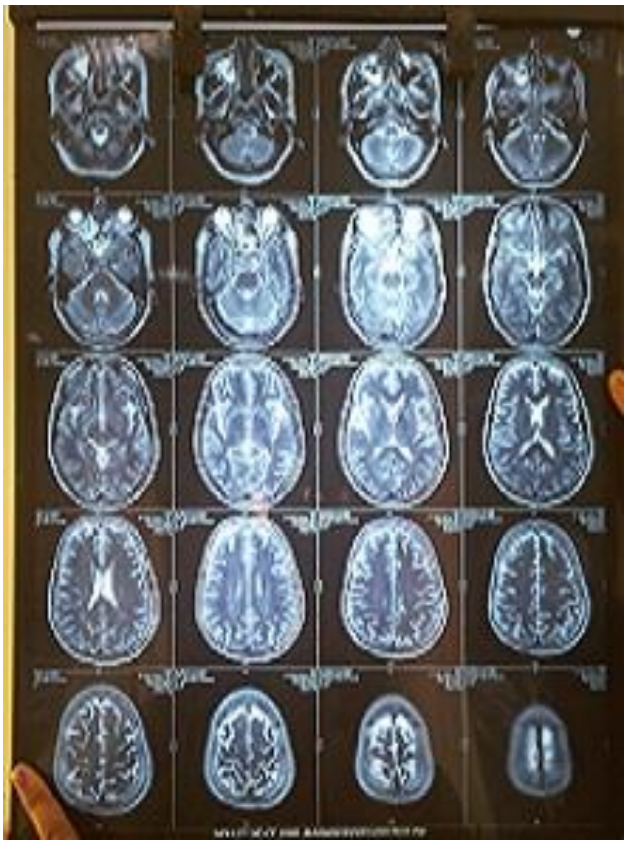

Figure 2: Normal MRI scan of brain.

On clinical examination, vitals were within normal limits and she was afebrile. On examination of the central nervous system there was total external ophthalmoplegia on both sides (Figure 1), the power of both upper and lower limbs was 5/5, deep tendon reflexes were absent and plantar reflexes were flexor on either side. There was ataxia and sensations were intact except the joint and position sense in the limbs. There were no signs of cerebellar involvement and signs of meningeal irritation were absent. Fundoscopy revealed no abnormality. She was admitted to the ICU with a provisional diagnosis of Acute idiopathic demyelinating polyneuropathy and the differential diagnosis of posterior circulation stroke, intracranial space occupying lesion and Myasthenia gravis were kept in mind.

On investigation, routine blood tests revealed no abnormality except for an elevated serum cholesterol level. MRI scan of brain was normal (Figure 2). Nerve conduction velocity study revealed evidences of sensory neuropathy in upper and lower limbs. Repetitive nerve stimulation study was within normal limits. CSF study done on the 9th day after the onset of symptoms revealed a protein of 85 mg/dl. and a cell count of 2 cells per Cu.mm. Hence a clinical diagnosis of MFS was made and the patient was managed conservatively with intravenous fluids and antibiotics in ICU. For diagnostic certainty serum was sent for serum anti GQ1b antibody titre which came out to be positive. On the 10th day of admission, ptosis started improving and there was no further clinical deterioration. On subsequent days, the patient was clinically improving and the patient was discharged on the 14th day of admission with an advice to follow up in outpatients department at frequent interval for further monitoring.

DISCUSSION

MFS is commonly seen in males but here we had dealt with a female case. In our case there was a prior history of upper respiratory tract infection, which is the most common prodromic entity described.[5-7] This case exactly satisfies the clinical triad of ophthalmoplegia, ataxia and areflexia without significant limb weakness.[1-3] In our case, serum anti GQ1b antibody was found positive, which is also seen in more than 90% cases.[1] CSF study showed albuminocytologic dissociation, which is usually seen 1 week after the onset of symptoms.[3] As reported in most of the studies, MFS usually follows a self-limiting course[1-3] and the same happened with our case and the patient started improving with conservative treatment only.

CONCLUSION

MFS is a rare variant of GBS and the familiarity with this rare syndrome will give a clue to the clinician to consider MFS as at least a differential diagnosis in a patient presenting with ataxia, areflexia and ophthalmoplegia.

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REFERENCES

1. Donaghy M. Polyneuropathy. Brain's Diseases of the Nervous System. 12th Edition; Chapter 21; 2009:563-568.
2. Yepishin IV, Allison RZ, Kaminskas DA, Zagorski NM, Liow KK. Miller Fisher syndrome: a case report highlighting heterogeneity of clinical features and focused differential diagnosis. Hawai J Med Pub Health. 2016;75(7):196-199.
3. Fokke C, Van Den Berg B, Drenthen, J, Walgaard C, Van Doorn PA, and Jacobs BC. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. Brain. 2014;137:33-43.
4. Fisher M. An unusual variant of acute idiopathic polyneuritis (syndrome of ophthalmoplegia, ataxia and areflexia) N Engl J Med. 1956;255:57-65.
5. Mori M, Kuwabara S, Fukutake T, Yuki N, Hattori T. Clinical features and prognosis of Miller Fisher syndrome. Neurology. 2001; 56(8):1104-6.
6. Lo YL. Clinical and immunological spectrum of the Miller Fisher syndrome. Muscle Nerve. 2007;36(5):615-27.
7. Snyder LA, Rismondo V, Miller NR. The Fisher variant of Guillain-Barré syndrome (Fisher syndrome). J Neuro-Ophthalmol. 2009;29(4):312-24.

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