Recurrent Miller Fisher Syndrome

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Abstract
Miller Fisher syndrome (MFS) is a variant of Guillan Barre syndrome characterized by the triad of ophthalmoplegia, ataxia and areflexia. Recurrences are exceptional with Miller Fisher syndrome. We are reporting a case with two episodes of MFS within two years. Initially he presented with partial ophthalmoplegia, ataxia. Second episode was characterized by full-blown presentation characterized by ataxia, areflexia and ophthalmoplegia. CSF analysis was typical during both episodes. Nerve conduction velocity study was fairly within normal limits. MRI of brain was within normal limits. He responded to symptomatic measures initially, then to steroids in the second episode. We are reporting the case due to its rarity.

INTRODUCTION
Miller Fisher syndrome (MFS) is usually a monophasic illness of sub acute onset, characterized by ataxia, areflexia and ophthalmoplegia, usually with no limb weakness. Even though the presentation is often alarming, it usually has a benign outcome with almost total recovery within a few weeks. MFS can be considered as a peripheral variant of AIDP, the Guillan Barre syndrome. AntiGQ1b antibody has been found to be strongly associated with MFS. Though recurrent GBS is a well known entity, recurrent MFS is exceptional, we report an unusual case of recurrent MFS.

CASE REPORT
A 33 year old male, goldsmith by profession, was admitted with history of vertigo and dizziness of 4 days duration. He was relatively asymptomatic prior to the episode. He was initially admitted in ENT department with provisional diagnosis of acute labyrinthitis and treated with labyrinthine sedatives. While on treatment he developed diplopia with vertical separation of images. Soon after he noticed imbalance while walking and developed tendency to fall on either side. He was bedridden. He had associated paresthesia of upper limb. There was no history of any lower limb paresthesia or weakness of any limbs. He gave no history of headache, seizures, bowel or bladder disturbances. There was no history of fever, gastrointestinal symptoms or recent history of vaccination or surgery. We reviewed the past history. He gave history of diplopia, gait imbalance two years back. At that time also he did not have any limb weakness or bowel or bladder symptoms. He was diagnosed to have a demyelinating disorder at that time. The symptoms were relieved within 12 days with symptomatic measures. There is no history of hypertension, diabetes, COPD etc. After reviewing the history clinical examination revealed; a moderately built and well nourished male. There was no pallor, cyanosis, jaundice, clubbing or lymphadenopathy. Blood pressure was normal with no orthostatic fall. Pulse rate was normal. There was no evidence of respiratory embarrassment. His mental functions were within normal limits. Cranial nerve examination revealed bilateral partial ptosis, bilateral total ophthalmoplegia with pupillary involvement in the form of dilated and fixed pupil. All other cranial nerves were within normal limits including optic fundus. Motor system showed normal bulk, tone and power. There was areflexia of all limbs. Plantar was flexor bilaterally. There was gait ataxia. There was downward and outward movement of the outstretched hands. There was no dysdiadochokinesis or cerebellar dysarthria. Sensory system was within normal limits. CVS, GIT, respiratory system were normal.

With the above signs, a LMN lesion of extra-axial lesion was considered, primarily due to demyelination. His routine blood, urine, serum electrolytes, ECG, chest, x-ray, RFT, LFT were normal. HIV status, other viral studies were negative. Possibility of sarcoidosis, Lyme’s disease, botulism were ruled out. CSF examination revealed an albuminocytological
dissociation. Initial nerve conduction study was within normal limits. MRI scan of brain revealed normal study. Diagnosis of Miller Fisher syndrome was made with the clinical criteria needed for the same, which included;

1. Bilateral, relative symmetric weakness of several extraocular muscles and ptosis.
2. Limb and gait ataxia with cerebellar tremor.
3. Areflexia of all limbs by 1 week.
4. Progression of aforementioned three features over several days to 3 months.
5. Minimal or no limb/facial or oropharyngeal weakness.
6. Normal alertness; no cerebellar dysarthria.
7. CSF s/o albuminocytological dissociation.

So previous history of diplopia, ataxia was considered as a fragment of the full blown MFS. Patient was put on i/v methylprednisolone, and symptomatically became better.

**DISCUSSION**

The initial symptom of MFS is usually diplopia with limb and gait ataxia appearing 3 or 4 days later, or at times concurrently with diplopia. Patients may complain of dizziness without vertigo. Normal or diminished reflexes persist for several days, but invariably these reflexes are absent in completely developed cases by the end of a week. There is a paucity of other symptoms, except paresthesia in approximately half the number of patients and mild proximal weakness in approximately a third. Ptosis occurs in most patients, but associated pupillary paralysis has been reported in minority MFS by itself is a rare entity, which is a variant of GBS. Recurrence rate of GBS is 3-6%. Recurrence of MFS is exceptional.1 Dewarrat et al reported one case of recurrent MFS. Among the Indian literature, a case of recurrent MFS was reported by Sitajayalakshmi et al. They reported that antiGQ1b antibodies were persistently present in these patients. We were unable to estimate the same due to unavailability of the test and cost factor. Among MFS patients 1/3rd may have respiratory embarrassment. Pupillary involvement, presence of vertigo are extremely rare in MFS, which were present in our patient. Numerous patients with MFS have been treated with plasma exchange, most with apparent improvement. The underlying mechanism of the disease and response to treatment are presumably same as for typical GBS, but this would be difficult to prove without a trial. It seems prudent to treat with plasma exchange patients who can no longer walk because of severe ataxia or superimposed weakness. Steroids have been used, but, as in typical GBS, their effect is difficult to judge. Alternating eye patching, corneal care and physical therapy to assist gait have been used. Our patient was put on intravenous methyl prednisolone 1 gram once daily for 3 days, he showed significant response to the same and was maintained on supportive therapy.

**REFERENCES**


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**Book Review**

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