

Unusual Clinical Features Of Miller-Fisher Syndrome: Case Report

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Citation

P SB, J VM, M VR. *Unusual Clinical Features Of Miller-Fisher Syndrome: Case Report*. The Internet Journal of Neurology. 2009 Volume 12 Number 2.

Abstract

Miller Fisher Syndrome is an uncommon clinical variant of Guillain-Barre syndrome and has rarer variants. The classic triad is ophthalmoplegia, ataxia and areflexia. But rarer clinical features have been described. Here we describe a case in which all ocular muscles were not involved simultaneously and pupils were involved right from the beginning. We also describe the clinical course of the disease.

INTRODUCTION

Miller Fisher syndrome is an uncommon variant of GBS (Guillain-Barre Syndrome) and few cases have been examined.¹ Usually the pupils are spared in MFS (Miller Fisher syndrome). Pupillary paralysis occurs mainly in advanced cases of GBS.² Here we describe a case of MFS which had pupillary involvement from the beginning. Also all the extra-ocular muscles were not involved simultaneously.

CASE REPORT

A 48 year old Hindu male patient, married, educated, belonging to middle socio economic class presented with complains of diplopia which occurred when looking to right or left, difficulty in vision and unsteadiness in walking since four days. These complains were progressive over four days. There were no complains of unconsciousness, convulsion, speech problems, hearing defects, vertigo, tinnitus, dysphagia or hoarseness of voice. There were no complains of motor weakness elsewhere in the body, sensory symptoms or bowel or bladder symptoms. He had a history of common cold 11 days prior to admission. There was no history of fever or vaccination and his personal and family histories were unremarkable.

The patient was vitally stable and his general examination was unremarkable. The patient had normal higher functions. The cranial nerve examination revealed decreased visual acuity in both eyes (Right eye 6/12 and Left eye 6/9 on Snellen's chart). The visual field and fundus both were normal. On examination of the ocular movements, it was

found that he had decreased movements present on lateral, medial and upward gaze. These were present on testing individual eye as well as both eyes together. However at the time of admission, there was normal movement on down gaze. The pupils were bilaterally equal, semi-dilated and not reacting to light or accommodation. There was no ptosis. Rests of the cranial nerves were normal to examination. The motor system examination was normal. Sensory examination was normal. There was absence of the deep tendon reflexes in both upper and lower limbs. The finger nose test and the knee heel test were normal. dysdiadochokinesia was absent. Romberg's sign was negative. The gait was ataxic and broad based. Other systems were normal at examination.

At admission, the complete blood counts (including platelet count), urea, creatinine and random sugar were all normal. A MRI scan of the brain was done which was also normal.

Based on the characteristic triad of ataxia, areflexia and ophthalmoparesis in the absence of any motor weakness anywhere else in the body, a clinical diagnosis of Miller Fischer syndrome was made. The patient was admitted to the hospital for observation of the clinical course.

By day 2 (two), the lateral, medial and upward movements of both eyes further decreased. The down movement was also now affected. By day 4 (four), the eye was immobile in upward and sideway gazes. Downward movement also progressively decreased from day 2 (two) but it was still present. By day 7 (seven), the eye was totally immobile in all directions. Areflexia and ataxia were still present. The pupils were still paralyzed. By day 8 (eight), there was some

regain of eye movement. At this stage, down movement was absent. Upward and lateral movements started to reappear. Reflexes also reappeared but were hypoactive. However, ataxia was still present. The patient requested a neurologist opinion and visited a neurologist at another city who gave the same clinical impression on 8th (eighth) day of hospital admission.

Although the patient had started to improve by day 8 (eight) without any specific treatment, he was started on oral prednisolone 40 mg/day according to neurologist's advice. It was tapered to 20 mg/day after 7 days. It was continued at 20 mg/day for 15 more days and then omitted.

At 6 (six) weeks follow up (from the time of admission) the patient was neurologically normal with complete recovery.

COMMENTS

MFS is a rare variant of GBS and few cases have been studied in detail.¹ The condition is characterized by a classic triad of ataxia, areflexia and ophthalmoplegia. The clinical features of MFS were reviewed by Mori et al who found that in addition to the classic triad, papillary abnormalities, ptosis and facial palsy were also seen.³ A mean recovery period of 10.1 weeks has been reported.⁴ Various variants of MFS have also been reported in the literature including cases with only ophthalmoparesis.

This case highlights the following:

Muscles for upward, downward and side-to-side gaze may not be involved simultaneously in all cases. Muscles for downward movement were the last to be affected in this case. (And also in a few other cases reported)

Ptosis may be absent in spite of involvement of third cranial nerve.

Although the disease characteristically causes external ophthalmoplegia, internal ophthalmoplegia (as evident by loss of pupillary reflexes) may also be present.

LIMITATIONS

EMG-NCV study and anti GQ1b antibody test could not be done in our patient because of the non availability of these tests at our institute.

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