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Case Report for Miller Fischer Syndrome

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Abstract

Miller Fisher Syndrome is an acute monophasic polyneuropathy characterized by the clinical triad of ataxia, areflexia and ophthalmoplegia, with diplopia (due to ophthalmoplegia) usually being the first clinical presentation, followed by appearance of gait disturbances. It is generally seen more in males than females, with incidence increasing linearly with age, mean age being 43.5 years. We present a case of a 60 year old lady complaints of drooping eyelid, unsteady gait, generalized weakness and dizziness. On examination, she had ophthalmoplegia grade 2 palsy, ataxic gait, impaired left finger nose finger test. Anti-ganglioside antibodies were negative. This case report highlights the importance of keeping MFS as a differential in seronegative patients not presenting with the classical triad.

Introduction

Guillain Barre syndrome or Landry's paralysis is described as an acute onset auto-immune inflammatory condition involving the peripheral nerves. Under the "umbrella of GBS"', various subtypes exist depending upon involvement of motor, sensory, cranial and autonomic nerves (AMAN, AMASN). One such rare focal form is Miller Fisher syndrome (MFS) [1].

The MFS variant comprises a very small portion of an already uncommon condition, representing just 1-2 in 1,000,000 cases of GBS. Nonetheless, it forms a larger percentage of cases in the Asian population i.e 25% in contrast to just 5% in western population [2].

MFS is an acute monophasic polyneuropathy characterized by the clinical triad of ataxia, areflexia and ophthalmoplegia, with diplopia (due to ophthalmoplegia) usually being the first clinical presentation, followed by appearance of gait disturbances [3].

It is generally seen more in males than females, with

incidence increasing linearly with age, mean age being 43.5 years [4].

The mechanism of injury to nervous tissue in Miller Fisher Syndrome is still poorly understood, especially in cases where no antibodies are detected in the patient's sera. The most accepted theory states production of antibodies as a consequence of molecular mimicry due to an antecedent infection of gastrointestinal or upper respiratory tract, the latter being more commonly involved. These anti-ganglioside antibodies target motor nerves supplying extraocular muscles as well as group 1a neurons of dorsal nerve root (DRG) ganglion controlling proprioception (because of increased concentration of GQ1b gangliosides in these areas) causing ophthalmoplegia and sensory ataxia with areflexia respectively, thus completing the clinical picture. However, a central mechanism affecting cerebellum has also been proposed, especially in anti-GQ1b antibody negative patients, causing ataxia which is cerebellar in origin, further confirmed by presence of anti-cerebellar antibodies in these patients [3].

In this report is a 28-year-old male patient diagnosed with autism spectrum disorder with associated low IQ and attention deficit disorder. He had problems with multiple social issues and had a score of 163 in the Indian Scale for assessment of autism (ISAA) (see table 1).

Case Report

A 60 year old lady presented with drooping of left eyelid, unsteadiness of gait, generalized weakness and dizziness for 5 days. She had not sustained a fall or a head injury, nor had she had episodes of loss of consciousness or seizures. She, however, did have complaints of decrease in vision, more so in her left eye. She had been in good health previously with no comorbid illnesses except her being diagnosed with hypertension for which she managed herself by dramatically reducing her salt intake.

On examining her she was found to have restricted eye movements in both her eyes (ophthalmoplegia, grade 2 palsy). Her gait was ataxic and her left finger nose finger test was found to be impaired. Sensory and motor examination did not reveal any positive findings, however she was found to have a reduced gag reflex.

Apart from MFS, alternative possible diagnosis of posterior circulation stroke was considered due to presence of cerebellar impairment. ADEM and brainstem encephalitis were other differentials thought to be possible.

Patient was investigated with CT, MRI, USG carotid vertebral arteries which revealed no abnormalities other than age related atrophic changes, thereby ruling out stroke. CSF examination was normal. Nerve conduction studies revealed bilateral carpal tunnel syndrome. EEG showed occasional epileptiform abnormalities arising from the left temporal region.

GQ1b, GM1 ganglioside antibodies were evaluated for the patient and found to be negative.

DISCUSSION

This patient had classical signs of ataxia and ophthalmoplegia at time of presentation but with normal reflexes. Since significant overlap exists between features of MFS and GBS with a wide range of neurological signs and symptoms being variably present, this diagnosis was made for the patient.

90% of the patients with MFS show seropositivity for GQ1b ganglioside complex which is concentrated in cranial nerves III, IV, VI thus clinically causing ophthalmoplegias4. However, our patient was seronegative for Anti-GQ1b as well as Anti-GM1 antibodies. Intravenous immunoglobulin was started for her before testing for these antibodies and treatment was continued even after the negative results due to

continued clinical improvement. In addition, she was ambulation.

To begin with, the patient was followed up on an out-patient basis monthly. She had an improvement of about 80% on her first visit. Two year follow up of the patient reveals full gain in functional status.

This case report highlights the importance of keeping a diagnosis of MFS in mind in patients otherwise presenting with signs and symptoms pointing towards a central cause. Even in Anti-GQ1b seronegative patients, clinical improvement with intravenous immunoglobulin should guide further management.

Conclusion

Miller Fisher Syndrome is an important differential diagnosis to consider in patients otherwise presenting with signs and symptoms that might point towards a central cause. MFS can be present even in patients with negative anti-ganglioside antibodies and an incomplete triad, provider they show improvement with intravenous immunoglobulin.

encouraged to do visual tracking exercises along with

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