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Case Report

Miller Fisher syndrome-a success story

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ABSTRACT

Acute inflammatory demyelination polyneuropathy (AIDP) or acute idiopathic polyneuritis are the other key synonyms for Guillain-Barre syndrome (GBS) with ascending characteristics of weakness, mild sensorium loss, areflexia or hyporeflexia. GBS has been seen most commonly among the younger age group, and a male predominance has been reported. The case reports demonstrate a case of a 66-year-old female admitted to the causality department with abrupt onset of giddiness, diplopia, and headache the patient was diagnosed with myasthenia gravis and atypical GBS and shifted to the ward for further treatment. The patient was in the intensive-care unit due to mild ischemic changes in the MRI brain and bilateral facial neuropathy. The patient was treated with intravenous immunoglobulins and I.V. glucocorticoids. The patient was discharged on the 25th day of admission without support walk and full extraocular movements. The case report demonstrates the essential use of high clinical knowledge and investigation for a prompt treatment for a such rare disorder.

Keywords: GBS, Reflexes, Single breath count, Ataxia, Ptosis, Miller Fisher syndrome

INTRODUCTION

The triad of ataxia, areflexia, and ophthalmoplegia was first coined by James Collier in 1932, which was subsequently reported under the characteristics of GBS by Charles Miller Fisher into three clinical cases in 1956.[1] Hence, the name Miller Fisher syndrome is one of the variants of GBS reported in 1-5% of the population in the Western region and 19-25% in Taiwan and Japan respectively. The GBS has been majorly associated with a causative factor of infectious disease, most commonly seen pathogens including; *Campylobacter jejuni* and *Haemophilus influenza*.[2]

In addition to classic ascending weakness, patients may show clinical symptoms such as paralysis, neurological defects following a top-down pattern, and diplopia in the initial stages. The prevalence of MFS is seen in the range of 8-10 days depending on the severity and comorbid conditions. In addition, major complications such as respiratory failure or cardiac arrest have been reported in 30% of patients with GBS. The disease-modifying treatment such as intravenous immunoglobulin and plasmapheresis are usually adopted.[3] The treatment is generally recommended to prevent further complications and resolve symptoms faster.[4]

CASE REPORT

A 66-year-old female was admitted to the causality department with complaints of giddiness, diplopia, and headache which was abrupt in nature. During the history interview, the patients were seen with difficulty opening her eye. The patient was photophobic and numbness was seen in the upper and lower limb of the patient. Based on the general examination, the patient did not have any history of fever, dyspnea, chest pain, or weakness of the upper and lower limb. The medical history interview reported no sleep disturbances and bowel/bladder issues. Fundus examination was reported to be normal. On examination, the patient was found to be conscious and oriented with stable vitals. However, systemic

examination revealed bilateral ptosis and restriction of extraocular movements with pupils reactive to light. The power of four limbs was evaluated which reports as 4/5 and normotonia for all the 4 limbs and absence of deep tendon reflex. The cranial nerve examination revealed nerve palsy in the 3rd, 4th, and 6th cranial nerve with positive signs of Romberg's sign and ataxia on a single breath count of 20 per breath. The patient was suspected of myasthenia gravis and GBS based on the clinical findings. The patient was shifted to the ward and treatment with intravenous glucocorticoids and physostigmine was initiated. Routine laboratory tests and specific myasthenia gravis profiles were further evaluated. HRCT chest did not report any abnormalities however, MRI-brain demonstrated small vessel ischemia changes Fazekas grade 1. The RNS study did not reveal any significant abnormalities. The patient was reported with Bilateral facial neuropathy by nerve conduction study of bilateral facial nerves and blink study. Routine blood parameters were reported in the normal range. The clinical condition of the patient worsened on the 4th day of admission which resulted in the shifting of the patient to the intensive care unit. The patient was reported with drowsiness, bilateral ptosis, mid-dilated pupils, and no reactiveness to light. In addition, the patient clinical condition resulted in decreased limb powers to 2/5 and bilateral lower limb to 3/5 with a single breath count decreasing to 13 per breath. Negative results were reported for ANA, p-ANCA, c-ANCA, ACH-R antibody, Musk antibody, COVID IgG, and IgM. In addition, anti-TPO and ASKA were also negative. CSF analysis reported elevated levels of protein-103 mg/dl, glucose-95 mg/dl, and no total cells count however, albumin-cytological dissociation was seen with the CSF analysis. The patient was suspected with GBS. The patient was presented with a confirmed diagnosis of Miller Fisher variant of GBS. After suspecting the GBS in the patient, intravenous immunoglobulin (25 gm) for 5 days was initiated with a combination of corticosteroids, supportive management, and physiotherapy. The treatment resulted in an overall improvement of patient limb power, single breath count was increased to 16 per breath however, areflexia was consistent during the admission. The sixth day revealed significant improvement in the overall power of the patient with $4^{+}/5$ in bilateral upper limb and lower limbs, improved gait abnormality, and pupils reacting to light. In addition, improvement with respect to ptosis and extraocular movement was also reported. Single breath count was seen as 18 per breath with persistent areflexia. Further to this, Anti GQ_{1B} was reported significantly higher at 1: 3200 (normal range-<1:100). The patient's treatment continued for 25 days with discharge on the 25th day. Significant improvement was seen in the patient with full extraocular movement, a single breath count of 20 per breath, and walking ability without support.

DISCUSSION

Miller-Fisher syndrome is associated with characteristic traits of ophthalmoplegia, ataxia, and areflexia without overt sensory deficits which is one of the variants of GBS also known as acute idiopathic neuritis. Miller-Fisher syndrome has been associated with notable neurological features however, studies have shown that the peripheral nervous system has been the targeted system. The atypical abnormalities of the triad were similar between the reported literature and our patient during the course of treatment. The major defining features are ataxia, ophthalmoplegia, and areflexia which are the essential symptoms for differential diagnosis of Miller-Fisher syndrome.[5] The prevalence of other symptoms may hamper the clinical decision-making of the clinician.

The use of GQ_{1B} ganglioside complex has been most commonly used for correlation of Miller-Fisher diagnosis and reported to be positive among 90% of the patients.[6] This is due to the GQ_{1B} autoantibodies that target the epitopes on the cranial nerves III, IV, and VI which results in the characteristic ophthalmoplegia.[7]

The treatment with intravenous glucocorticoids and immunoglobulins for a minimum of five days has seen to be effective in improving the overall functioning of our patient. The patient significantly improved concerning to the power of upper and lower limbs. A case report by Yepishin et al also reported similar findings of the patients with ataxia, areflexia, and ophthalmoplegia in a 55-year-old patient which was also detected for Miller-Fisher syndrome by $GQ_{1B}$and treated with I.V. glucocorticoids and immunoglobulins.[5] The recovery period of GBS varian of Miller-Fisher syndrome clinically depends on the patient condition and the treatment of immunoglobulins which results in a faster recovery from the symptoms. However, it is essential to rule out other clinical disease and risk of complications by prompt diagnosis with $GQ_{1B}$and classic clinical symptoms of Miller-Fisher syndrome.

CONCLUSION

The prevalence of GBS variant of Miller-Fisher syndrome is uncommon. However, with the use of appropriate diagnostic criteria and clinical symptoms of ataxia, areflexia, and ophthalmoplegia can be used for an early detection of the disease. The prevalence of neurological symptoms can confound the clinical decision making and affects the overall outcome of the treatment. The case reports no significant history or causative factor for GBS or Miller-Fisher syndrome but has been diagnosed with the condition. An early detection of Miller-Fisher syndrome on the 6th day has led to prompt immunoglobulin treatment which significantly improved the patient overall outcome. Hence, it is essential for clinician to keep a track on three major symptoms ataxia, areflexia, and ophthalmoplegia for ruling out the disease and providing the appropriate treatment.

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