Case Report

A rare case of miller fisher syndrome

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Abstract

Background: Miller fisher syndrome is one of the variant of Guillain Barre Syndrome which constitutes approximately 5 % of all the cases of Guillain Barre Syndrome. We present you a case of a middle-aged female who presented with classical triad of ophthalmoplegia, ataxia and areflexia. She was started with plasmapheresis, which proved to be highly effective in her treatment.

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INTRODUCTION

Miller Fisher syndrome (MFS) is a rare and treatable disease. It is characterized by ophthalmoplegia, ataxia and areflexia. It is one of the variant of Guillain Barre Syndrome (GBS). If diagnosed early it responds well to the treatment.[1] Diagnosis of MFS is made on clinical grounds but use of nerve conduction studies, cerebrospinal fluid analysis can help us to confirm diagnosis.[2] GQ1b ganglioside antibody can be measured in serum of patients having prominent ataxia or ophthalmoplegia. Absence of these antibody doesn't exclude diagnosis of MFS.[3] Plasmapheresis and intravenous immunoglobulin therapy remains highly effective modality in treatment of MFS.[4] Above mentioned treatment modalities can reduce the time taken for recovery to occur.[5]

CASE PRESENTATION

A 38-year female presented with 10 days history of tingling sensation over both upper and lower limbs, unsteadiness of gait and difficulty in swallowing with 8 days history of double vision. Patient was apparently alright 10 days back when she developed sudden onset tingling numbness in both upper limb and lower limb after getting up in the morning. It was not associated with weakness and fatigability. It started in both hand and feet and gradually progressed to both upper and lower limb over a period of 4 hrs. She also developed unsteadiness of gait, giddiness and double vision which was progressive in nature. It was so severe that she was unable to get up from bed. She also had difficulty in swallowing and had nasal twang of voice for 8 days. She denied diminution of vision, difficulty in breathing or any motor weakness. There were no signs of autonomic dysfunction, neck pain, paralytic ileus or bowel and bladder abnormalities. She gave history of upper respiratory tract infection 15 days back which got relieved after taking over the counter medications. She also had no history of convulsion, headache, altered sensorium, loss of consciousness and vomiting. There was no history suggestive of head injury or spinal injury. After 15 days of upper respiratory tract infection, she developed above neurological symptoms and signs. She had nonsignificant past, family, medical or surgical history. Patient was conscious, alert and oriented to time place and person. She had normal vital parameters including blood pressures, heart rate, respiratory rate and body temperature. The respiratory, cardiac, pulmonary and per abdominal examination were found to be normal. Patient had moderate level of discomfort and preferred to keep her eyes closed. She had intact memory and was able to communicate. He had no aphasia, dysarthria and had normal comprehension of language. Her pupils were of equal size and were reactive to light on both sides. She had

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normal accommodation reflex. However, her extraocular movement were totally absent for both the eyes and there was bilateral ptosis which was suggestive of external ophthalmoplegia. She had normal hearing and had no weakness of tongue. Muscles of both upper and lower limb had normal power. She had no sensory deficit. She had areflexia for both superficial and deep tendon reflexes. She was unable to perform finger to nose and heel to shin test because of ataxia. Her routine blood examination like complete blood count, thyroid profile, metabolic panel and biochemical investigations including liver and renal function were within the normal range. Random blood sugar, serum electrolytes including calcium levels were within normal range. There was no elevation in erythrocyte sedimentation rate, C-reactive protein or creatine kinase. She was non-reactive for HIV. Lumber puncture revealed cytoalbuminologic dissociation with no cells and elevated protein which was 248 mg/dl. Nerve conduction studies revealed absent H reflexes with rest of parameters within normal limit. Electromyography and evoked potential studies were negative. Her CT brain and MRI were normal. GQ1b antibody were found to be negative. Patient was diagnosed to have Miller Fisher Syndrome on the basis of clinical triad of ophthalmoplegia, ataxia and areflexia, NCV and CSF studies. Patient was started on plasmapheresis and supportive management on day of admission itself. 5 cycles of plasmapheresis were done. Patient gradually improved over the period of 12 days. All the neurological symptoms and signs improved over period of 12 days. She was discharged on 15th day of admission after completion of therapy.

DISCUSSION

Miller Fisher Syndrome is seen approximately 5 % of all the cases of GBS. Unlike GBS, MFS causes involvement of multiple cranial nerves, ataxia and absence of motor weakness.[1] MFS is an autoimmune disease seen in patients following infection to Campylobacter jejuni, Cytomegalovirus and Epstein Barr virus. Histological examination of peripheral nerves has made it clear that both cell mediated and humoral immunity play an important role in pathogenesis of disease.[5] With classical triad of ophthalmoplegia, ataxia and areflexia, MFS can be easily diagnosed clinically.[2] Alcohol use, paraneoplastic cerebellar degeneration, Hashimoto encephalopathy, spinocerebellar ataxia, brain tumors and vestibular neuritis remains close differentials of MFS.[6] Use of nerve conduction studies, cerebrospinal fluid analysis and radio imaging studies can help us to confirm diagnosis of MFS. [2] Antibodies against GQ1b remains specific for MFS and is seen in majority of patients who have predominant symptoms of ataxia and ophthalmoplegia. However, few patients of MFS doesn't have these antibodies. Hence absence of this antibody in serum doesn't rule out the diagnosis of MFS. [3] These antibodies causes neuromuscular blocks which explains pathogenesis of MFS.[5] Plasmapheresis and intravenous immunoglobulin therapy are the most commonly used treatment modalities for MFS. Plasmapheresis is commonly used as compared to intravenous immunoglobulin due to relative cost effectiveness. [4] Both the treatment modalities have shown to decrease progression of disease and time taken for recovery. Even after successful treatment 8 % mortality and 20 % morbidity is seen in patients of MFS.[5]

CONCLUSION

Miller Fisher syndrome remains one of the rarest form of Guillain Barre Syndrome. This case concludes that absence of GQ1b ganglioside antibody doesn't exclude diagnosis of MFS. Clinical presentation along with nerve conduction studies and cerebrospinal fluid analysis can help to diagnose MFS. Plasmapheresis remains one of the highly effective treatment for patients of MFS. Early initiation of plasmapheresis and supportive management have shown to decrease the progression and time taken for recovery.

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