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Case Study

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CASE REPORT, MILLER FISHER SYNDROME

Hannah Elizabeth Sabu*[1], Merin Elza Anil[2], Hannah Joseph[3] and Pooja Gopan[4]

[1,2]Pharm D, Nazareth College of Pharmacy, Othera, Thiruvalla.
[3]Clinical Pharmacist, Pharm D. Department of Neurology, Believers Church Medical. College Hospital, Thiruvalla, Kerala.
[4]Clinical Pharmacist, Pharm D, Department of Neurology, Believers Church Medical College Hospital, Thiruvalla, Kerala.

***Corresponding Author: Hannah Elizabeth Sabu**

Pharm D, Nazareth College of Pharmacy, Othera, Thiruvalla.

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ABSTRACT

MFS is an acute, autoimmune polyneuropathy, a subgroup of Guillain-Barre Syndrome (GBS) that is clinically characterized by triad ataxia, ophthalmoplegia, and areflexia.[1] Here we present a case report of a 5-year-old with MSF who presented with no clinical symptoms and was diagnosed with MSF on IgG antibody screening.

KEYWORDS: Miller-Fisher syndrome, Guillain-Barré syndrome, Immunoglobulin therapy.

INTRODUCTION

Miller Fisher syndrome (MFS), also known as Fisher syndrome and the Miller Fisher Variant of Guillain-Barré syndrome. In honor of Dr. Charles Miller Fisher, the Miller Fisher syndrome (MFS) was named. MFS is an acute, autoimmune polyneuropathy, a subgroup of Guillain-Barre Syndrome (GBS) that is clinically characterized by triad ataxia, ophthalmoplegia, and areflexia.[1]

The major difference between MFS and other variants of GBS is that the first group of nerves to demyelinated are commonly located in the cranium which results in difficulties with balance and coordination, ocular muscle movement, vision impairment, and neuronal reflexes.

MFS is a clinical diagnosis but often goes undiagnosed due to its low prevalence. MFS is a clinical diagnosis that can be confirmed serologically with positive anti-ganglioside GQ1b antibodies.[2]

Infectious, autoimmune, and neoplastic disorders can be linked to MFS. The pathogens commonly involved are Campylobacter jejuni and Haemophilus influenzae. The premonitory disease most frequently reported is an upper respiratory infection, followed by a gastrointestinal condition.[3]

Although documented cases of MFS have been reported for most of the cranial nerves, it is primarily linked to dysfunction of the third, fourth, and sixth cranial nerves.[4]

Figure 1 shows the Pathogenesis of Miller Fisher Syndrome associated with IgGanti-GQ1Bb Antibody after Campylobacter Jejuni Enteritis. C.Jejuni bears GQ1b-like LPS that is associated with Penner's serotype-2 (PEN-2) antigenic determinant. PEN-2 may induce GQ1b-like LPS to increase the synthesis of IgG anti-GQ1b antibody with the help of T-cells and B-cells. These IgG anti-GQ1b antibodies bind to third, fourth, and sixth cranial nerves and to cerebellar nuclei resulting in the development of clinical features of MFS (Ophthalmoplegia, Ataxia).[5]

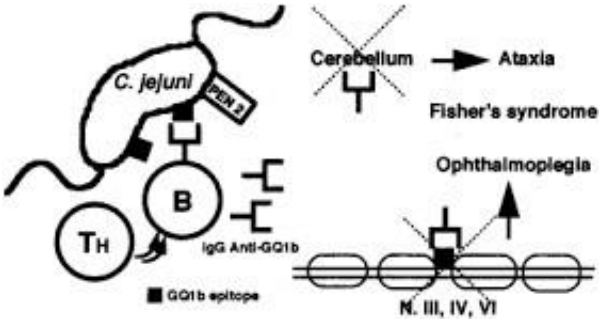

Figure 1: Shows the Pathogenesis of Miller Fisher Syndrome associated with IgGanti-GQ1Bb Antibody after Campylobacter Jejuni Enteritis.

PEN-2 = Penner's serotype 2, Th = T cells, B = B cells, III = Oculomotor nerve IV = Trochlear nerve, VI= Abducens nerve.

Other than the most prevalent clinical triad of ataxia, ophthalmoplegia, and areflexia, MFS is associated with distal paraesthesia, diplopia, dysarthria, blepharoptosis, mild (grade 4) motor weakness; pupillary palsies; limb dysesthesia; and micturition disturbance, etc.[6]

Due to their similar clinical signs to MFS, the patient may be misdiagnosed with Guillain-Barré Syndrome (GBS) and Bickerstaff brainstem encephalitis (BBE). GBS and MFS can be diagnosed on clinical grounds (Clinical history, cardinal symptoms), it can be confirmed with the help of imaging (e.g. ultrasound and MRI), serologic testing (e.g.Anti-GQ1b antibodies, etc), cerebrospinal fluid (CSF) analysis and electrodiagnostic (EMG, nerve conduction, or evoked potential).[6,7]

The two main treatment options available for MFS are immunoglobulin therapy (IVIG) and plasmapheresis.

IVIG involves delivering high protein dosages used by the immune system to combat infections. IVIG is given to patients who have MFS with dysphagia and breathing difficulties. IVIG dose is 2 g/kg divided over 2 to 5 days. In children and adolescents, a dose of 1 g/kg per dose IV daily for 2 days is recommended. In patients with renal impairment, approximately 50% of the usual dose should be used by physicians.

Plasmapheresis is the procedure in which the RBC and WBC are removed from the plasma part of the blood and these cells are reintroduced to the body without the plasma. When given within 2 weeks of illness onset in patients who are unable to walk, plasma exchange is effective and is highly effective within seven days of weakness onset.

CASE REPORT

A 5-year-old male came to the emergency department with complaints of vomiting containing food particles and sputum for 2 days (4 episodes), abdominal pain, and generalized tiredness. He also had nasal congestion and a sore throat. The patient did not have any allergies or any other comorbidities. He had a contact history of acute febrile illness with his family member and took a Tab. Panadol. Physical examination showed that the patient was conscious, obeying verbal commands, and having memory intact. On cardiac auscultation, a regular heartbeat with no murmur was heard and a heart rate of 82 beats/min was recorded. Respiratory auscultation revealed symmetrical breath sounds and normal bronchial airway entry with a respiratory rate of 20 breaths/min. His abdomen was soft, regular, and non-tender, with no organomegaly. Acute gastritis, viral fever, or appendicitis was suspected and provided with Tab. Emeset 4mg Stat, Tab. Pantoprazol 40mg Stat, Junior Lansoprazole 30mg BD for 3days, Tab. Azithromycin 500mg OD for 5 days and Syrup Fexofenedine 5ml. The patient was advised to do a Ganglioside antibody evaluation panel in serum. The result showed IgG-GQ1b antibody 210 %, IgG-GD1b antibody 32%, IgG-GM1 antibody 18%, IgM-GD1b antibody 17%, IgM-GQ1b antibody 56% and IgM-GM1 antibody 16%. It was interpreted that the serum sample tested strongly positive for IgG antibody to GQ1b and equivocal for GD1b and negative for GM1. Seropositivity of Anti GQ1b-IgG is a diagnostic marker for Miller Fisher Syndrome and seronegativity of Anti-TgM conveys that the patient was not having sensory ataxia.

DISCUSSION

Miller-Fisher's syndrome is a rare variant of Guillain-Barré's syndrome characterized by the acute development of ataxia, ophthalmoparesis, and areflexia. Patients typically seek medical attention because of a rapid decrease in vision over days and/or difficulty walking. These changes are frequently preceded by a viral or diarrheal illness 1 to 4 weeks earlier.[8] In a study conducted by Koga M et.al, preceding infection is an important clue for differential diagnosis in MFS, Campylobacter jejuni and Haemophilus influenzae infections were evident in 21% and 8% of MFS patients.[9] Besides the characteristic clinical triad (ophthalmoplegia, ataxia, and areflexia), pupillary abnormalities, blepharoptosis, and facial palsy are frequent in MFS, whereas sensory loss is unusual despite the presence of profound ataxia [(10)]. MSF is associated with acute-phase IgG antibodies to GQ1b and GT1a gangliosides. This testing helped the physicians to lead to a conclusion of Miller Fisher Syndrome. Another reported study done by Mori M et.al showed that the positivity rate for anti-GQ1b antibodies has been reported as more than 80% in MFS.[11]

Despite the fact that Miller-Fisher Syndrome is a self-limiting autoimmune condition, immunomodulatory treatments such as intravenous immune globulin and plasmapheresis are used for recovery. In this case, the patient didn't need any management for MSF, he was stable on providing symptomatic treatment. Pediatric MFS patients tended to recover faster than adult MFS patients.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

ABBREVIATIONS

MSF - Miller Fisher syndrome GBS - Guillain-Barré syndrome IVIG - Immunoglobulin therapy.

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