

A boy with multiple sclerosis and Miller-Fisher syndrome: a case report

Saqer bulayhid H albulayhid¹, Abdullah Ibrahim A Bin Eid², Waleed A. Alluhidan³, Saleh swailem⁴

^{1,2} Neurology Resident at King Saud Medical City, Riyadh, Saudi Arabia

³ Neurology Department, Prince Sultan Military Medical City, Riyadh

³ Adult Neurologist & Epileptologist at King Saud Medical City, Riyadh, Saudi Arabia

Correspondence to: Saqer bulayhid H albulayhid, Neurology Resident at King Saud Medical City, Riyadh, Saudi Arabia, Email: Sa.Albulayhid@ksmc.med.sa

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ABSTRACT

Background: Miller-Fisher syndrome (MFS) is a rare condition that is well-described in the literature as a variant of Guillain-Barré syndrome (GBS). Guillain-Barre syndrome (GBS) and multiple sclerosis (MS) are autoimmune demyelinating disorders of the peripheral and central nervous systems, respectively. Anti-GQ1b IgG antibody is a biomarker for and MFS. The co-occurrence of these MFS and MS conditions is rare in the literature.

Case Report: Our case report presents a complex case of a 15-year-old male with a history of T1DM and hypothyroidism, which came with ataxia, intentional tremor, suspected white matter disorder, and recent double vision and eyelid ptosis. Initially he was diagnosed with multiple sclerosis (MS), further investigation revealed Miller-Fisher syndrome (MFS) the rarest variant of Guillain-Barre syndrome (GBS) with anti-GQ1b antibody positivity.

Conclusion: Despite rarity, the co-existence of GBS and MS must be kept in mind by neurologists. The well-established diagnostic criteria should be met to diagnose both diseases. To the best of our knowledge, this is the first reported patient with this unusual concurrence of MS with MFS.

Keywords:

Miller-Fisher syndrome, demyelinating disease, Multiple sclerosis, Guillain-Barré syndrome, neurological disease, case report.

Introduction

Miller-Fisher syndrome (MFS) is a rare variant of Guillain-Barre syndrome (GBS), the most common GBS variant [1]. Chiba et al. first described the correlation between anti-GQ1b IgG antibody and MFS, suggesting this antibody as a biomarker for MFS [2].

MFS has been co-existing with other neurological diseases, such as Myasthenia Gravis [3], central nervous system demyelination, [4, 5], and overlap with GBS, making diagnosis challenging and potentially causing complications [6].

Multiple sclerosis (MS) is the most common chronic autoimmune demyelinating disease of the central nervous system (CNS) [7]. Pathologically, MS is described by inflammation, demyelination, and axonal damage, with progressive neurodegeneration, caused by an autoimmune response to self-antigens, leading to loss of motor and sensory function [8].

The co-existence of Miller-Fisher syndrome (MFS) and Multiple Sclerosis (MS) in an individual's lifespan has been rarely reported. Herein we describe a case of a 15-year-old boy with a spectrum of neurological symptoms who had a positive anti-GQ1b IgG antibody and was diagnosed with a co-existence of Miller-Fisher syndrome (MFS) and Multiple Sclerosis (MS).

Case Report:

A 15-year-old male with a history of diabetes mellitus type 1 (T1DM), hypothyroidism, and upper respiratory tract illness (URTI) presented with double vision and bilateral eyelids dropping two weeks before the presentation. The patient was born at 38 weeks of gestation and had a family history of seizures, weakness, and left orchidopexy. He had a 9-month history of ataxia, falls, intentional tremors, and slurred speech. His mother and father are first-degree cousins.

Upon arrival, the physical examination found that the patient exhibited left horizontal gaze complete failure and right horizontal gaze impairment, bilateral lower motor neuron facial nerve palsy, deep tendon reflexes absent all over his upper and lower limbs, appendicular ataxia on the left side more than the right, and impaired proprioception and vibration on distal lower limbs.

The initial laboratory tests were all within normal ranges. The autoimmune panel was done; it showed a positive AntiGQ1B antibody. Whole exon sequencing was positive for gene MPP7 heterozygous, POLR.

Electroencephalogram (EEG) showed Interictal Epileptiform Dischargers (IEDs). A brain computed tomography (CT) revealed bilateral supratentorial scattered white matter changes. He was subsequently admitted under service for further evaluation. Magnetic resonance imaging (MRI) brain and whole spin with contrast showed multiple supratentorial, infratentorial, and spinal cord lesions suggesting underlying demyelinating disease, multiple sclerosis (Figure 1).

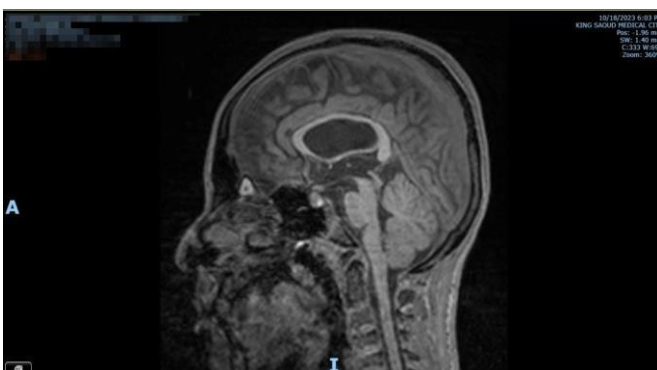


Fig. 1

There was a faint incomplete ring-enhancing lesion in the left periventricular area worrisome for the active lesion. Nerve conduction study results showed prolonged latency, low

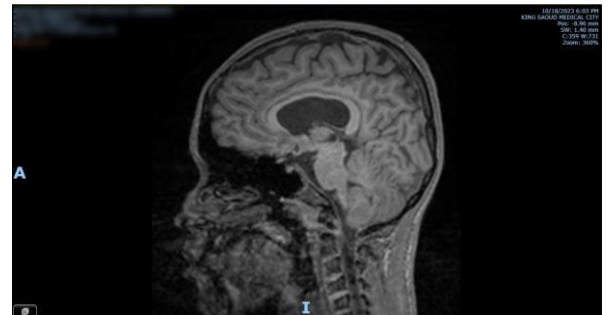


Fig. 2

of Guillain-Barré syndrome (Figure 2). The patient received intravenous immunoglobulin (IVIG) for 5 days. He was discharged with an outpatient appointment for follow-up after 6 weeks with the neuroimmunology clinic for further evaluation regarding the demyelinating lesion on MRI and for possible LP to send for demyelinating workup.

Discussion:

The basic clinical trial of Miller-Fisher syndrome (MFS), a variation of Guillain-Barre syndrome (GBS), involves urticaria, ophthalmoplegia, ataxia, and areflexia [9, 10]. It is a neurological condition that is quite uncommon, affecting around 5% of GBS patients [9]. More than 80% of MFS patients have distinct anti-ganglioside antibodies, mainly IgG anti-GQ1b antibodies [11], indicating that the illness is immune-mediated. T1DM, neurological disorders such as cerebellar ataxia, refractory epilepsy, stiff-person syndrome, and aberrant eye movements have all been associated with these antibodies [12]. Our patient had a history of T1DM, hypothyroidism, ataxia, falls, tremors, slurred speech, URTI, double vision, and bilateral eyelids. His family history of seizures and weakness suggests a genetic susceptibility to autoimmune disorders. AntiGQ1B antibody positive, directing diagnosis toward MFS. Electrodiagnostic investigations for MFS may reveal diminished or nonexistent sensory responses without causing a slowdown in sensory conduction studies [13]. The cauda equina and intrathecal spinal nerve roots, and some augmentation of the spinal nerve roots, may be seen on CT/MRI scans of the spine.

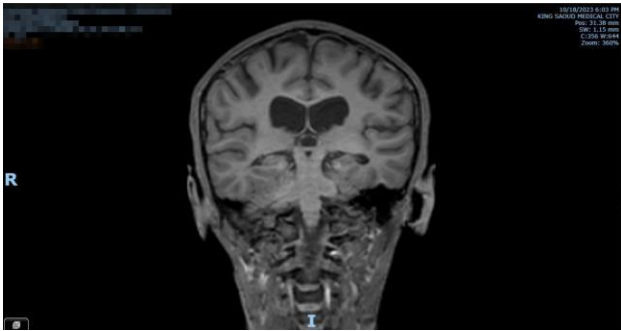


Fig. 3

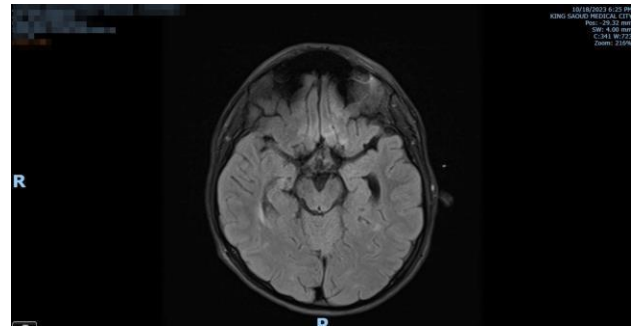


Fig. 4

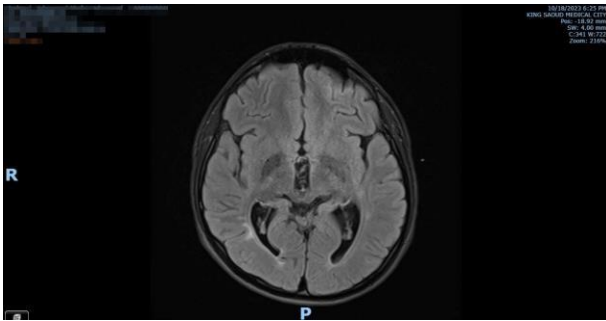


Fig. 5

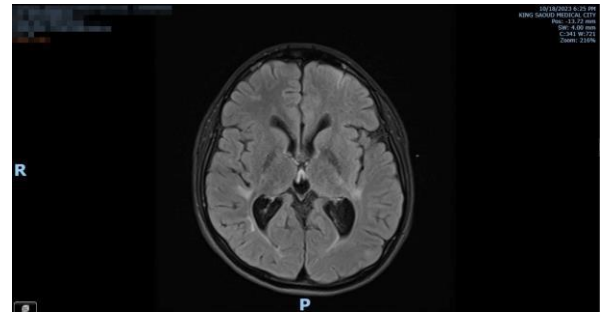


Fig. 6



Fig. 7

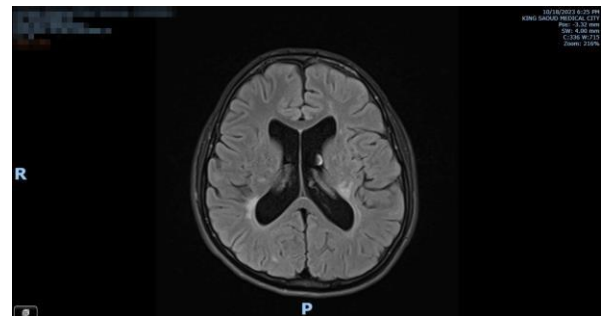


Fig. 8

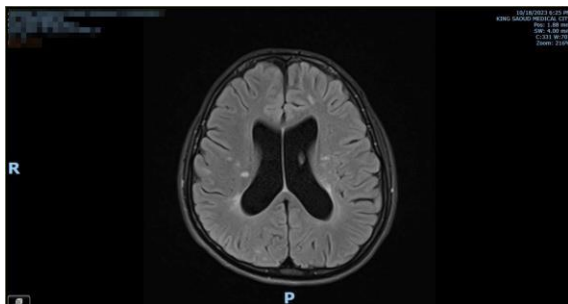


Fig. 9

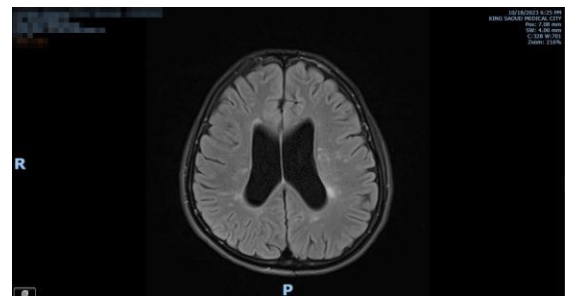


Fig. 10

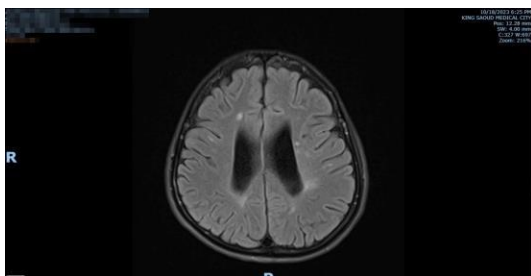


Fig. 11

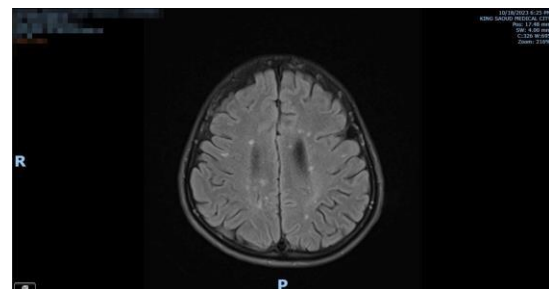


Fig. 12

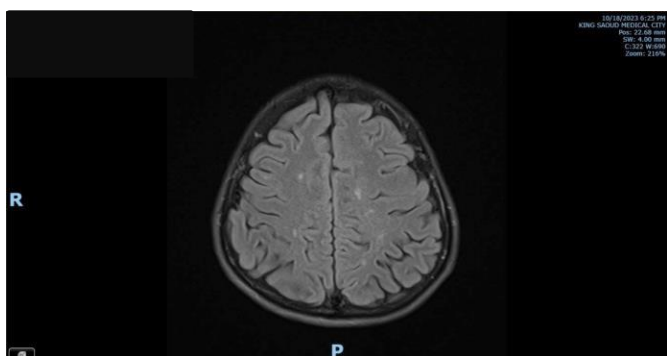


Fig. 13

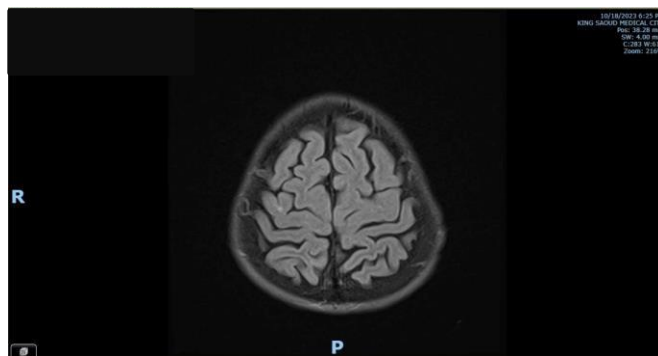


Fig. 14

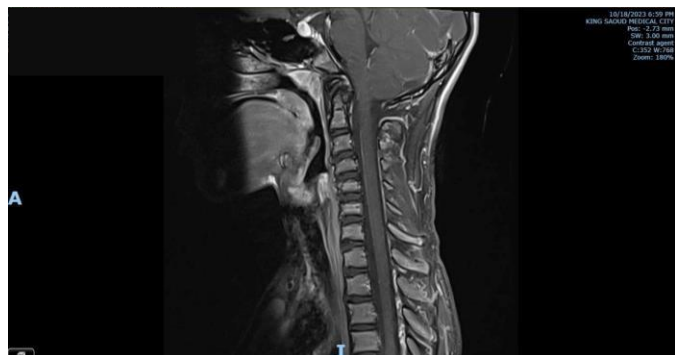


Fig. 15



Fig. 16



Fig. 17

Findings:

Compared to previous MRI of the brain and spine done on January 24, 2023:

Interval stability of multiple supratentorial and infratentorial white matter lesions with some appearing perpendicular to the lateral ventricles and involving the corpus callosum. No newly developed lesions. There is a single faint incomplete ring-enhancing lesion in the left periventricular area. Bilateral optic nerves appeared unremarkable.

No acute brain result

The rest of the exam shows no interval changes.

Unchanged a few scattered intramedullary white matter lesions in the cervical and upper thoracic spinal cord. No abnormal enhancement. No cord expansion or atrophy.

No canal stenosis at any level. No disc abnormality. Visualized vertebral bodies appeared unremarkable apart from mild anterior wedging from C4 up to T2.

Anomalies of the brain's oculomotor, abducens, and facial nerves, as well as the spinal cords posterior columns, have been documented in the literature [14]. Regarding our case, electroencephalogram (EEG) showed IEDs. A brain CT showed bilateral supratentorial scattered white matter changes. Brain MRI showed multiple supratentorial, infratentorial, and spinal cord lesions suggesting multiple sclerosis. Nerve conduction study results showed prolonged latency, low amplitude, and slowed velocity, which fulfills the criteria of GBS, and MFS. Therefore, this case reported the coexistence of two distinct autoimmune disorders that affect the nervous system concurrently. This is supported by the presence of anti-GQ1b antibodies (specific to GBS, MFS) and MRI findings suggestive of multiple sclerosis. To the best of our knowledge, seven individuals with concurrent diagnoses of MS and GBS have been reported in a population-based study by Etemadifar et al. [4], and only ten cases have been detailed in various case report studies.

Intravenous immunoglobulin (IVIG) and plasma exchange are effective treatments for GBS and severe MFS cases, with no difference in mortality, disability, or intubation length. IVIG is preferred due to convenience, availability, and minimal side effects, but can be costly for low-income or underinsured cases [15]. The patient received five days of IVIG therapy for GBS, which is effective in reducing symptoms and improving recovery. His prognosis is good, with significant improvement within weeks to months. However, further evaluation and monitoring are needed due to the possible underlying demyelinating disease. A lumbar puncture will be performed to analyze cerebrospinal fluid for specific markers of MS and other demyelinating diseases. This will help in making a definitive diagnosis and guide long-term management strategies.

Regular neurological examinations and neuro-imaging scans are crucial for monitoring the patient's neurological status and identifying potential disease progression, with long-term management potentially involving disease-modifying therapies based on the definitive diagnosis of the demyelinating disease.

Conclusion:

Despite rarity, the co-existence of GBS and MS must be kept in mind by neurologists. The well-established diagnostic criteria should be met to diagnose both diseases. To the best of our knowledge, this is the first reported patient with this unusual concurrence of MS with MFS.

Acknowledgement:

None

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