CASE REPORT

Miller fisher syndrome and Lyme disease: An exceptional case

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SUMMARY

Miller Fisher syndrome (MFS), a rare variant of Guillain-Barré syndrome, and Lyme disease (LD), a tick-borne illness caused by Borrelia burgdorferi, are distinct entities. Both can trigger immune-mediated neurological complications. However, their coexistence is exceedingly rare, with only one case previously reported. Here, we describe a 17-year-old boy with concurrent seropositivity for Borrelia burgdorferi IgM and anti-GQ1b IgM and IgG antibodies, supporting the co-occurrence of LD and MFS. He presented with bilateral ptosis and binocular diplopia following a riverside picnic one week prior, along with headaches, nausea, and lower extremity paraesthesia. Neurological examination revealed gait ataxia and areflexia, but brain imaging: CT and MRI, was unremarkable. Lumbar puncture showed elevated cerebrospinal fluid protein. The patient was treated with a five-day course of intravenous immunoglobulin and two weeks of oral doxycycline. He responded well to therapy and achieved complete neurological recovery within one month. This case illustrates the rare coexistence of MFS and LD, highlighting the diagnostic complexities and demonstrating the efficacy of timely immunoglobulin and antibiotic therapy.

INTRODUCTION

Miller Fisher syndrome (MFS) is an immune-mediated neuropathy characterised by ophthalmoplegia, ataxia, and areflexia. With an annual prevalence of one to two per million, MFS is more frequently reported in Asia, accounting for 15-25% of GBS cases, compared to approximately 5% in Western countries.[1] While MFS is commonly triggered by infections, particularly Campylobacter jejuni and Haemophilus influenzae, the potential role of Borrelia burgdorferi, the causative agent of Lyme disease (LD), in triggering post-infectious MFS-related neuropathies is less established. Although neurological complications of LD and MFS have been individually documented, serological co-occurrence remains exceedingly rare, with only one previous case reported.[2] Here, we describe an exceptional case of MFS in a teenager with confirmed Borrelia burgdorferi IgM and anti-GQ1b IgM seropositivity, suggesting a recently acquired Borrelia infection alongside concurrent immune-mediated neuropathy.

CASE PRESENTATION

A 17-year-old boy with no known comorbidities presented with a four-day history of bilateral ptosis and binocular diplopia, accompanied by fever, headache, nausea, and paraesthesia over the lower extremities. Upon further questioning, he reported attending a riverside picnic one week prior to the symptom onset. However, he was unaware of any tick bites during the outing.

His vision was 6/18 in both eyes, improving to 6/9 with a pinhole test. Ocular examination showed partial ptosis in both eyes, sparing the visual axis, complete ophthalmoplegia (Fig. 1) and tonic dilated pupils with diminished light reflex (Fig. 2A). The anterior segment and fundus examinations were unremarkable. Neurological examination revealed areflexia and gait ataxia.

Brain computed tomography (CT) and magnetic resonance imaging (MRI) of the brain and spine were unremarkable. Lumbar puncture revealed elevated levels of CSF protein. Infective serology testing confirmed Borrelia burgdorferi IgM positivity. Immunoserology testing for anti-GQ1b IgM, IgG, and anti-GT1a IgG antibodies was also positive. A diagnosis of MFS and LD was established. The patient received a five-day treatment with intravenous immunoglobulin (IVIG) and a two-week course of oral doxycycline 200mg once daily. A complete recovery was observed within one month, as evidenced by improved light reflexes (Figure 2B), resolution of bilateral ptosis and external ophthalmoplegia (Figure 3). At three-month follow-up, the patient remained asymptomatic.

DISCUSSION

MFS is an antibody-mediated neurological disorder with anti-GQ1b ganglioside antibodies, detected in up to 80% of cases. These autoantibodies selectively target ganglioside-rich regions in the brainstem and peripheral nerves, leading to demyelination and axonal injury. The resulting neuropathy typically manifests as the classic triad of ophthalmoplegia, ataxia and areflexia.[1] Our patient presented with this hallmark triad, along with systemic symptoms including fever, headache, nausea, and limb paraesthesia, prompting a thorough neurological evaluation. The simultaneous detection of Borrelia burgdorferi IgM, anti-GQ1b IgM, IgG, and anti-GT1a IgG antibodies strongly supports a diagnosis of MFS potentially triggered by a recent LD infection.

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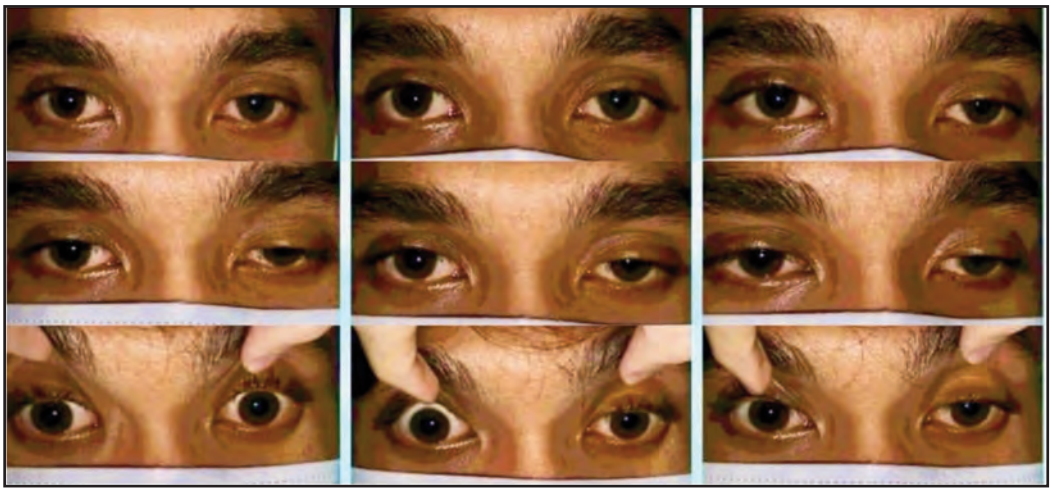

Fig. 1: Partial ptosis with "frozen eye" appearance in both eyes at presentation

Fig. 2: Pupillary examination at presentation (A) shows bilateral tonic dilated pupils, with improved light reflexes at one-month follow-up following immunoglobulin therapy (B).

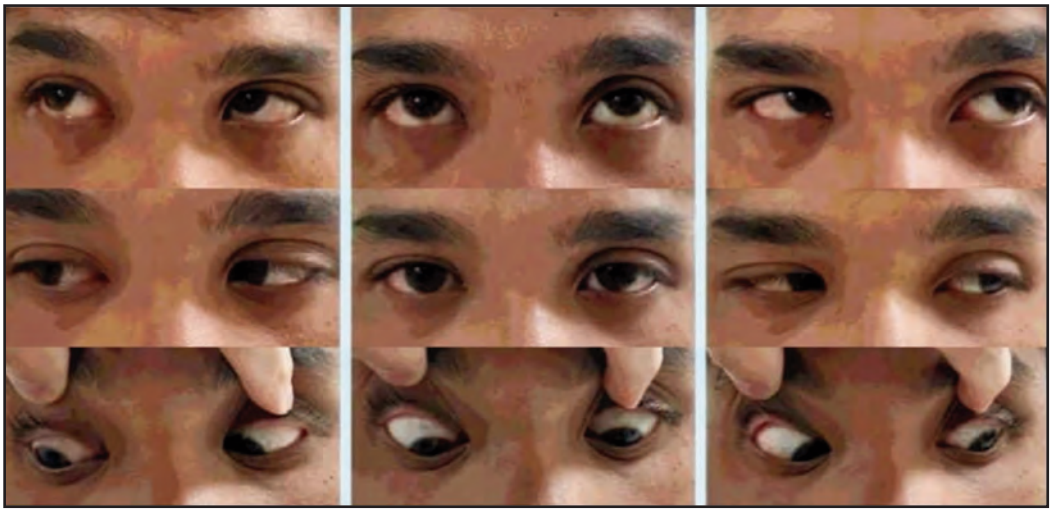

Fig. 3: Complete recovery of ptosis and ophthalmoplegia during one-month follow-up.

There are scarce reports describing cases of GBS and MFS associated with LD to suggest that Borrelia burgdorferi infection may trigger immune-mediated neuropathic complications. One comparable case involved an MFS patient without a known history of tick bites, who tested positive for Borrelia burgdorferi IgM in the CSF and seropositive for anti-GQ1b antibodies.[2] Notably, this patient showed neurological improvement by day four following treatment with IVIG and ceftriaxone,[2] contrasting with the more prolonged recovery observed in our case. Other documented cases include LD-associated GBS in a pregnant woman who presented with painful, asymmetric polyradiculopathy six weeks after a tick bite,[3] and MFS in an adult with a history of LD during adolescence.[4] Interestingly, previous cases occurred in individuals in their mid-20s to 30s.[2-4] In contrast, our case represents the youngest patient described to date, suggesting that Borrelia burgdorferi may trigger MFS across a broader age range than previously recognised.

While Borrelia burgdorferi is not known to induce ganglioside antibodies directly, infections may serve as nonspecific immune triggers in genetically predisposed individuals.[2-4] An essential diagnostic consideration in our case is the potential for a false-positive Borrelia burgdorferi IgM result, which may occur due to cross-reactivity with other infections or autoimmune processes. The lack of CSF analysis for anti-Borrelia antibodies and confirmatory Western blot and IgG seroconversion also introduces diagnostic uncertainty. However, the absence of other antecedent infections strengthens the hypothesis that LD contributed to immune activation in our patient, leading to MFS. Although classic LD features such as erythema migrans or joint pain were absent, the limb paraesthesia suggestive of radiculopathy, and the recent outdoor exposure raises suspicion for LD infection.[1]

Diagnostic evaluation plays a crucial role in confirming MFS while ruling out other neurological conditions.[1] CSF analysis in our patient revealed elevated protein levels, consistent with previously reported cases of albuminocytologic dissociation.[4] However, it is important to note that normal CSF protein levels do not exclude MFS, especially in the early stages due to the CSF test timing and individual variations.[1] Additionally, neuroimaging (CT and MRI) is mandatory in all cases presenting with neurological deficits to exclude inflammatory or infective processes in the brain or space-occupying lesions.[1,3] In this case, the diagnosis of MFS was confirmed through characteristic clinical findings and positive serology, with otherwise normal imaging studies.

The patient's favourable response to IVIG and doxycycline therapy underscores the importance of prompt diagnosis and treatment in both MFS and LD. While the management of MFS is primarily supportive, IVIG remains a cornerstone of treatment.[6] Given that MFS is an autoimmune disorder targeting gangliosides, early IVIG administration is critical, as it modulates the immune system, neutralises pathogenic antibodies, and minimizes further nerve damage.[3,6] MFS generally has a good prognosis and outcome, but prompt IVIG therapy shortens illness duration, promotes early recovery, and prevents lasting neurological complications.[6] The addition of doxycycline, the first line treatment of LD, effectively addressed the active infection, preventing potential long-term sequelae.[5] Though often costly, serological testing is essential for accurate and confirmatory diagnosis, especially in patients with atypical or overlapping symptoms in complex neurological diseases.

CONCLUSION

This case highlights the rare coexistence of MFS and LD, underscoring diagnostic and management challenges. Early treatment with immunoglobulin and antibiotic therapy is crucial for optimal recovery and improved neurological outcomes.

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DECLARATIONS

Consent was obtained from the patient prior to publication. There is no conflict of interest related to this study. This study was made without any financial support. This manuscript has been read and approved by the named authors.

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