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Recognizing pediatric Miller Fisher syndrome: A case of postinfectious neuropathy in a 7-year-old child

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Abstract

Background: Miller Fisher Syndrome (MFS) is a rare postinfectious variant of Guillain–Barré syndrome characterized by the clinical triad of ophthalmoplegia, ataxia, and areflexia. With an estimated incidence of 1-2 cases per 1,000,000 individuals annually, MFS is an exceptionally uncommon diagnosis in the pediatric population and may be difficult to recognize early. Delayed recognition may allow continued immune-mediated demyelination and prolonged recovery.

Case presentation: This case describes a previously healthy 7-year-old girl who developed MFS following an upper respiratory infection. She initially presented with cough, congestion, and right eyelid ptosis. She was referred to ophthalmology, delaying neurologic evaluation by approximately 24 hours. Within one day, she developed bilateral ptosis, ataxia, lower extremity weakness, and areflexia. Magnetic resonance imaging demonstrated ventral nerve root enhancement consistent with demyelinating neuropathy. Cerebrospinal fluid analysis was normal, and anti-GQ1b antibody testing was positive. She was treated with intravenous immunoglobulin (2 g/kg total) and received multidisciplinary inpatient rehabilitation, including physical, occupational, and speech therapy. At two-month follow-up, she demonstrated approximately 60-70% improvement in baseline motor function, with residual areflexia, mild balance difficulties, and fatigue on exertion.

Conclusion: This case highlights the diagnostic challenges of pediatric MFS and emphasizes the importance of early recognition and timely immunotherapy. Even brief delays in neurologic evaluation may influence early recovery trajectories. Maintaining a high index of suspicion for MFS in children presenting with acute cranial nerve deficits and gait instability is essential to facilitate prompt treatment and optimize outcomes.

Keywords: Miller fisher syndrome; Peripheral neuropathy; Pediatrics; Intravenous immunoglobulin; Early diagnosis; Postinfectious neuropathy.

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Introduction

Miller Fisher Syndrome (MFS) is a rare variant of Guillain–Barré Syndrome (GBS), an immune-mediated polyneuropathy affecting the peripheral nervous system [1]. According to the literature, MFS is exceptionally rare, affecting approximately 1–2 individuals per 1,000,000 people annually [1,2]. This syndrome presents with a classic triad of ophthalmoplegia, ataxia, and areflexia [1]. Although typically self-limited, delayed recognition may prolong recovery. Timely diagnosis and early intervention with immunotherapy, supported by coordinated rehabilitation, are critical for optimizing outcomes and minimizing long-term neurological sequelae [3].

This case describes a 7-year-old girl who developed Miller Fisher syndrome following an upper respiratory infection, highlighting the importance of early recognition and diagnosis, coordinated management, and the potential impact of treatment timing on recovery.

Case presentation

A previously healthy 7-year-old girl presented with cough, nasal congestion, and right eye swelling following a one-week history of upper respiratory symptoms. Rapid influenza and streptococcal antigen tests were negative. Physical examination revealed right upper eyelid ptosis and a left erythematous tympanic membrane. She was diagnosed with acute left otitis media and prescribed amoxicillin (400 mg/5 mL, 10-day course). She was also referred to ophthalmology for acute onset ptosis. The family was advised to seek emergency evaluation if her symptoms worsened.

Within 24 hours, she presented to the emergency department with diffuse bilateral leg pain, unsteady gait, and a softening of voice. Physical examination revealed bilateral ptosis, right predominant lower extremity weakness, areflexia at the patellar and ankle tendons, and bilateral absent Babinski reflexes. Her speech was soft but without evidence of dysphagia. Based on these findings, Guillain-Barré syndrome (Miller Fisher syndrome variant) was suspected, and diagnostic studies were initiated.

Laboratory evaluation was largely unremarkable aside from mildly decreased vitamin D levels and low-titer autoantibody elevations. Cerebrospinal fluid analysis showed normal glucose and protein without albuminocytologic dissociation. Complete laboratory values are presented in (Table 1). Brain MRI demonstrated subtle ventral nerve root enhancement. Myasthenia testing was negative, neurofilament light chain levels were elevated, and anti-GQ1b antibodies were positive.

She received two doses of intravenous immunoglobulin (IVIG; total dose 2 g/kg over two days) along with supportive inpatient management. Multidisciplinary therapy, including physical, occupational, and speech therapy was initiated during hospitalization. Speech therapy evaluation demonstrated oral motor muscle weakness, reduced breath support, and limited verbal output. Physical therapy assessment noted decreased balance, impaired manual dexterity, generalized weakness, and visual deficits that affected her activities of daily living.

Following clinical stabilization, she was discharged with close outpatient follow up and counseling regarding concerning signs such as persistent fever or seizures. After discharge, she showed gradual improvement and was able to ambulate independently, though she required a rolling walker for long distances. Her parents reported persistent balance difficulties, frequent falls, and some regression in fine motor skills. One week later, she presented with a sore throat, cough, and fever. Physical examination revealed purulent nasal drainage, erythematous tonsils, and bilateral crackles on auscultation. A rapid streptococcal test was negative, and she was diagnosed with *Mycoplasma pneumoniae* infection, which was treated with a five-day course of azithromycin.

Table 1: Comprehensive laboratory evaluation during hospitalization.

Category	Test	Result	Reference range
Special Chemistry	CRP	0.5	<1.0 mg/dL
	Vitamin D (25-Hydroxy)	27*	30-100 ng/mL
Urinalysis	Color	Yellow	Yellow
	Clarity	Turbid	Clear
	Glucose	Negative	Negative
	Bilirubin	Negative	Negative
	Ketones	Negative	Negative
	Specific Gravity	1.029	1.005-1.030
	Blood	Negative	Negative
	pH	7	5.0–8.0
	Protein	Trace	Negative–Trace
	Urobilinogen	Normal	0.2-1.0 mg/dL
	Nitrite	Negative	Negative
	Leukocyte Esterase	Negative	Negative
	RBC (Automated)	2	0-3 /hpf
	Mucus	Rare	None–Rare
Immunology / Serology	Anti-GQ1b Antibody	Positive*	Negative
	AChR Binding Ab	<0.30*	<0.24 nmol/L
	AChR Blocking Ab	<1	<15% inhibition
	Centromere Ab	0.7	<0.9 AI
	dsDNA IgG	1.3*	<1.0 IU/mL
	IgA	119.367	33-236 mg/dL
	Jo-1 IgG	0.3	<0.9 AI
	RNP-70	1.6*	<0.9 AI
	SCL-70	1.2*	<0.9 AI
	SM IgG	1.2*	<0.9 AI
	SSA/Ro	0.6	<0.9 AI
	SSB/La	0.5	<0.9 AI
	Striated Muscle Ab	Negative	Negative
	U1-RNP	2.1*	<0.9 AI
CSF Studies	Color	Colorless	Colorless
	Appearance	Clear	Clear
	Glucose	57	40–70 mg/dL
	Lymphocytes (%)	42	40-80%
	Monocytes (%)	34	15-45%
	Neutrophils (%)	23*	0–6%
	Protein	37	15-45 mg/dL
	RBC	2*	0-0 /mm³
	WBC	1	0-5 /mm³

* Indicates value outside the reference range

Table 2: Clinical timeline summarizing symptom progression, diagnostic findings, and management course in a 7-year-old with Miller Fisher syndrome.

Day	Clinical event	Findings	Management
Day 0	URI onset	Cough, congestion, fever	Supportive care
Day 7	Pediatric visit	Right ptosis, left acute otitis media	Amoxicillin started. Referred to Ophthalmology.
Day 8	Evaluated in the emergency department	Bilateral ptosis, ataxia, areflexia	Admitted; MRI, LP, labs
Day 8-11	Hospital course	IVIG administered (2 g/kg total)	PT/OT/SLP initiated
Day 11	Discharge	Improved strength, persistent fatigue	Outpatient rehab
Day 18	Pediatric visit	Congestion, cough, fever (Mycoplasma Pneumoniae)	Azithromycin started
Month 2	Follow-up	60-70% baseline recovery	Continued therapy

At her two-month neurology follow-up, she demonstrated approximately 60-70% improvement in baseline motor function, with residual fatigue on exertion and improving ataxia. Mild clumsiness and balance difficulties persisted, and she occasionally reported having to lift her hip when walking or running. Motor examination showed normal muscle bulk and tone in all extremities and no abnormal movements. Reflexes remained diminished, 1+ graded reflexes in the biceps and triceps and absent in the patellar and Achilles tendons bilaterally, consistent with the understanding that reflexes are typically the last neurological finding to recover, if they recover at all. Continued physical and occupational therapy were recommended, and her recovery remained on a favorable trajectory. Table 2 demonstrates the patient's clinical timeline and hospital course.

Discussion

Pathophysiology

Miller Fisher Syndrome (MFS) is a postinfectious, autoimmune-mediated neuropathy recognized as a rare variant of Guillain-Barré Syndrome (GBS). Its uncommon presentation often contributes to delays in recognition, particularly in pediatric patients. The syndrome is most commonly preceded by infections with *Campylobacter jejuni, Mycoplasma pneumoniae, Epstein–Barr virus,* or *Cytomegalovirus*, among other viral and bacterial pathogens capable of provoking postinfectious autoimmunity [4,5]. Its pathogenesis involves molecular mimicry, with microbial antigens eliciting antibodies that cross react with gangliosides in cranial and peripheral nerves, leading to demyelination and impaired neural transmission [5].

Clinical features and diagnosis

Clinically, these antibodies most commonly target the lower cranial and facial nerves, producing the hallmark triad of ophthalmoplegia, ataxia, and areflexia [1]. Symptoms typically peak within one to two weeks of onset and may include visual disturbances, dysarthria, and paresthesias, particularly involving the third, fourth, and sixth cranial nerves [1]. On examination, patients often exhibit facial paresis, hyporeflexia without upper motor neuron involvement, and loss of dorsal column sensation [6,7].

Anti-GQ1b and anti-GT1a antibodies are detected in over 90% of patients and serve as key diagnostic markers [1,5]. Additional supportive findings may include ventral nerve root enhancement on MRI or albuminocytologic dissociation on cerebrospinal fluid analysis [1]. Nerve conduction studies can further support the diagnosis by demonstrating reduced or absent sensory nerve action potentials with relative preservation of motor responses. Accurate and timely diagnosis is crucial, as recovery timelines in MFS may depend on prompt recognition and early initiation of immunomodulatory therapy.

Management and multidisciplinary care

Treatment is primarily supportive, emphasizing immunomodulation and prevention of complications. Both Intravenous Immunoglobulin (IVIG) and plasma exchange have demonstrated efficacy in accelerating recovery [8]. Severe presentations may also lead to secondary complications such as deep vein thrombosis, respiratory failure, or infection. Adjunctive measures include pain control and prevention of secondary complications [1]. In the present case, IVIG administration and comprehensive multidisciplinary rehabilitation aided in the patient's recovery.

Impact of early recognition and treatment timing

Early recognition and timely initiation of immunotherapy can significantly influence the clinical course of Guillain Barré spectrum disorders. In this case, the patient was initially referred to ophthalmology for acute ptosis, delaying neurological evaluation and treatment by approximately 24 hours. Earlier identification of Miller Fisher syndrome and prompt administration of IVIG might have mitigated symptom progression and hastened recovery [9]. At her two-month neurology follow up, she demonstrated only 60-70% return to baseline function, with persistent areflexia in the lower extremities, diminished upper extremity reflexes, residual fatigue on exertion, and mild balance difficulties.

Given that most pediatric Miller Fisher syndrome cases achieve near complete recovery within one to three months, it is plausible that this brief delay in recognition and treatment may have contributed to her slower neurological improvement and residual deficits. The delay may have allowed ongoing immune mediated demyelination, extending the time required for remyelination and functional recovery [1,10]. While MFS is generally self-limiting, treatment aims to accelerate recovery and prevent neuromuscular respiratory failure. Both plasmapheresis and Intravenous Immunoglobulin (IVIG) have demonstrated comparable efficacy in reducing disease duration.[6] Earlier administration of IVIG may lessen symptom severity and shorten recovery by neutralizing pathogenic antibodies before extensive nerve involvement occurs [7], as early immunotherapy is a known modifiable prognostic factor in Guillain Barré syndrome [9].

While the impact of treatment on recovery remains debated, Mori et al. (2007) reported that IVIG modestly accelerated early improvement in ophthalmoplegia and ataxia. However, overall recovery duration was comparable between treated and untreated patients, likely reflecting the inherently favorable prognosis of MFS [1,10]. In addition, a large retrospective study of 92 patients reported that IVIG modestly improved recovery time by decreasing the binding affinity of anti-GQ1b antibodies and reducing their pathogenic effects [1,11]. Therefore, this case illustrates that even modest delays in diagnosis can influence the early trajectory of recovery. Maintaining a high index of suspicion for MFS in children presenting with acute cranial nerve deficits is essential to expedite neurologic referral and initiate immunotherapy promptly.

Prognosis

Miller Fisher syndrome is generally self-limiting and treatment responsive, with a mortality rate of less than 5% [5,12]. Recovery follows a gradual course, with ataxia resolving within approximately 35 days, ophthalmoplegia improving over about three months, and areflexia showing the greatest variability in duration, ranging from 10 to 650 days [1]. Recurrence of MFS has been reported in some rare cases; therefore, continued monitoring is essential [13].

Overall, the prognosis for pediatric MFS is favorable, with children achieving full recovery within six months [1]. This case underscores that prompt recognition, early immunotherapy, and coordinated multidisciplinary follow-up are critical for minimizing functional impairment and ensuring optimal neurological recovery.

Conclusion

Miller Fisher syndrome is an extremely uncommon, yet important postinfectious neuropathy in children. Although rare in the pediatric population, it should be considered in patients presenting with ophthalmoplegia, ataxia, and areflexia following an upper respiratory infection. Early recognition and rapid initiation of Intravenous Immunoglobulin (IVIG) therapy are key to reducing disease severity and preventing complications. This case emphasizes that even brief diagnostic delays may affect early recovery milestones, reinforcing the importance of clinician awareness, timely diagnosis, and multidisciplinary management to achieve favorable long-term outcomes.

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