Cureus
Part of SPRINGER NATURE

Open Access Case Report

Overlapping Miller Fisher Syndrome and Pharyngeal–Cervical–Brachial Variant of Guillain–Barré Syndrome Following Seasonal Influenza Vaccination: A Case Report

Aslihan Arslan Argin [1], Salma Hammad [2], Doaa Amir [1], Hazim Hamada [3]

1. Respiratory Medicine, Blackpool Teaching Hospitals NHS Foundation Trust, Blackpool, GBR 2. Internal Medicine, Blackpool Teaching Hospitals NHS Foundation Trust, Blackpool, GBR 3. Acute Medicine, Blackpool Teaching Hospitals NHS Foundation Trust, Blackpool, GBR

Corresponding author: Aslihan Arslan Argin, dr.aslihanarslan@gmail.com

Review began 09/29/2026
Review ended 10/29/2026
Published 11/05/2026

© Copyright 2026
Arslan Argin et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119181

Abstract

Guillain-Barré syndrome (GBS) covers a broad spectrum of immune-mediated nerve disorders, including regional subtypes such as Miller Fisher syndrome (MFS) and the pharyngeal-cervical-brachial (PCB) variant. Overlapping features of these variants are recognized but remain uncommon. While it is established that flu vaccination carries a small increased GBS risk, post-vaccination MFS is rare, with overlap presentations being even less frequently reported.

A 28-year-old man with no prior medical issues presented with neurological symptoms six days after receiving a seasonal influenza vaccine. His initial presentation included acute bulbar impairment with dysarthria and dysphagia, marked neck weakness causing head drop, binocular diplopia, and distal paresthesia. Examination revealed bilateral ptosis, facial diplegia, left abducens nerve palsy, hypoglossal weakness, limb and gait ataxia, and generalized areflexia. Limb power remained fully preserved. Brain magnetic resonance imaging showed no acute abnormalities. Cerebrospinal fluid analysis demonstrated albuminocytologic dissociation (protein 0.98 g/L). Nerve conduction studies and electromyography identified severe acute motor denervation involving the cranial, cervical, and proximal upper limb muscles, with preserved sensory responses. These findings supported the diagnosis of overlapping MFS and the PCB variant of GBS. Treatment consisted of intravenous immunoglobulin (2 g/kg administered over five days) together with nasogastric feeding because of severe bulbar dysfunction. He made a gradual but significant recovery, with only mild residual imbalance and lagophthalmos at the two-month follow-up.

This case highlights a rare overlap of MFS and PCB variants presenting shortly after influenza vaccination, although causality cannot be established. This case highlights the need to think of GBS spectrum disorders in patients who have acute bulbar and oculomotor problems, even with preserved limb strength. Early diagnosis is paramount, as it allows for vigilant monitoring and proactive management of life-threatening sequelae, including respiratory insufficiency and aspiration pneumonia.

Categories: Neurology, Public Health, Internal Medicine
Keywords: guillain-barré syndrome, influenza vaccine, miller-fisher syndrome, miller fisher variant, pharyngeal-cervical brachial variant

Introduction

Guillain-Barré syndrome (GBS) is an acute immune-mediated neuropathy of the peripheral nervous system, presenting with rapidly evolving limb weakness, sensory impairment, and diminished or absent deep tendon reflexes. Although the precise pathogenesis remains incompletely understood, current evidence supports an autoimmune mechanism in which antibodies targeting peripheral nerve structures result in either inflammatory or axonal injury [1,2].

GBS comprises several recognized clinical subtypes. Acute motor axonal neuropathy (AMAN), acute inflammatory demyelinating polyneuropathy (AIDP), and acute motor-sensory axonal neuropathy (AMSAN) represent the predominant GBS phenotypes. Less frequently encountered regional variants, including the pharyngeal-cervical-brachial (PCB) and Miller Fisher syndrome (MFS) variant, are also well recognized because of their distinctive clinical manifestations [3].

MFS is a rare GBS variant, with an estimated annual incidence of roughly one to two cases per million people and predominantly affecting males [4]. It is classically defined by the triad of ophthalmoplegia, areflexia, and ataxia. By contrast, the PCB variant is characterized by rapidly advancing dysphagia, neck weakness, oropharyngeal weakness, and proximal upper limb involvement accompanied by upper limb areflexia [5,6].

How to cite this article
Arslan Argin A, Hammad S, Amir D, et al. (November 05, 2026) Overlapping Miller Fisher Syndrome and Pharyngeal–Cervical–Brachial Variant of Guillain–Barré Syndrome Following Seasonal Influenza Vaccination: A Case Report. Cureus 18(11): e119181. DOI 10.7759/cureus.119181

Increasing evidence suggests that these variants exist along a clinical spectrum rather than as distinct entities. Overlapping features between MFS, PCB variant, and other GBS phenotypes have been reported. Sekiguchi et al. demonstrated that overlap syndromes occur in up to 50% of patients with Fisher syndrome, including overlap with PCB variant and classical GBS [7].

The 1976 U.S. influenza vaccination program was associated with a heightened GBS incidence and first raised concerns about a possible link with vaccination; however, seasonal influenza remains a major global health concern, and vaccination is an essential public health measure [2,8,9].

Anti-ganglioside antibodies, particularly anti-GQ1b and anti-GT1a, are associated with MFS and PCB phenotypes, respectively [10-12].

We report a rare case of overlapping MFS and PCB syndrome presenting shortly after seasonal influenza vaccination. We highlighted the diagnostic challenges of overlapping GBS phenotypes and the importance of recognizing significant disease despite preserved limb strength.

Case Presentation

A previously well 28-year-old male received a seasonal influenza vaccination on day 0. He remained asymptomatic for four days. On day 4 post-vaccination, he developed fever, shortness of breath, and flu-like symptoms. Over the next 24 hours, he noted progressive bilateral eyelid swelling, neck weakness with head drop, dysarthria, and dysphagia. He also reported distal paresthesia affecting his hands and feet in a glove-and-stocking distribution, along with constipation. On day 6 post-vaccination, due to worsening bulbar symptoms and difficulty maintaining head posture, he presented to the hospital. Figure *1* illustrates the patient's clinical course from seasonal influenza vaccination (day 0) through symptom onset, hospitalization, intravenous immunoglobulin (IVIG) treatment, and recovery, with follow-up at two months.

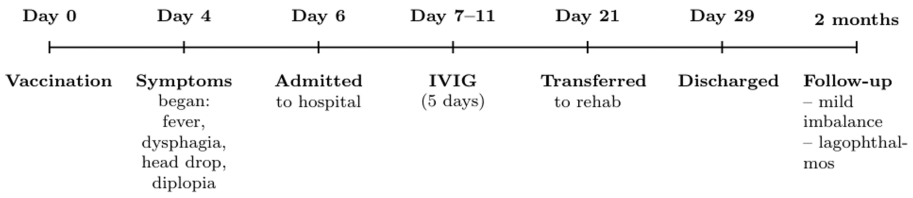


FIGURE 1: Clinical timeline illustrating the patient's clinical course from seasonal influenza vaccination (day 0) through symptom onset, hospitalization, treatment, and recovery.

IVIG, intravenous immunoglobulin

On admission, he was hemodynamically stable with a National Early Warning Score (NEWS) of 3. Oxygen saturation was 96% on room air, respiratory rate was 24 breaths/min, heart rate was 103 beats/min, and blood pressure was 128/86 mm Hg. He was afebrile at 36.8°C. There was no clinical evidence of respiratory distress. Neurological examination demonstrated bilateral ptosis, facial diplegia, dysarthria, and dysphagia. There was marked neck flexion weakness resulting in head drop. Limb power was preserved at Medical Research Council grade 5/5 throughout both upper and lower limbs, although he was initially unable to mobilize independently due to axial weakness and ataxia. Deep tendon reflexes were absent throughout. Light touch and pinprick sensation were preserved, though he described subjective distal paresthesia. Proprioception was intact. Mild limb ataxia was present. Cranial nerve examination revealed a left abducens nerve palsy with diplopia on lateral gaze and a milder contralateral deficit. Pupillary reflexes were normal. Tongue protrusion was incomplete with rightward deviation, suggestive of hypoglossal involvement.

Given the rapid progression of bulbar dysfunction and the potential risk of respiratory compromise, he was admitted to the intensive care unit for close monitoring. Mechanical ventilation and inotropic support were not required at any stage. Chest radiography demonstrated no acute pathology. Computed tomography of the head was normal.

Magnetic resonance imaging of the brain was initially reported as showing mild ventricular enlargement; however, specialist neuroradiology review confirmed that this represented flow artefact, and no structural abnormality was identified. Lumbar puncture performed on day 8 demonstrated albuminocytologic dissociation, with cerebrospinal fluid protein elevated at 0.98 g/L and a white cell count of 2 cells/mm^3. Serum immunoglobulin G was 21 g/L. Anti-acetylcholine receptor and anti-muscle-specific kinase antibodies were negative. Anti-glycolipid antibody testing was requested; however, the result was never

obtained.

Given the pattern of ophthalmoplegia, areflexia, bulbar involvement, and preserved limb strength, a Guillain-Barré spectrum disorder was suspected, most consistent with a PCB variant with overlapping Miller Fisher features. IVIG was commenced on day 7 at a total dose of 2 g/kg administered over five days.

Following speech and language therapy assessment, nasogastric feeding was initiated due to unsafe swallowing. Gabapentin was prescribed for neuropathic pain. He remained hemodynamically stable throughout his intensive care admission.

Gradual neurological improvement was observed over the subsequent days. Neck flexion strength improved first, followed by recovery of swallowing function. The nasogastric tube was removed once adequate oral intake was achieved. Eye protection measures were implemented due to lagophthalmos.

Neurophysiological studies performed on day 29 demonstrated normal sensory nerve conduction studies in both upper and lower limbs. Motor conduction studies showed normal distal latencies, amplitudes, and conduction velocities, with preserved F-wave latencies. Repetitive nerve stimulation of the facial nerves bilaterally and the right spinal accessory nerve revealed no significant decrement, with no electrophysiological evidence of a neuromuscular junction disorder.

Concentric needle electromyography demonstrated severe acute motor denervation in bilateral facial muscles, with positive sharp waves and fibrillation potentials. No voluntary motor unit activity was detected in the frontalis, orbicularis oculi, or orbicularis oris muscles. Acute denervation changes were also observed in the trapezius, deltoid, biceps, triceps, and extensor digitorum communis bilaterally, with symmetrically reduced recruitment patterns. Distal hand muscles were relatively preserved. Sensory responses remained intact. These findings supported a predominantly motor neuropathy affecting cranial, cervical, and brachial musculature, consistent with a Guillain-Barré spectrum disorder involving the PCB region. However, consultant neurophysiologist acknowledged significant involvement of the brachial, cervical, and facial muscles, making the clinical presentation challenging to distinguish from MFS. Given the presence of diplopia and marked ptosis, the findings were also consistent with MFS. Consultant's overall impression was that the presentation represented an overlap of GBS variants. Electromyography findings are presented in Table *1*.

Muscle	Spontaneous Activity	Motor Unit Potentials	Recruitment
Cranial muscles			
Frontalis	PSW ++, Fib ++	None detected	Absent
Orbicularis oculi	PSW ++, Fib ++	None detected	Absent
Orbicularis oris	PSW ++, Fib ++	None detected	Absent
Cervical muscles			
Trapezius	PSW +, Fib +	Reduced	Severely reduced
Upper limb muscles			
Deltoid	PSW +, Fib +	Reduced	Severely reduced
Biceps brachii	PSW +, Fib +	Reduced	Severely reduced
Triceps brachii	PSW +, Fib +	Reduced	Severely reduced
EDC	PSW +, Fib +	Reduced	Severely reduced
Distal hand muscles			
FDI, APB	None	Normal	Normal
Sensory studies			
All sensory studies	All normal	-	-

TABLE 1: Electromyography findings.

Concentric needle electromyography demonstrated severe acute motor denervation in cranial, cervical, and proximal upper limb muscles, with relative sparing of distal hand muscles. Sensory nerve conduction studies were normal throughout.

PSW, positive sharp waves; Fib, fibrillation potentials; EDC, extensor digitorum communis; FDI, first dorsal interosseous; APB, abductor pollicis brevis

He was transferred to a neuro-rehabilitation unit on day 21. At the time of discharge from rehabilitation on day 29, neck flexion strength had returned to normal, ptosis had significantly improved, and dysphagia had resolved. He was mobilizing independently.

At follow-up approximately two months later, he reported significant overall improvement. He remained independently mobile but described mild residual imbalance and persistent unilateral eyelid and facial muscle weakness. Ophthalmology review confirmed bilateral lagophthalmos, and conservative management was advised.

Discussion

This report describes a 28-year-old man with no prior medical issues who developed overlapping MFS and the PCB variant of GBS within one week of seasonal influenza vaccination. Given the limited number of similar reports, this case adds to the literature describing MFS-PCB overlap following influenza vaccination and reinforces the need to recognize overlapping GBS phenotypes after recent immunization.

Clinical presentation and diagnostic considerations

The patient presented with the classic clinical features of MFS, including ophthalmoplegia, ataxia, and areflexia, in addition to prominent bulbar dysfunction, neck weakness, and proximal upper limb involvement characteristic of the PCB variant. Patients with MFS classically exhibit ophthalmoplegia, areflexia, and ataxia, while those with the PCB variant present with pharyngeal and facial weakness. MFS is an uncommon disorder, occurs at a rate of roughly 1-2 per 1,000,000 population, and occurs more frequently in males [10].

The initial differential diagnosis included brainstem stroke, myasthenia gravis, and botulism. The diagnostic process can be complicated in such cases, frequently necessitating further testing to refine the differential diagnosis, especially during the initial phase of presentation [11]. This diagnostic challenge is well-recognized in the literature, as the descending pattern of weakness in the PCB variant can closely mimic botulism [6]. It is common for PCB patients to be mistakenly diagnosed with brainstem stroke, myasthenia

gravis, or botulism at first. In most cases, a careful review of the patient's history and a thorough physical examination are enough to exclude these other conditions [6].

Our patient exhibited cranial nerve involvement beyond the typical oculomotor nerves, including facial diplegia, hypoglossal weakness, and bulbar dysfunction. In MFS-GBS overlap syndromes, cranial nerve involvement extends beyond the typical oculomotor nerves more often than not: the facial nerve is affected in 30-50% of cases, while the glossopharyngeal (IX), vagus (X), and hypoglossal (XII) nerves, which mediate swallowing and phonation, are involved in 13% of cases [11]. It is uncommon for patients with MFS to present with nasal speech due to palatal paresis, with only sparse reports in the literature [11]. Furthermore, dysphagia was found in only 2% of patients in a large Japanese series of 50 MFS patients [11].

Overlap syndromes in the GBS spectrum

The coexistence of MFS and PCB features in our patient supports the concept of GBS as a continuous spectrum rather than a collection of isolated syndromes [10]. Cases demonstrating overlapping features of MFS and PCB variants are uncommon but clinically significant [10]. Overlap between GBS variants is frequently encountered in clinical practice. In a study by Sekiguchi et al., such overlaps were identified in 50% of patients with MFS, 23% overlapped with the PCB variant, and 15% with conventional GBS [7].

A growing body of evidence from clinical, radiological, immunological, and electrophysiological studies supports the classification of GBS, MFS, and Bickerstaff brainstem encephalitis as interconnected disorders within the anti-GQ1b IgG antibody syndrome [12]. This concept of an anti-GQ1b antibody syndrome helps explain the overlapping features observed in our patient.

Diagnostic findings

The classic cerebrospinal fluid analysis demonstrated albuminocytologic dissociation, with a normal white cell count and cerebrospinal fluid protein elevated at 0.98 g/L, supporting the diagnosis [11].

The neurophysiological and electromyography findings demonstrated severe acute motor denervation in cranial, cervical, and brachial musculature with preserved sensory responses. According to serial nerve conduction studies, PCB is a localized GBS variant distinguished by axonal neuropathy rather than demyelination [6]. This axonal predominance was also reflected in the neurophysiological findings of our patient, aligning with the established pattern.

Temporal association with influenza vaccination

The onset of neurological symptoms four to six days following seasonal influenza vaccination in our patient raises the question of a potential causal association. Co-administration of the seasonal influenza vaccine and the novel influenza A (H1N1) vaccine was followed 13 days later by the onset of MFS in a patient, as documented in one case report [13]. In keeping with our patient's presentation, all prior cases of post-vaccination MFS have occurred within 14 days of vaccination and have shown clinical improvement following treatment with IVIG [13].

Applying the World Health Organization's criteria for causality assessment, the association between vaccine administration and disease development in some patients has been classified as "very likely" [13]. However, these observations do not establish causality in the present case, given the limitations inherent in a single case report.

Epidemiological context

The link between influenza vaccination and GBS has been under scrutiny ever since the 1976 swine influenza campaign. That U.S. immunization program was suspended due to reports of vaccine-associated GBS [9]. This translated to an attributable risk of about one additional GBS case per 100,000 people vaccinated, with the vaccine conferring a four- to eight-fold elevated risk within six weeks of administration [9].

Subsequent epidemiological studies have demonstrated a modest but statistically significant elevation in GBS risk following influenza vaccination, whether seasonal or pandemic. For seasonal vaccines, however, the increase is only marginally significant [14]. Pandemic influenza vaccines carried a greater GBS risk (RR = 1.84; 95% CI, 1.36-2.50) than seasonal formulations (RR = 1.22; 95% CI, 1.01-1.48), according to a meta-analysis by Martín Arias et al., which reported an overall relative risk of 1.41 (95% CI, 1.20-1.66) following influenza vaccination [14].

Importantly, the risk posed by influenza infection itself is considerably greater than that linked to vaccination. Following an influenza-coded healthcare visit, the relative incidence of GBS within six weeks was 15.81 (95% CI 10.28-24.32), a figure significantly higher than that associated with vaccination [15]. The attributable risk of GBS was also substantially greater after influenza illness (17.2 admissions per million encounters) than after vaccination (1.03 admissions per million vaccinations) [15]. Although both influenza vaccines and influenza infection carry small attributable risks, the risk associated with the infection far

exceeds that of the vaccine [15].

Pathophysiological mechanisms

The pathogenesis of GBS and its variants is thought to involve molecular mimicry, particularly in relation to antecedent infections. Molecular mimicry is considered the most plausible mechanism through which infectious agents provoke an immune response [12]. This process constitutes a key pathogenic pathway, and its deeper understanding could carry significant therapeutic implications [12].

In the context of vaccination, similar mechanisms may be operative. Studies have shown that influenza vaccines can raise autoantibody levels in humans [13]. For instance, several influenza vaccines, including the 1976 H1N1 vaccine, caused experimental allergic neuritis-like illness in rabbits [13]. Furthermore, the 1976 H1N1 vaccine could stimulate anti-GM1 antibodies in mice without the presence of anti-Campylobacter jejuni antibodies, and this effect was also seen with seasonal flu vaccines [13].

The anti-GQ1b antibody, which is strongly associated with MFS, targets gangliosides concentrated in the oculomotor nerves and neuromuscular junctions. The GQ1b ganglioside is found in high concentrations within the cranial nerves innervating the extraocular muscles, as well as at the presynaptic neuromuscular junction, a site that lacks a blood-nerve barrier. This may make it susceptible to autoimmune attack [12]. Although the anti-GQ1b antibody result was never obtained, the clinical phenotype strongly suggested an anti-GQ1b spectrum disorder. Also potentially relevant are anti-GT1a antibodies, which are frequently identified in PCB patients and correlate with bulbar palsy in up to 70% of GBS cases [11]. Owing to cross-reactivity, these antibodies commonly co-exist with anti-GQ1b antibodies, and their concurrent detection further bolsters the overlap hypothesis [11].

Treatment and outcome

Our patient received IVIG at a total dose of 2 g/kg over five days, with subsequent gradual neurological improvement. In one series, three of five patients with post-vaccination GBS variants had a favorable outcome after one course of IVIG infusion [16]. Another case report described a patient treated with plasmapheresis over five sessions who was successfully extubated on the third day of hospital stay and discharged after 15 days without apparent sequelae [17].

Ongoing assessment of bulbar function and respiratory effort is mandatory in hospital, informing decisions about nasogastric feeding and ventilator support when needed. This is because bulbar weakness can progress quickly, making prompt diagnosis of PCB and exclusion of other treatable causes essential [6]. Our patient required intensive care monitoring and nasogastric feeding due to bulbar dysfunction, though mechanical ventilation was not ultimately necessary.

At the two-month follow-up, our patient demonstrated significant improvement but had residual mild imbalance and persistent unilateral lagophthalmos. In earlier reports, some post-vaccination cases had incomplete recovery, though this finding may be attributable to limited follow-up periods [13]. By six months post-onset, two patients in a larger atypical GBS series had recovered to their baseline activities, and one patient was near that level of function [16].

Strengths and limitations

This case report has several strengths, including detailed clinical characterization, comprehensive neurophysiological assessment, and clear documentation of the temporal relationship with vaccination. However, there are limitations. Anti-ganglioside antibody results remained pending at the time of discharge and could not subsequently be obtained because the samples were no longer available, though the clinical phenotype was strongly supportive of the diagnosis. In hospital settings where antibody testing is not readily accessible, the detection of complex anti-ganglioside antibodies may assist in diagnosing cases where alternative differentials remain unresolved [17]. Additionally, a single case cannot establish causality, and the association with vaccination may be coincidental given the background incidence of GBS.

Conclusions

This case represents a rare occurrence of overlapping MFS and PCB variant GBS presenting shortly after seasonal influenza vaccination. It reinforces the concept of GBS as a continuous spectrum disorder where regional variants can coexist, creating diagnostically challenging clinical pictures. The temporal association with vaccination, while not proof of causation, adds to the limited literature on post-vaccination MFS and highlights the importance of considering immune-mediated complications in the differential diagnosis of patients presenting with acute cranial nerve and bulbar deficits, even when limb strength is preserved. This case serves as a critical reminder for clinicians that early recognition of these atypical variants is paramount to ensure timely monitoring and management of life-threatening bulbar and respiratory complications.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Aslihan Arslan Argin, Salma Hammad, Doaa Amir, Hazim Hamada

Acquisition, analysis, or interpretation of data: Aslihan Arslan Argin, Salma Hammad, Doaa Amir

Drafting of the manuscript: Aslihan Arslan Argin, Salma Hammad, Doaa Amir

Critical review of the manuscript for important intellectual content: Aslihan Arslan Argin, Salma Hammad, Doaa Amir, Hazim Hamada

Supervision: Hazim Hamada

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. van Doorn PA, Ruts L, Jacobs BC: Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome. Lancet Neurol. 2008, 7:939-50. 10.1016/S1474-4422(08)70215-1
2. Bae JS, Yuki N, Kuwabara S, Kim JK, Vucic S, Lin CS, Kiernan MC: Guillain-Barré syndrome in Asia. J Neurol Neurosurg Psychiatry. 2014, 85:907-13. 10.1136/jnnp-2013-306212
3. Wakerley BR, Yuki N: Mimics and chameleons in Guillain-Barré and Miller Fisher syndromes. Pract Neurol. 2015, 15:90-9. 10.1136/practneurol-2014-000937
4. Willison HJ, Jacobs BC, van Doorn PA: Guillain-Barré syndrome. Lancet. 2016, 388:717-27. 10.1016/S0140-6736(16)00339-1
5. van den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, van Doorn PA: Guillain-Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. Nat Rev Neurol. 2014, 10:469-82. 10.1038/nrneurol.2014.121
6. Wakerley BR, Yuki N: Pharyngeal-cervical-brachial variant of Guillain-Barre syndrome. J Neurol Neurosurg Psychiatry. 2014, 85:339-44. 10.1136/jnnp-2013-305397
7. Sekiguchi Y, Mori M, Misawa S, Sawai S, Yuki N, Beppu M, Kuwabara S: How often and when Fisher syndrome is overlapped by Guillain-Barré syndrome or Bickerstaff brainstem encephalitis?. Eur J Neurol. 2016, 23:1058-63. 10.1111/ene.12983
8. World Health Organization. Influenza (Seasonal) [Internet]. (2026). Accessed: March 16, 2026: https://www.who.int/news-room/fact-sheets/detail/influenza-(seasonal).
9. Schonberger LB, Bregman DJ, Sullivan-Bolyai JZ, et al.: Guillain-Barre syndrome following vaccination in the National Influenza Immunization Program, United States, 1976--1977. Am J Epidemiol. 1979, 110:105-23. 10.1093/oxfordjournals.aje.a112795
10. Kumar A, Garg S, Bharti P, Zutshi B: Double trouble: Guillain-Barré syndrome (GBS) presenting as overlapping Miller Fisher Syndrome (MFS) and pharyngeal-cervical-brachial (PCB) variant. Cureus. 2025, 17:e98367. 10.7759/cureus.98367
11. Tamaoui L, Rahmani M, Touati H, Benabdeljlil M, Aidi S, Erguig L: Pharyngo-cervico-brachial variant of Guillain-Barré or Miller Fisher syndromes when the overlap is misleading. Neurol Int. 2020, 12:52-6. 10.3390/reports3040034
12. Lo YL: Clinical and immunological spectrum of the Miller Fisher syndrome. Muscle Nerve. 2007, 36:615-27. 10.1002/mus.20835
13. Shoamanesh A, Chapman K, Traboulsee A: Postvaccination Miller Fisher syndrome. Arch Neurol. 2011, 68:1327-9. 10.1001/archneurol.2011.236
14. Martín Arias LH, Sanz R, Sáinz M, Treceño C, Carvajal A: Guillain-Barré syndrome and influenza vaccines: a meta-analysis. Vaccine. 2015, 33:3773-8. 10.1016/j.vaccine.2015.05.013
15. Kwong JC, Vasa PP, Campitelli MA, et al.: Risk of Guillain-Barré syndrome after seasonal influenza vaccination and influenza health-care encounters: a self-controlled study. Lancet Infect Dis. 2013, 13:769-76. 10.1016/S1473-3099(13)70104-X
16. Shaikh AG, Termsarasab P, Nwankwo C, Rao-Frisch A, Katirji B: Atypical forms of Guillain-Barré syndrome and H1N1-influenza vaccination. Vaccine. 2012, 30:3251-4. 10.1016/j.vaccine.2012.03.013
17. Monge RA, Ocegueda T: An atypical case of Guillain-Barré: Overlap of Miller Fisher syndrome with pharyngeal-cervical-brachial variant. Front Emerg Med. 2022, 10:412. 10.18502/fem.v6i3.9404