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Bilateral Lower Motor Neuron Facial Palsy in a Mild AIDP Variant of Guillain–Barré Syndrome: A Case Report

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ABSTRACT

Introduction: Guillain–Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness, areflexia, and variable sensory disturbances. Although cranial nerve involvement occurs in a subset of patients, bilateral lower motor neuron (LMN) facial palsy as a prominent feature is considered an uncommon variant. Early recognition and appropriate monitoring are essential to prevent respiratory or autonomic complications.

Case Presentation: A 37-year-old woman presented with a 16-day history of progressive paresthesia in the extremities accompanied by bilateral LMN facial palsy and mild lower limb weakness. Neurological evaluation showed decreased deep tendon reflexes, glove-stocking sensory disturbance, and preserved respiratory function. Cerebrospinal fluid analysis revealed albuminocytologic dissociation, and electrophysiological studies demonstrated demyelinating motor polyradiculoneuropathy consistent with the acute inflammatory demyelinating polyneuropathy (AIDP) variant of GBS. The patient showed continuous clinical improvement with supportive management, without requiring immunotherapy.

Discussion: This case represents an uncommon cranial-nerve–predominant form of GBS in which bilateral facial palsy appeared as the dominant manifestation despite mild limb involvement. The patient's favorable clinical trajectory, along with improving motor and sensory deficits, aligns with literature indicating that mild AIDP cases may recover well with conservative therapy alone. This highlights the importance of individualized assessment, as immunotherapy may be unnecessary in patients who remain ambulatory and exhibit early spontaneous improvement.

KEYWORDS: Guillain–Barré Syndrome, Facial Diplegia, AIDP, Demyelinating Polyradiculoneuropathy, Case Report.

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INTRODUCTION

Guillain-Barré Syndrome (GBS) is an acute-onset immune-mediated neuropathy and remains the most common cause of acute flaccid paralysis worldwide.[1] Variants include acute inflammatory demyelinating polyneuropathy (AIDP), axonal forms, and cranial nerve–predominant forms. Bilateral facial palsy occurs in 27–50% of GBS cases, but isolated or prominent facial diplegia with mild limb weakness remains uncommon. Early recognition and monitoring for respiratory involvement are essential.[1,2]

CASE REPORT

A 37-year-old woman presented with a four-day history of progressive facial weakness and sensory symptoms. The symptoms began with a thickened sensation around the mouth, followed by stiffness of the right side of the face, and shortly thereafter the left side became similarly affected. She reported difficulty wrinkling her forehead, with the weakness persisting since its first day onset. Two days later, she developed generalized body aches predominantly in both hands and feet, beginning with pain around the ankles and the soles of the feet, which then progressed to involve the hands up to the wrists accompanied by intermittent numbness. Despite these symptoms, she remained able to eat and drink without choking, although she noted frequent abdominal fullness. She also reported mild headaches, with an NRS score of 2–3, but denied blurred or double vision. However, since 4 days of onset, she had been unable to fully close both eyes. She denied shortness of breath, cough, fever, diarrhea, or other systemic symptoms.

Her past medical history was unremarkable except for hyperuricemia diagnosed one month prior. She denied diabetes mellitus, hypertension, similar prior episodes, stroke, tumors, seizures, or other neurological disorders. She was not on any routine medications. The progression of bilateral facial weakness, ascending limb discomfort, and sensory disturbances raised clinical suspicion for a cranial-nerve–predominant variant of Guillain–Barré Syndrome.

On examination, the patient was alert, fully oriented, and cooperative with a Glasgow Coma Scale score of 15 (E4V5M6). Her vital signs showed blood pressure of 126/87 mmHg, tachycardia with a heart rate of 107 beats per minute, respiratory rate of 20 breaths per minute, oxygen saturation of 100% on room air, and a temperature of 37.0°C. There were no signs of meningeal irritation, with both neck stiffness and Brudzinski's signs I–IV testing negative. Cranial nerve evaluation revealed intact visual

acuity and pupillary responses, as well as normal extraocular movements; however, bilateral lower motor neuron facial palsy was evident, while tongue movements remained normal.

Motor examination demonstrated mild weakness, predominantly in the proximal lower limbs, with muscle strength ranging from 4+ to 5/5. Sensory testing showed glove-stocking–type paresthesia involving both the upper and lower extremities. Deep tendon reflexes were globally diminished (+1/+1), while pathological reflexes such as Babinski, Chaddock, and Hoffman–Tromner were absent. Coordination and cerebellar function were intact, and autonomic parameters appeared stable. The Wartenberg test was negative bilaterally. Respiratory assessment showed preserved function, with a Single Breath Counting value of 26, indicating no immediate respiratory compromise.

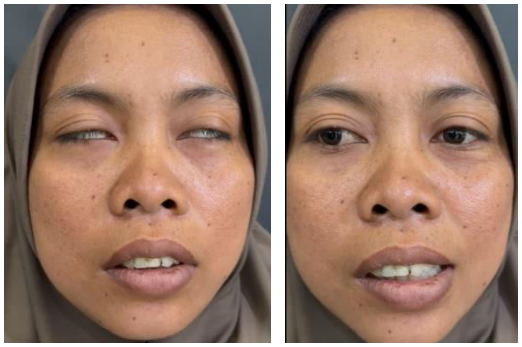

Figure 1. Bilateral Facial Palsy during Physical Examination

Laboratory evaluation revealed mild leukocytosis with a white blood cell count of 12.92 × 10^3/μL, while hemoglobin and platelet count were within normal range. Electrolytes, renal function, coagulation parameters, and inflammatory markers were unremarkable, excluding metabolic or infectious mimics of weakness.

Cerebrospinal fluid (CSF) analysis demonstrated albuminocytological dissociation, a classic feature of GBS, with an elevated protein of 221 mg/dL and an almost normal cell count of 0.01/μL. CSF glucose was normal relative to simultaneous blood glucose.

Cervical spine radiographs and chest imaging showed no abnormalities. On further evaluation, nerve conduction study (NCS) and electromyography were performed on 20 October 2025. Sensory nerve action potentials (SNAP) of the right and left sural nerves showed normal responses, indicating relative preservation of sensory fibers. In contrast, motor conduction studies demonstrated marked abnormalities: the right peroneal nerve exhibited prolonged distal latency, conduction block, reduced nerve conduction velocity, and low-amplitude compound muscle action potentials (CMAPs), while the left peroneal nerve showed prolonged distal latency with decreased conduction velocity and low-amplitude CMAPs. Bilateral tibial nerves also demonstrated prolonged distal latency with low-amplitude CMAPs. F-wave studies of the right and left tibial nerves revealed absent responses, and H-reflex recordings from the bilateral gastrocnemius–soleus (G–S) pathways were also absent. These findings were consistent with a demyelinating motor polyradiculoneuropathy pattern, supporting the diagnosis of Guillain–Barré syndrome of the AIDP variant.

The patient received intravenous hydration, parenteral neurotropic vitamins, analgesics as needed, and antihypertensive therapy (candesartan and carvedilol). The patient was hospitalized for a total of five days for close neurological and respiratory monitoring. During the course of hospitalization, her symptoms showed gradual and continuous improvement. The bilateral facial weakness, which initially prevented full eye closure, progressively recovered; by the fifth day of treatment, the patient was able to close both eyes completely without lagophthalmos. Limb paresthesia also diminished, and motor strength remained stable with no evidence of further progression. Throughout admission, there were no signs of respiratory decline, autonomic instability, or new neurological deficits, supporting a favorable clinical trajectory consistent with a mild AIDP variant of Guillain–Barré Syndrome. The patient was subsequently deemed fit for discharge with continued outpatient follow-up.

DISCUSSION

This case demonstrates an atypical phenotype of Guillain–Barré Syndrome (GBS), characterized by prominent bilateral lower motor neuron facial palsy with relatively mild limb weakness—an uncommon pattern within the AIDP spectrum as described in major epidemiological studies.[1] Bilateral facial diplegia is reported in a significant subset of GBS cases, yet it rarely presents as the predominant neurological deficit, often creating diagnostic uncertainty with other causes of facial palsy such as myasthenia gravis, sarcoidosis, or pontine lesions.[1] The classical combination of glove-stocking paresthesia, areflexia, and albuminocytological dissociation in cerebrospinal fluid observed in this patient aligns with well-established diagnostic features of AIDP, as widely outlined in contemporary reviews.[3,4] Neurophysiological evidence of demyelinating motor polyradiculoneuropathy further corroborates the diagnosis, consistent with patterns observed across electrophysiological studies in GBS variants.[4]

Current literature emphasizes that treatment selection in GBS must be guided by severity, progression, and functional status rather than the mere presence of cranial nerve involvement.[1,3] Immunotherapy—either IVIG or plasma exchange—is recommended primarily for patients who are non-ambulatory, exhibit rapid progression, or possess bulbar or respiratory compromise, while its

benefit in mild or improving cases remains limited.[1] The patient presented here retained independent ambulation (Hughes score 2), had preserved respiratory function, and demonstrated early neurological improvement, making conservative therapy an evidence-based and appropriate choice, supported by observational data showing that mild AIDP cases frequently recover without IVIG or plasmapheresis.[2,4] Continuous monitoring, particularly during the first four weeks when deterioration is most likely, remains essential to ensure timely escalation should progression occur.[1]

The favorable clinical trajectory in this case is in line with outcomes reported for mild GBS variants with cranial nerve involvement, where supportive management alone—such as neuropathic pain control, vitamin supplementation, autonomic monitoring, and guided physiotherapy—has been shown to facilitate steady recovery.[2,4] Importantly, bilateral facial palsy, despite its alarming appearance, does not independently predict poor prognosis and should not influence treatment decisions in the absence of other severity markers.[3,5] This case reinforces the importance of individualized clinical judgment and highlights that selective use of immunotherapy is both safe and rational when guided by established prognostic criteria and current evidence-based guidelines.[1,5]

CONCLUSION

This case illustrates an uncommon variant of Guillain–Barré Syndrome characterized by prominent bilateral facial palsy with mild limb weakness and preserved respiratory function. Early improvement, supportive findings on CSF and electrophysiology, and stable clinical status indicated a mild AIDP course. Consistent with current evidence, conservative management without immunotherapy was appropriate and resulted in favorable recovery. This case reinforces the importance of individualized assessment and close monitoring in managing mild GBS variants.

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