

## Case Report

# Bifacial Weakness with Paresthesia: A Rare Regional Variant of Guillain-Barré Syndrome

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## Abstract

Guillain-Barré Syndrome (GBS) is an acute, immune-mediated polyneuropathy often preceded by an infection characterized by ascending muscle weakness, hyporeflexia or areflexia, and variable sensory disturbances. While the classical GBS phenotype is characterized by rapidly progressive bilateral limb weakness with hyporeflexia or areflexia, electrophysiological subtypes include Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and axonal forms such as Acute Motor Axonal Neuropathy (AMAN) and Acute Motor and Sensory Axonal Neuropathy (AMSAN); the clinical spectrum, however, includes several distinct regional and functional variants. Cranial nerve involvement, particularly facial weakness, is well recognised in GBS.

**Keywords:** Guillain-Barré syndrome; Facial diplegia; Facial diplegia with paresthesia; Acute peripheral polyneuropathy

## Introduction

Guillain-Barré Syndrome (GBS) is an acute, immune-mediated polyneuropathy often preceded by an infection characterized by ascending muscle weakness, hyporeflexia or areflexia, and variable sensory disturbances. While the classical GBS phenotype is characterized by rapidly progressive bilateral limb weakness with hyporeflexia or areflexia, electrophysiological subtypes include Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and axonal forms such as Acute Motor Axonal Neuropathy (AMAN) and Acute Motor and Sensory Axonal Neuropathy (AMSAN); the clinical spectrum, however, includes several distinct regional and functional variants. Cranial nerve involvement, particularly facial weakness, is well recognised in GBS [1]. GBS variants with isolated Bifacial Weakness with Paresthesias (BFWP) also referred to as the Facial Diplegia with Paresthesias (FDP) variant represent a rare regional phenotype. Patients with BFWP present with rapidly progressive bilateral Lower Motor Neuron (LMN) facial weakness accompanied by distal sensory symptoms in the limbs, but lack significant limb weakness, ataxia, or ophthalmoplegia [2]. Because peripheral motor power is preserved, diagnosis is frequently delayed or misattributed to idiopathic bilateral Bell's palsy, Lyme disease, or brainstem pathology [3]. Recognizing BFWP as a localized subtype of GBS is essential, as early initiation of immunotherapy can accelerate recovery and prevent potential secondary progression. Here, we present a clinical case of BFWP, detailing its electrodiagnostic characteristics, Cerebrospinal

Fluid (CSF) profile, diagnostic workflow, and clinical response to Intravenous Immunoglobulin (IVIG).

## Case Presentation

A 50-year-old woman presented to the emergency department with a history of tingling sensation of both hands for 20 days. On day 5 of disease onset, she noted tingling sensation of both feet. The patient was able to appreciate hot and cold temperature on both hands and had no difficulty in performing her daily activities. On day 18 after symptom onset, she noted difficulty closing both eyes, associated with difficulty pursing her lips. She also gave a history of dribbling of saliva from the sides of the mouth. Tearing and taste sensation were preserved. She denied any history of weakness in the limbs, gait imbalance, bowel or bladder disturbances. There was no history of similar complaints in the past. She had received her first dose of COVID-19 vaccination two months prior; however, no temporal association was established. There was no history of recent travel, infection, or trauma.

On examination, her pulse was 60 beats/min, regular, and normal in volume. Her blood pressure was 157/94 mmHg in supine and sitting posture. Her respiratory rate was 14/minute, oxygen saturation was 96% on room air, and she was afebrile. Neurological examination revealed bilateral lower motor neuron type of facial nerve palsy, difficulty frowning, smiling, pursing her lips, or closing her eyes tightly. Other cranial nerve examinations were unremarkable. Her motor system examination, including muscle bulk, tone, and power, was normal. Deep tendon reflexes were present except for diminished ankle reflexes. Her plantar reflexes were flexor bilaterally. On sensory examination, decreased touch and pain sensation was noted over the medial aspect of both hands up to the wrists, also in the medial aspect of the dorsum of the feet however, joint position and vibration senses were normal. Other systemic examinations revealed no abnormalities.

Initial investigations, including complete blood count, ESR, serum electrolytes, renal and liver function tests, were normal. Serology was negative. ANA and ANCA were negative, summarised in Table 1. Nerve conduction studies were performed, motor nerve

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conduction studies (Table 2) demonstrate prolonged distal latencies in the bilateral median, peroneal, and tibial nerves, reduced conduction velocities in selected nerves. Bilateral facial motor responses from the orbicularis oculi and nasalis muscles show reduced CMAP amplitudes. Sensory nerve conduction studies (Table 3) demonstrate absent bilateral median sensory nerve action potentials, with preserved bilateral ulnar, sural, and superficial peroneal sensory responses. Lumbar puncture was performed, showed a cell count of 5 cells/mm<sup>3</sup>, CSF protein was 79 mg/dL, and glucose was 56 mg/dL. CSF was negative for MTB complex, and cultures showed no growth. The combination of progressive distal paresthesias, bilateral lower motor neuron facial weakness, diminished ankle reflexes, peripheral nerve conduction abnormalities with facial nerve involvement, and CSF albuminocytological dissociation supported a diagnosis of BFWP, a regional variant of GBS. The patient was treated with Intravenous Immunoglobulin (IVIG) at a dose of 0.4 g/kg/day over 5 days with adequate hydration and other supportive treatment. Swallow therapy and facial physiotherapy were also initiated. The patient's symptoms improved by day 4 of IVIG. Facial weakness improved, and she could retain 2-3 spoonfuls of liquid in her mouth; her paresthesias were reduced significantly by completion of the IVIG dose.

**Table 1:** Initial laboratory investigations.

| Investigations             | Patient Value                           | Reference Range                                |
|----------------------------|-----------------------------------------|------------------------------------------------|
| Haemoglobin                | 13.2 g/dL                               | 12.0 - 15.0 g/dL                               |
| Total Leukocyte Count      | 7,860 /mm <sup>3</sup>                  | 4,000 - 11,000 /mm <sup>3</sup>                |
| Platelet Count             | 2.47 × 10 <sup>5</sup> /mm <sup>3</sup> | 1.50 - 4.50 × 10 <sup>5</sup> /mm <sup>3</sup> |
| ESR                        | 12                                      | 0-20                                           |
| Serum Urea                 | 24 mg/dL                                | 15 - 40 mg/dL                                  |
| Serum Creatinine           | 1.04 mg/dL                              | 0.70 - 1.20 mg/dL                              |
| Serum Uric Acid            | 7.1 mg/dL                               | 3.5 - 7.2 mg/dL                                |
| Sodium                     | 138 mmol/L                              | 136 - 145 mmol/L                               |
| Potassium                  | 4.6 mmol/L                              | 3.5 - 5.1 mmol/L                               |
| Calcium                    | 9.6 mg/dL                               | 8.5 - 10.5 mg/dL                               |
| Magnesium                  | 1.9 mg/dL                               | 1.7 - 2.2 mg/dL                                |
| Total Bilirubin            | 0.8 mg/dL                               | 0.2 - 1.2 mg/dL                                |
| Direct Bilirubin           | 0.04 mg/dL                              | 0.00 - 0.30 mg/dL                              |
| AST                        | 24 U/L                                  | 10 - 40 U/L                                    |
| ALT                        | 22 U/L                                  | 7 - 56 U/L                                     |
| GGT                        | 76 U/L                                  | 44 - 147 U/L                                   |
| Total Protein              | 7.2 g/dL                                | 6.0 - 8.3 g/dL                                 |
| Albumin                    | 4.2 g/dL                                | 3.5 - 5.0 g/dL                                 |
| Random Blood Glucose       | 94 mg/dL                                | 70 - 140 mg/dL                                 |
| ANA profile                | Negative                                |                                                |
| ANCA (p-ANCA & c-ANCA)     | Negative                                |                                                |
| HbA1c                      | 4.9%                                    | <5.7%                                          |
| CSF Cell Count             | 5                                       | 0-5                                            |
| CSF Cell type              | Lymphocytes 100%                        | >80%                                           |
| CSF Protein                | 79                                      | 15-45                                          |
| CSF Glucose                | 56                                      | 40-70                                          |
| Simultaneous Blood Glucose | 98                                      | 70-140                                         |
| CSF Gram Stain & Culture   | No growth                               | No growth                                      |
| CSF MTB PCR (GeneXpert)    | Negative                                | Negative                                       |

## Discussion

Although Bell's palsy is a common cause of facial paralysis, simultaneous bilateral involvement should prompt consideration of systemic, infectious, inflammatory, neoplastic, and immune-mediated disorders. Keane's analysis of 43 cases of bilateral seventh nerve palsy demonstrated the heterogeneous nature of this presentation, with GBS among the important underlying causes [3]. In a retrospective review of approximately 8,600 patients referred for serological evaluation,

Susuki, et al. [4] identified 22 patients with acute progressive bifacial weakness accompanied by distal paresthesias and hyporeflexia, supporting this presentation as a regional variant of GBS; they noted that 64% showed demyelinating features on NCS and all had CSF albumin cytologic dissociation, providing supportive evidence for a GBS-spectrum disorder despite the absence of significant limb weakness. Wakerley and Yuki subsequently classified bifacial weakness with paresthesias as a localized GBS phenotype characterized by rapidly progressive bilateral facial weakness without limb weakness, ataxia, or other cranial neuropathies [5]. Our patient closely matched this phenotype. Paresthesias began in the hands, extended to the feet, and were followed by bilateral facial weakness. Limb strength remained normal, while ankle reflexes were reduced. The temporal sequence and distribution of neurological deficits therefore provided an important clue to the diagnosis. Several cases have been described in the literature, summarised in Table 4.

Electrodiagnostic testing is particularly valuable in this setting because clinically preserved limb power does not exclude peripheral nerve involvement. In our patient, abnormalities were demonstrated in multiple motor and sensory nerves, together with bilateral reduction of facial motor responses. The overall pattern supported a peripheral neuropathic process with facial nerve involvement. CSF examination provides additional supportive evidence. Albuminocytological dissociation, characterized by elevated protein with a normal or minimally elevated cell count, is a classic supportive feature of GBS [11]. The timing of lumbar puncture is relevant because CSF protein may remain normal early in the disease [12]. In this patient, CSF protein was elevated at 79 mg/dL with a cell count of 5 cells/μL, supporting the diagnosis in the appropriate clinical context. The differential diagnosis of bilateral facial palsy in this patient included infectious disorders such as Lyme disease, HIV infection, syphilis, tuberculosis, and leprosy; inflammatory conditions such as neurosarcoidosis brainstem inflammatory or structural lesions; and other immune-mediated neuropathies [13]. The absence of a structural lesion on MRI further reduced the likelihood of brainstem or cervical structural causes.

The Brighton Collaboration case definition provides standardized levels of diagnostic certainty for GBS [12]. However, its validation was based predominantly on patients with the classical phenotype characterized by bilateral limb weakness, limiting its applicability to regional variants such as BFWP. Therefore, diagnosis of BFWP requires integration of the characteristic clinical phenotype with supportive electrodiagnostic and CSF findings and exclusion of alternative causes. Treatment evidence specific to BFWP remains limited because of its rarity. IVIG and plasma exchange are established disease-modifying therapies for GBS, but there are no specific evidence-based treatment protocols exclusively for BFWP [14]. Early recognition is clinically important because progressive or functionally significant disease may warrant standard GBS immunotherapy. In the present case, progression from distal sensory symptoms to bilateral facial weakness, with impairment of eye closure and oral competence, prompted treatment with standard-dose IVIG. Clinical improvement in both facial weakness and paresthesias followed treatment.

Acute distal paresthesias should not be overlooked, particularly when accompanied by bilateral facial weakness. The presence of bifacial weakness should prompt consideration of Guillain-Barré Syndrome (GBS) variants, even when limb strength is preserved. In patients with bilateral facial palsy, associated distal paresthesias and

**Table 2:** Showing facial and motor nerve conduction studies with F-wave latencies.

| Nerve                      | Side | Stim Site          | Latency (ms) | Amplitude(mv) | CV (m/s) | F-wave latency |
|----------------------------|------|--------------------|--------------|---------------|----------|----------------|
| Facial (orbicularis oculi) | L    |                    | 2.95         | 1.43          |          | -              |
| Facial (orbicularis oculi) | R    |                    | 2.45         | 1.16          |          |                |
| Facial (nasalis)           | L    |                    | 2.6          | 1.25          | -        |                |
| Facial (nasalis)           | R    |                    | 2.2          | 1.36          |          |                |
| Median                     | L    | Wrist              | 7.7          | 4.29          | -        | 33.2           |
| Median                     | L    | Elbow              | 13.4         | 4.22          | 38.6     |                |
| Median                     | R    | Wrist              | 7.4          | 4.6           | -        | Absent         |
| Median                     | R    | Elbow              | 28           | 4.53          | 36.7     |                |
| Ulnar                      | L    | Wrist              | 3.4          | 12.7          | -        | 28.9           |
| Ulnar                      | L    | Below elbow        | 8.6          | 11.9          | 48.1     |                |
| Ulnar                      | L    | Above elbow        | 10.5         | 11.5          | 57.9     |                |
| Ulnar                      | R    | Wrist              | 3.3          | 13.72         | -        | 28.8           |
| Ulnar                      | R    | Below elbow        | 8.1          | 12.01         | 52.1     |                |
| Ulnar                      | R    | Above elbow        | 10           | 11.78         | 57.9     |                |
| Peroneal                   | L    | Ankle              | 5            | 3.46          | -        | 52.7           |
| Peroneal                   | L    | Below Femoral Head | 13.1         | 2.77          | 40.7     |                |
| Peroneal                   | L    | Popliteal          | 14.8         | 2.75          | 58.8     |                |
| Peroneal                   | R    | Ankle              | 6.1          | 2.43          | -        | 52.8           |
| Peroneal                   | R    | Below femoral head | 13.3         | 1.67          | 45.8     |                |
| Peroneal                   | R    | Popliteal          | 15.4         | 1.61          | 47.6     |                |
| Tibial                     | L    | Medial ankle       | 6.7          | 4.44          | -        | 60.1           |
| Tibial                     | L    | Popliteal          | 16.3         | 3.56          | 40.6     |                |
| Tibial                     | R    | Medial ankle       | 6.8          | 3.86          | -        | 59.1           |
| Tibial                     | R    | Popliteal          | 16.7         | 3.61          | 38.4     |                |

**Table 3:** Showing sensory nerve conduction studies.

| Nerve                | Side | Stim Site              | Latency (ms) | Amplitude(mv) | CV (m/s) |
|----------------------|------|------------------------|--------------|---------------|----------|
| Median               | L    | Wrist                  | Absent       | Absent        | -        |
| Median               | R    | Wrist                  | Absent       | Absent        | -        |
| Ulnar                | L    | Wrist                  | 2.88         | 11.5          | 34.7     |
| Ulnar                | R    | Wrist                  | 2.46         | 11.1          | 40.7     |
| Sural                | L    | Posterior lateral calf | 2.67         | 16.8          | 44.9     |
| Sural                | R    | Posterior lateral calf | 2.43         | 16            | 49.4     |
| Superficial Peroneal | L    | Lateral ankle          | 2.58         | 17.3          | 50.4     |
| Superficial Peroneal | R    | Lateral ankle          | 3.09         | 16.6          | 38.8     |

**Table 4:** Selected published cases of bifacial weakness with paresthesias (BFWP) reported between 2021 and 2025.

| Study                        | Age/Sex | Duration to presentation | First symptom                        | Sensory complaints                                | NCS/EMG                                                                                              | CSF | Antiganglioside Ab         | Treatment | Outcome                                                         |
|------------------------------|---------|--------------------------|--------------------------------------|---------------------------------------------------|------------------------------------------------------------------------------------------------------|-----|----------------------------|-----------|-----------------------------------------------------------------|
| Hand, et al. [6]             | 30/F    | 5 days                   | Limb paresthesias                    | Mild limb paresthesias; sensory ataxia            | Demyelinating polyneuropathy                                                                         | ACD | Anti-GM2 positive          | IVIG      | Improved                                                        |
| Xu, et al. [7]               | 33/M    | 7 days                   | Left facial palsy                    | Right toe numbness                                | Facial axonal involvement with mild peripheral nerve involvement                                     | ACD | Anti-GT1a IgM/IgG positive | IVIG      | Numbness recovered by day 10; partial facial recovery           |
| Boubga, et al. [8]           | 46/M    | 2 days                   | Tongue numbness with facial symptoms | Tongue numbness; dysgeusia                        | Severe bilateral facial demyelinating neuropathy; limb NCS essentially normal; abnormal blink reflex | ACD | Negative                   | IVIG      | Marked clinical improvement                                     |
| Hiraga, et al. [9]           | 19/M    | 16 days                  | Loss of taste                        | None reported                                     | Reduced median CMAP and ulnar SNAP                                                                   | ACD | Negative                   | IVIG      | Taste improved day 15; facial palsy day 17; paresthesias day 20 |
| Strawbridg, et al. 2022 [10] | 26/M    | ~2 weeks                 | Right facial palsy                   | Finger/toe numbness; glove-stocking sensory loss  | Facial nerve proximal demyelinating involvement; peripheral demyelinating neuropathy                 | ACD | Anti-GD1a IgG/IgM positive | IVIG      | Improved over several days                                      |
| Sardar, et al. [11]          | 34/M    | 1 day                    | Bilateral facial weakness            | Tongue numbness; no limb sensory deficit reported | Demyelinating neuropathy                                                                             | ACD | Not reported               | IVIG      | Complete recovery by 1 month                                    |

reduced or absent reflexes should raise suspicion for the Bilateral Weakness with Paresthesias (BFWP) variant and prompt appropriate electrodiagnostic and cerebrospinal fluid evaluation. This case reinforces that preserved limb strength should not be used to exclude GBS or its regional variants.

## Conclusion

Bilateral weakness with paresthesias is an uncommon regional variant of GBS that may be overlooked because significant limb weakness can be absent. Bilateral lower motor neuron facial weakness accompanied by distal paresthesias and reduced reflexes should raise suspicion for this phenotype and warrant further evaluation with electrodiagnostic studies and CSF examination, while imaging and targeted investigations help exclude alternative causes of bilateral facial palsy. Early recognition is important because progressive or functionally significant disease may warrant immunotherapy.

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