TURKISH JOURNAL OF EMERGENCY MEDICINE

Case Report/Case Series

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Quick Response Code:

Website: https://turkjemergmed.com/

DOI: 10.4103/tjem.tjem_152_24

# Navigating the complexities of bilateral facial nerve palsy: A case report

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**Abstract:**

A 32-year-old male presented with a 2-week history of progressive bilateral facial weakness, initially diagnosed as unilateral Bell's palsy. Upon development of bilateral symptoms, further investigations revealed normal hematological, biochemical, and imaging results, ruling out common infectious and autoimmune causes. Electromyography and nerve conduction studies were normal, and lumbar puncture results excluded Guillain–Barré syndrome. The patient was ultimately diagnosed with idiopathic bilateral facial nerve palsy (FNP) after exhaustive exclusion of other etiologies. Treatment with corticosteroids led to symptomatic improvement. This case underscores the importance of a systematic approach in diagnosing rare presentations of FNP and highlights the favorable prognosis with appropriate management.

**Keywords:**

Bell's palsy, bilateral facial nerve palsy, corticosteroids, electromyography, Guillain–Barré syndrome, idiopathic facial palsy

## Introduction

Bilateral facial nerve palsy (FNP) is a rare condition, accounting for < 1% of all facial palsy cases. Its presentation poses a significant diagnostic challenge due to the broad differential diagnosis that includes infectious, autoimmune, and neurological causes. This case report details the presentation, diagnostic process, and management of a 32-year-old male with progressive bilateral FNP. The initial unilateral presentation, followed by the development of bilateral symptoms, necessitated comprehensive investigations to rule out various potential etiologies. This report aims to contribute to the limited literature on bilateral FNP by providing insights into the diagnostic approach and management strategies for such rare cases.

## Case Report

A 32-year-old male presented with a 2-week history of progressive bilateral facial weakness. Initially, he experienced left-sided facial weakness for 5 days before seeking medical attention at a daycare clinic. At the clinic, he was diagnosed with Bell's palsy based on clinical examination. The neuro examination revealed left-sided lower motor neuron FNP, characterized by weakness in both the upper and lower parts of the face, inability to raise the eyebrow, and incomplete eye closure on the left side. He was prescribed a 5-day course of prednisone (dosage unspecified) without antibiotics or further investigations. Despite completing the course, his symptoms persisted. Twelve days later, he developed right-sided facial weakness, characterized by an inability to close both eyes and difficulty puffing his cheeks bilaterally.

Submitted: 07-08-2024
Revised: 16-12-2024
Accepted: 20-12-2024
Published: 01-07-2025

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**How to cite this article:** Moses CN, Mohamad WS, Baharuddin KA, Mafauzy MM, Shukri MF. Navigating the complexities of bilateral facial nerve palsy: A case report. Turk J Emerg Med 2025;25:239-41.

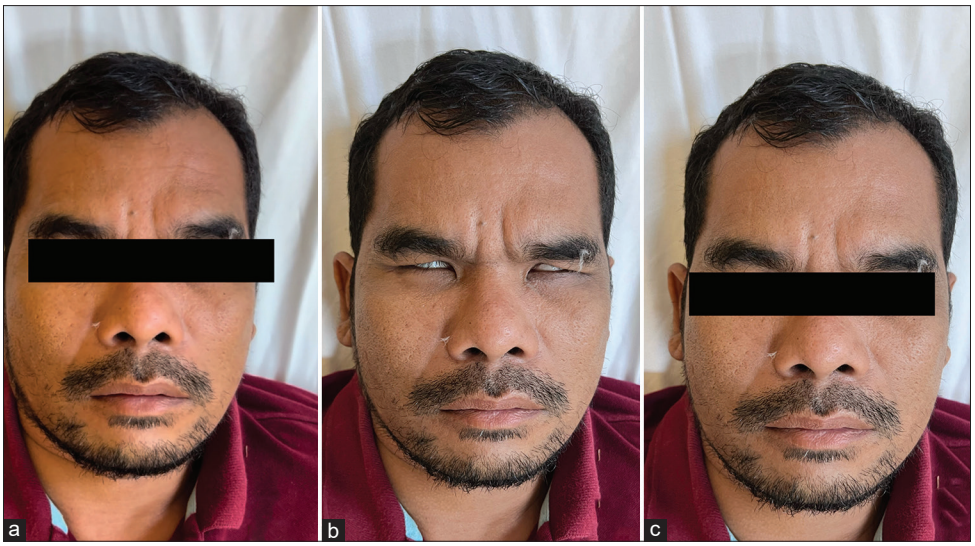


**Figure 1:** Clinical presentation of the patient demonstrating bilateral facial nerve palsy. (a) Inability to wrinkle the brow bilaterally, indicating compromised frontalis muscle function (b) Inability to close the eyes bilaterally, highlighting weakness in the orbicularis oculi muscles. (c) Asymmetrical smile bilaterally, showing reduced control over the zygomaticus major muscles[1]

He also reported perioral numbness, altered tongue sensation, and worsening right facial droop but retained the ability to swallow. He denied experiencing ear pain, upper respiratory tract infection symptoms, sore throat, facial swelling, headache, body weakness, dizziness, neck pain, limb weakness, chest pain, shortness of breath, or palpitations. He also had no constitutional symptoms such as weight loss, loss of appetite, or weight. He is a nonsmoker with no underlying medical illness.

In the emergency department, the patient appeared comfortable and was not in distress. His vital signs were normotensive with a blood pressure of 128/60 mmHg. He was not tachycardic and had an oxygen saturation of 98% under room air. Examination of his head, eyes, ears, nose, and throat was unremarkable with no rash or lymph nodes palpable. Neurological examination revealed bilateral lower motor neuron FNP, characterized by weakness in both the upper and lower parts of the face, inability to raise the eyebrows, and incomplete eye closure on both sides, without any other neurological deficits [Figure 1]. Hematological and biochemical tests, including liver function tests and electrolytes, were within normal ranges. A chest X-ray and contrasted computed tomography brain scan were also unremarkable, although he experienced an allergic reaction to the contrast medium, which was managed with hydrocortisone and chlorphenamine with good effect. He was admitted to the neuromedical department for further evaluation and observation.

During his hospital stay, an initial workup for Guillain–Barre syndrome (GBS) was considered. The patient subsequently agreed to a lumbar puncture, which returned normal results, effectively ruling out GBS. Further investigations were conducted to find the cause of the bilateral lower motor neuron FNP. Electromyography (EMG) and nerve conduction study (NCS) were performed and returned normal, indicating no evidence of demyelination or axonal damage. Infective screenings for human immunodeficiency virus (HIV), hepatitis, and syphilis returned negative results. Lyme disease serology, though not initially conducted, was eventually performed and returned negative. Immunological studies, including autoimmune panel tests such as antinuclear antibody and antibody to double-stranded deoxyribonucleic acid, were negative. A magnetic resonance imaging (MRI) of the brain and cranial nerves, scheduled 2 weeks later, showed normal results.

There was no progression of his symptoms during admission, and he was discharged 3 days later with a prescription for prednisolone 60 mg OD for 6 days continued by a taper dose for a total of 10 days. A follow-up MRI 2 weeks later confirmed normal results. The patient exhibited improvement in symptoms during this visit. Despite bilateral facial weakness persisting, the right side was more affected than the left, while other cranial nerves remained intact. Follow-up plans included a neuromedical clinic appointment in 1 month, and referral to speech therapy. A verbal and written consent was then obtained from the patient to publish this case report.

## Discussion

This case presents a diagnostic challenge due to its rarity and broad differential diagnosis. Initially diagnosed with unilateral Bell's palsy, the development of bilateral symptoms necessitated further investigation. Bilateral FNP is unusual and often indicative of a more complex

underlying condition than unilateral cases, highlighting the necessity for a comprehensive diagnostic approach.

Bilateral FNP accounts for <1% of all facial palsy cases.[1] In the absence of systemic symptoms, the patient's normal hematological and biochemical tests initially pointed away from infectious etiologies such as Lyme disease or HIV. Lyme disease, although a recognized cause of bilateral FNP, was ruled out with negative serology. This finding aligns with the literature that suggests bilateral facial involvement in Lyme disease is uncommon unless systemic symptoms are present.[2]

GBS can present with bilateral FNP, particularly in the Miller Fisher variant. However, the lack of limb weakness, areflexia, and normal cerebrospinal fluid (CSF) analysis effectively excluded GBS as a diagnosis. This is consistent with reports that emphasize the need for characteristic findings such as albumin cytologic dissociation in CSF for GBS diagnosis.[3,4] Furthermore, normal EMG and NCS ruled out demyelinating or axonal neuropathy, differentiating this case from typical neuropathies.[5]

Autoimmune conditions, including sarcoidosis and systemic lupus erythematosus, were considered. Negative autoimmune panels and normal MRI findings excluded these, reflecting their typical presentation with additional systemic symptoms and specific imaging findings.[6]

The diagnosis of idiopathic bilateral FNP, though rare, emerged as a diagnosis of exclusion after ruling out other potential causes. This case emphasizes the importance of a systematic and thorough diagnostic approach in cases of bilateral FNP. Idiopathic bilateral facial palsy, while less common than unilateral, should be considered, particularly when no other etiological factors are identified.[1]

Corticosteroid therapy, which led to some symptomatic improvement in this patient, remains the mainstay of treatment, paralleling the management of unilateral Bell's palsy.[7] The patient's gradual improvement also aligns with existing literature indicating a generally favorable prognosis for idiopathic cases with appropriate treatment.[1]

One limitation of this case is the lack of longitudinal follow-up beyond the initial recovery period, which limits the ability to assess long-term outcomes and potential recurrences. Future research could explore the pathophysiology of idiopathic bilateral facial palsy, focusing on genetic and environmental factors that might predispose individuals to this condition.

Further studies could also investigate the long-term outcomes of idiopathic bilateral FNP, particularly in relation to different treatment modalities. Comparative research into corticosteroids versus other potential therapeutic agents could offer insights into optimizing management strategies for these patients.

## Conclusion

This case highlights the complexity and diagnostic challenges of bilateral FNP. Through a detailed exclusion of other potential causes, idiopathic bilateral FNP emerged as the likely diagnosis. This stresses the importance of comprehensive diagnostic evaluations and supports the use of corticosteroids in managing such cases. Continued follow-up and supportive therapies are crucial for patient recovery and improving quality of life.

**Acknowledgments**

We would like to express our sincere gratitude to the patient for his cooperation and consent to participate in this case report. We extend our appreciation to the medical and nursing staff involved in the care and management of the patient. We acknowledge the support of our institution for providing the necessary resources for the successful completion of this case report.

**Author contributions**

CNM: Conceptualization (lead); Writing - Original draft (lead); Writing - Review and editing (equal) WS: Writing - Review and editing (equal); Writing - Original draft (supporting) KABB: Writing - Review and editing (equal) MMBM: Writing - Original draft (supporting) MFB: Writing - Review and editing (equal).

**Conflicts of interest**

None Declared.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

**Funding**

None.

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