International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Case Report

Atypical Guillain–Barré Syndrome Presenting with Unilateral Partial Ptosis and Mild Cerebrospinal Fluid Pleocytosis: A Diagnostic Challenge

Dr. Moumita Roy[1], Dr. Satrajit Ghosal[2], Dr Nabanita Roy[3]

[1]MD Anaesthesiology Medical Officer, ESIS Hospital Srirampur, Hooghly, West Bengal, India.
[2]MD Psychiatry, Medical Officer, ESIS Hospital Srirampur, Hooghly, West Bengal, India.
[3]MD Anaesthesiology M G M Medical College Kishanganj

OPEN ACCESS

Corresponding Author:

Dr. Moumita Roy
MD Anaesthesiology Medical Officer, ESIS Hospital Srirampur, Hooghly, West Bengal, India.

Received: 27-05-2026
Accepted: 10-06-2026
Available online: 03-07-2026

ABSTRACT

Background: Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that typically presents with rapidly progressive symmetrical weakness, areflexia, and albuminocytological dissociation. Atypical clinical and laboratory features may complicate diagnosis and mimic alternative neurological disorders.

Case Presentation: A 47-year-old male presented with acute flaccid quadriparesis of 7 days' duration associated with tingling sensations in all four limbs. Neurological examination revealed lower-limb hypotonia, areflexia, and an unusual finding of unilateral partial ptosis. Nerve conduction studies demonstrated prolonged bilateral F-wave latencies and absent H-reflexes, while routine motor and sensory conduction studies were largely preserved. Cerebrospinal fluid analysis showed albuminocytological dissociation with mild lymphocytic pleocytosis (10 cells/mm³). MRI brain revealed only mild chronic ischemic white matter changes (Fazekas grade 1). Owing to the atypical cranial nerve involvement and CSF pleocytosis, alternative diagnoses including neurolymphomatosis were considered. The patient was treated with intravenous immunoglobulin and physiotherapy, resulting in significant neurological improvement.

Conclusion: This case highlights an atypical presentation of GBS characterized by unilateral partial ptosis, mild CSF pleocytosis, and isolated late-response abnormalities on electrophysiological testing. Recognition of such uncommon manifestations is important to avoid diagnostic delay and to facilitate timely initiation of immunomodulatory therapy.

Keywords: Guillain–Barré syndrome; Acute inflammatory demyelinating polyradiculoneuropathy; Unilateral ptosis; Cerebrospinal fluid pleocytosis; Neurolymphomatosis; Cranial neuropathy.

INTRODUCTION

Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy and remains the most common cause of acute flaccid paralysis worldwide[(1).] It typically presents with rapidly progressive symmetrical limb weakness, generalized areflexia, and albuminocytological dissociation in cerebrospinal fluid (CSF)[(1-2).] Electrophysiological abnormalities include prolonged F-wave latencies, absent H-reflexes, and demyelinating or axonal changes on nerve conduction studies[(3).] However, atypical clinical and laboratory features such as cranial neuropathies, mild CSF pleocytosis,

or initially normal routine nerve conduction studies may create diagnostic uncertainty and raise suspicion for alternative inflammatory, infectious, or infiltrative neuropathies[4-5]. We report a case of early GBS presenting with unilateral partial ptosis and mild CSF pleocytosis, posing a diagnostic challenge with neurolymphomatosis.

CASE PRESENTATION

A 47-year-old male presented with acute flaccid paralysis of 7 days' duration. He complained that his legs felt "rubbery" and reported tingling sensations in all four limbs. Cutaneous sensory impairment was mild. There was no history of preceding fever, diarrhoea, respiratory tract infection, or recent vaccination. An atypical feature at presentation was unilateral partial ptosis.

On detailed neurological examination, the following findings were noted:

- Upper limbs (bilaterally): Normal tone, power grade 4/5, and normal superficial and deep reflexes.
- Lower limbs (bilaterally): Hypotonia, power grade 2/5, and absent reflexes.
- Cranial nerve examination was unremarkable except for partial ptosis of the right eyelid.
- Gait could not be assessed because the patient was unable to stand.
- Bowel and bladder functions were normal.
- General examination revealed blood pressure of 130/80 mmHg, pulse rate of 90/minute, SpO_2 of 98% on room air, and respiratory rate of 18/minute.
- Routine haematological and biochemical investigations, including complete blood count, liver function tests, renal function tests, fasting blood glucose, creatine kinase, and CK-MB levels, were within normal limits.
- MRI brain showed bilateral patchy periventricular and deep white matter T2/FLAIR hyperintensities without diffusion restriction, consistent with ischaemic white matter changes (Fazekas grade 1).
- Nerve conduction studies demonstrated prolonged bilateral F-wave latencies and absent H-reflexes.
- CSF analysis revealed albuminocytological dissociation with mild lymphocytic pleocytosis (protein: 226 mg/dL; glucose: 78 mg/dL; cell count: 10 cells/mm^3; all cells were lymphocytes).
- Based on the clinical presentation, electrophysiological findings, and CSF profile, a diagnosis of early Guillain–Barré syndrome, most likely acute inflammatory demyelinating polyradiculoneuropathy (AIDP), was considered.

The patient received intravenous immunoglobulin (IVIG) 20% for 5 days. Mild improvement in motor function was observed, evidenced by the patient's ability to stand independently. Subsequently, he underwent inpatient limb physiotherapy for an additional 7 days. Motor function improved significantly, and the patient was able to walk independently and attend to activities of daily living. He was discharged in a haemodynamically stable condition with lower-limb power of 5/5 and was advised to continue physiotherapy at home.

DISCUSSION

The present case demonstrated several characteristic features suggestive of GBS, including acute onset of lower motor neuron quadriparesis, generalised areflexia, flaccid paralysis predominantly involving the lower limbs, and electrophysiological evidence of proximal nerve root involvement in the form of prolonged F-wave latencies and absent H-reflexes despite relatively preserved routine motor and sensory conduction studies. Such findings may represent an early demyelinating polyradiculoneuropathy where distal conduction abnormalities are initially absent.

The markedly elevated CSF protein level (226 mg/dL) further supports the possibility of inflammatory polyradiculoneuropathy. However, the presence of mild lymphocytic pleocytosis (10 cells/mm^3) introduces diagnostic complexity. Although mild pleocytosis may occasionally occur in GBS, significant or persistent CSF cellularity warrants evaluation for alternative diagnoses, including infectious, neoplastic, or inflammatory etiologies.

An important atypical feature in this patient was unilateral partial ptosis, indicating cranial nerve involvement. Cranial neuropathies can occur in GBS, particularly in Miller–Fisher spectrum disorders[6], but asymmetric cranial findings may also suggest infiltrative neuropathies such as neurolymphomatosis. Furthermore, preserved bowel–bladder function, absence of autonomic instability, and lack of respiratory compromise may indicate either an early or localised GBS variant or a GBS mimic.

Neurolymphomatosis remains an important differential diagnosis because it can clinically resemble acute inflammatory neuropathies. Lymphomatous infiltration of nerve roots may produce proximal conduction abnormalities with relatively preserved distal nerve conduction studies in early stages. CSF may demonstrate elevated protein and lymphocytic pleocytosis. However cranial neuropathies, painful neuropathy, asymmetric deficits, weight loss, or systemic "B" symptoms were not present in this case,.The favourable response to IVIG strongly supported an inflammatory rather than neoplastic etiology.

The normal sensory and motor conduction studies with isolated late response abnormalities may represent very early GBS, particularly acute inflammatory demyelinating polyradiculoneuropathy (AIDP). In early GBS, F-wave prolongation and absent H-reflexes are among the earliest electrophysiological abnormalities because proximal nerve roots are preferentially affected initially.

MRI brain demonstrated bilateral patchy periventricular and deep white matter T2/FLAIR hyperintensities without diffusion restriction, consistent with chronic ischemic white matter changes (Fazekas grade 1). Fazekas scale grade 1 changes generally represent mild chronic microvascular ischemic alterations commonly seen with ageing or vascular risk factors and are usually considered nonspecific incidental findings. Importantly, the absence of diffusion restriction or focal enhancing lesions argues against acute ischemic pathology or central demyelinating processes contributing significantly to the acute presentation.

CONCLUSION

This case illustrates an atypical presentation of Guillain–Barré syndrome characterized by unilateral partial ptosis, mild lymphocytic CSF pleocytosis, and isolated late-response abnormalities on nerve conduction studies. Recognition of these uncommon features is important because they may mimic infiltrative neuropathies such as neurolymphomatosis and delay diagnosis. Early identification of proximal nerve root involvement through F-wave and H-reflex studies, together with prompt immunomodulatory therapy, can facilitate favourable neurological outcomes even when routine nerve conduction studies remain initially normal.

REFERENCES

1. Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. Lancet. 2016 Aug 13;388(10045):717-727.
2. van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome. Lancet Neurol. 2008 Oct;7(10):939-950.
3. Leonhard SE, Mandarakas MR, Gondim FAA, Bateman K, Ferreira MLB, Cornblath DR, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol. 2019 Nov;15(11):671-683.
4. Cornblath DR, Asbury AK, Albers JW, Feasby TE, Hahn AF, McLeod JG, et al. Research criteria for diagnosis of Guillain-Barré syndrome. Ann Neurol. 1990;27(Suppl):S21-S24.
5. Bae JS, Kim JK, Kim DS. Guillain-Barré syndrome with cerebrospinal fluid pleocytosis. J Clin Neurol. 2010;6(3):144-146.
6. Yuki N, Hartung HP. Guillain-Barré syndrome. N Engl J Med. 2012 Jun 14;366(24):2294-2304.