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GUILLAIN-BARRÉ SYNDROME: FACIAL DIPLEGIA WITH HYPERREFLEXIC VARIANT IN PREGNANCY- A RARE CASE REPORT

General Medicine

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ABSTRACT

Guillain–Barre syndrome (GBS) is a common cause of acute peripheral neuropathy and is characterized by hyporeflexia or areflexia. Hyperreflexia has been rarely reported with acute motor axonal neuropathy. 21-year-old pregnant women presented with asymmetrical weakness of upper and lower limbs and change of voice. Weakness progressed in the hospital with involvement of multiple cranial nerves, preserved deep tendon jerks with extensor plantar and normal abdominal reflexes atypical variants with preserved or brisk reflexes, asymmetrical weakness, bulbar involvement are rare and diagnostically challenging, particularly in pregnancy. She was treated with IV immunoglobulin and IV methylprednisolone. She able to walk with support with normal voice at the time of discharge. GBS should be a differential diagnosis in patients with acute quadriparesis even if there are preserved deep tendon reflexes.

KEYWORDS

Guillain-Barré Syndrome, Hyperreflexia, Facial Diplegia, Pregnancy, IVIG

INTRODUCTION

Guillain–Barré Syndrome (GBS) is an acute, post-infectious, immune-mediated neuropathy and the most frequent cause of acute flaccid paralysis worldwide. The classical presentation consists of symmetrical ascending weakness, areflexia, and cranial nerve involvement, often preceded by an antecedent infection. However, atypical variants exist, which may present with preserved or brisk reflexes, asymmetric weakness, or early cranial nerve and bulbar involvement. These uncommon features often complicate the diagnosis, especially in resource-limited settings where electrophysiological confirmation may be delayed.The occurrence of GBS during pregnancy is rare, with an estimated incidence of 1.2–1.9 cases per 100,000 pregnancies, similar to the general population, but with potentially higher maternal morbidity due to diagnostic delays and respiratory complications. Atypical variants, including those with hyperreflexia or asymmetric weakness, are increasingly recognized and may represent acute motor axonal neuropathy (AMAN) or conduction block neuropathy, where preserved sensory pathways and corticospinal hyperexcitability explain exaggerated reflexes. Pregnancy-specific reports describe variable outcomes. Some cases document successful management with intravenous immunoglobulin (IVIG), leading to good maternal and neonatal outcomes, while others highlight risks of preterm labor and maternal ventilatory failure when treatment is delayed. IVIG remains the preferred therapy in pregnancy due to its safety profile, while corticosteroids are less effective as monotherapy but may provide additional benefit in select situations.

CASE REPORT –

A 22-year-old G2P2L1 8month ANC, presented to the intensive care unit on 18/08/2025 with complaints of progressive weakness and difficulty in swallowing. She was apparently well until 8 days prior to admission, when she developed four episodes of loose stools without fever or blood. Two days later, she noticed difficulty in swallowing both liquids and solids, associated with change of voice. By 3rd day lower limbs patient developed weakness in both upper limb and lower limbs more on left side. She reported difficulty gripping objects, combing hair, and inability to stand with support associated with change of voice but denied sensory loss, bowel, or bladder involvement.

Examination

On general examination, the patient was conscious, oriented, and afebrile, with stable vital parameters. No cranial deformities or neck rigidity were noted. Neurological examination revealed normal muscle bulk with increased tone in both lower limbs. Motor power was reduced: shoulder abduction (Right 2+/5, Left 1+/5), elbow flexion/extension (Right 2+/5, Left 1+/5), wrist and grip weak, hip and knee (3/5 bilaterally), ankle dorsiflexion/plantar flexion (3/5 bilaterally).

Deep tendon reflexes were brisk (3+) in the knees and ankles, biceps and triceps were 2+, while jaw jerk was absent

Sensory examination was normal for touch, pain, vibration, and proprioception.

Cranial nerve assessment revealed bilateral lower motor neuron 7 th nerve palsy, weak gag reflex, and involvement of cranial nerves 10th, 11th and 12th. Other systemic examinations were unremarkable.

Investigations

- Haematology:
- Haemoglobin - 8.9 g/dL
- TLC-10,450/mm³
- Platelets -437 ×10⁹/L
- Peripheral smear: Microcytic hypo-chromic anaemia.
- Liver and renal function tests: Within normal limits
- Cerebrospinal fluid (CSF): Albumino-cytological dissociation.
- Nerve conduction study **AMAN (**acute motor Axonal neuropathy)

Nerve Conduction Velocity (NCV) Report

Nerve Conduction Velocity (NCV) study was performed in both upper and lower limbs.

- Sensory nerves (SNAPs): Median, ulnar, and sural nerves showed normal onset latencies, normal amplitude, and conduction velocity.
- Motor nerves (CMAPs): Median, ulnar, peroneal, and tibial nerves showed normal distal latencies, amplitudes, and conduction velocity.
- F waves: Recorded from median and tibial nerves were of normal latencies and were persistent.

Impression:
This NCV study suggests mild to moderate, generalized, predominantly motor polyneuropathy.
[? AMAN – Acute Motor Axonal Neuropathy]. Clinical correlation is advised.

MRI brain: Normal.

MRI spine: Normal

Obstetric ultrasonography (20/08/2025): Single live intrauterine

gestation, cephalic presentation, posterior grade II placenta, amniotic fluid index 10–11 cm.

- Based on above investigations and clinical examination patient was diagnosed with Atypical GBS with hyperreflexia variant. Pt was started on IVIG 2g/kg for 5days
- Pt muscle power significantly improved from 2/5 to 4/5 in upper limbs and 3/5 to 5/5 in lower limbs and she was able to walk without support.

DISCUSSION –

Areflexia is one of the two clinical features required for diagnosis of GBS.Normal-reflexes or hyperreflexia throughout the course of GBS is unusual. Deep tendon reflexes may be preserved throughout the disease course in patients with acute motor axonal neuropathy (AMAN) and have been considered-as indicators of rapid clinical recovery. Our patient's clinical-presentation and disease course typical of GBS except for preservation of reflexes. As for diverse disease entities within the GBS spectrum, the clinical and electrophysiological findings of the patient best fit in AMAN, in which the primary immunological attack is supposed to be directed against motor axons. Although hyperreflexia is a controversial symptom in patients with GBS, these findings indicate that there is functional corticospinal tract involvement in patients with a GBS variant. Moreover, 48% of Chinese and 33% of Japanese patients with AMAN showed hyperreflexia in the recovery phase1,2] In another study, patients with pure motor GBS had preserved tendon reflexes up to MRC grade 3 paresis. Acute facial diplegia with hyperreflexia has been described as a GBS variant and nerve conduction studies in limbs were normal in this report. Preserved reflexes and even hyperreflexia may occur in patients with pure motor GBS and are not inconsistent with the diagnosis. It is more appropriate to classify this neuropathy as a GBS variant, which Capasso et al., suggest calling "acute motor conduction block neuropathy," emphasizing the presence of conduction blocks and avoiding the pathophysiologic implication that all conduction blocks are demyelinating in nature. They propose that conduction block in their case was due to axonal block by the antibody at the node of Ranvir without demyelination. Recently, electrophysiologic evidence of lower motor neuron hyper excitability has been reported in some patients with AMAN. It has been suggested that the most common sites of nerve involvement in patients with GBS are not randomly distributed throughout the nerve. The distal motor nerves, the proximal segments, and the sites prone to compression are most vulnerable. Early in the disease, electrophysiologic abnormalities are often mild or nonspecific. Motor conduction blocks (CB) have been documented in only 2–15% of patients with GBS within 3 weeks from disease onset, and CB in intermediate nerve segments in the first days of the disease is uncommon. It is possible that mechanical impairment of the blood–nerve barrier at entrapment site may render these nerve segments more vulnerable immunologic attack. The pathologic basis of GBS is thought to be acute demyelination. Experimental studies of synchronously demyelinating lesions showed that recovery from GBS is characterized by dyssynchronization. It might be possible to explain the preservation of tendon reflexes in GBS by following factors. The presence of normal sensory function rather than motor is required for tendon jerks. As tendon jerks are dependent on synchronized volley of impulses, a purely axonal lesion would preserve tendon jerks better than a demyelinating lesion. In our patient, no electro diagnostic correlate of peripheral nerve demyelination was found.

CONCLUSION –

This case emphasizes that Guillain–Barré Syndrome may present with atypical features such as bulbar involvement, asymmetry, and preserved or brisk reflexes, which can mimic central nervous system disorders and delay diagnosis. In pregnancy, where diagnostic ambiguity and therapeutic concerns are heightened, maintaining a high index of suspicion is critical. Early recognition, timely initiation of IVIG, and multidisciplinary care not only ensure maternal neurological recovery but also safeguard favorable perinatal outcomes. This report adds to the limited literature on atypical GBS variants in pregnancy and highlights the importance of individualized, prompt intervention in achieving optimal results.

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