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Case Report

GUILLAIN–BARRE SYNDROME (GBS) RISE IN COMPLICATIONS DUE TO DIAGNOSTIC PITFALLS: A CASE REPORT

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ABSTRACT :

Guillain-Barré syndrome (GBS) is an acute condition that is immune-mediated and characterized by variant clinical presentations and polymorphous behaviour. Antecedent infections such as Campylobacter jejuni are widely considered and well recognized as triggers; however, evidence is emerging pointing to GBS in association with arboviral infections (for example, Chikungunya) and, although rarely, the vaccination for COVID-19. This case poses an interesting diagnostic challenge involving a 60-year-old man with progressive paraesthesia and symmetrical limb weakness, initially considered to be atypical due to neuroimaging findings (hypodense area in left caudate nucleus). Further testing, analysis of cerebrospinal fluid, and nerve conduction studies showed that the patient had acute inflammatory demyelinating polyneuropathy (AIDP), proving the missteps one could fall into when heavily reliant on imaging in GBS diagnosis.

After intravenous immunoglobulin (IVIG) treatment, the patient improved significantly, thereby reaffirming IVIG as a treatment mainstay. The case holds three main lessons: (1) GBS can imitate structural brain lesions on imaging and must be cleared when working through this differential; (2) arboviral infections and vaccines may be implicated in GBS pathogenesis where they are endemic; and (3) rapid immunotherapy reduces disability. Our findings advocate for heightened clinical suspicion for atypical presentation to avert any diagnostic delay.

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1. INTRODUCTION:

Guillain-Barré syndrome (GBS) is an autoimmune disorder that causes acute inflammation of demyelinating peripheral nerves, leading to progressive muscle weakness and sensory disturbances [1]. Infections well known to be antecedents include those with Campylobacter jejuni and cytomegalovirus infections, while emerging evidence now suggests that arboviruses such as Zika and Chikungunya and vaccinations like those against COVID-19 now fall under triggers for GBS in a paradigm shift in etiology [2, 3]. This case is unique because both antecedent events were in play, one being Chikungunya infection and the other recent COVID-19 vaccination, and arising from an initial diagnosis confounding, all accounted for counterintuitive neuroimaging findings, which have had scant documentation in literature.

GBS typically presents with ascending paralysis, areflexia, and albuminocytologic dissociation in cerebrospinal fluid (CSF) [4]. Any atypical presentation, such as aberrations in brain imaging early in the course suggesting a stroke, would delay the diagnosis resulting in aggravating the worst outcome as witnessed in our patient.

Despite its rarity (incidence: 1–2/100,000 annually), GBS demands urgent intervention; 20–30% of cases require mechanical ventilation, and 3–7% prove fatal due to autonomic or respiratory complications [5, 6]. Current management relies on IVIG or plasmapheresis, but timely administration hinges on clinical suspicion, especially in diagnostically ambiguous cases [7, 8].

This report underscores three critical gaps: (1) the under recognized role of arboviral infections in GBS pathogenesis, (2) the diagnostic pitfalls of overreliance on neuroimaging in GBS, and (3) the therapeutic implications of early immunotherapy in atypical presentations. By highlighting this unique case, we aim to refine diagnostic protocols for GBS in settings where arboviruses and vaccinations coexist as competing triggers.

2. CASE REPORT:

Structured Timeline of Patient Presentation, Diagnosis, and Management

Time	Event	Key Findings/Interventions
2 weeks prior	Antecedent viral infection	- Chikungunya - IgM +ve - Elevated serum CRP
Recent	COVID-19 vaccination	- Received 2 doses of COVISHIELD
Day 0 (Onset)	Initial symptoms	- Progressive weakness, pain, tingling in limbs - Difficulty walking, slurred speech, bifacial weakness
Day 7	Hospital admission	- Suspected stroke due to CT hypodensity in left caudate nucleus - Misdiagnosis initially
Day 7-14	Clinical deterioration	- Worsening weakness, unable to walk - Transferred to tertiary care center
Day 14	Advanced diagnostics	- **Repeat CT brain**: Atrophic changes (no acute ischemia)

		- **CSF analysis**: Elevated protein (32.2 mg/dL), normal glucose (60 mg/dL) - **NCV**: Demyelinating polyradiculoneuropathy (AIDP pattern) - Delayed SNAP/CMAP latencies, reduced amplitudes
Day 15	Confirmed GBS diagnosis	- Fulfilled Brighton criteria for GBS - AIDP subtype identified
Day 15-20	Treatment initiated	• Dose: 0.4 g/kg/day for 5 days (total 2 g/kg) • Rationale: First-line therapy for AIDP • Alternative considered: Plasmapheresis (unavailable) • Response: MRC improvement (2/5→3/5) by Day 3 • Adjuncts: Gabapentin 300mg TDS, physiotherapy This single-point table:
Day 21+	Post-treatment monitoring	- Partial recovery of limb movement observed - Ongoing rehabilitation

i. Clinical presentation:

A 59 year old male, presented with a 7 day history of progressive weakness, pain and tingling in both lower and upper limbs, difficulty in up-squating, slurring of speech, bifacial weakness and suddenly he was unable to walk.

ii. History:

Two weeks ago he had a viral Infection with viral fever. He had been treated by his local family doctor who had sent a blood sample for laboratory examination, which came back as ChikungunyaIgM +ve and High serum C-reactive protein.

1) Past medical history: Hypertension, Pre-diabetic
2) Past surgical history: Hernia surgery, neurovascular surgery
3) Recent Vaccination History:2 doses of COVISHIELD vaccine for (COVID 19)
4) Family history: No positive family history for any other chronic diseases
5) Social history: Lives with his family (Wife and 2 sons), non-smoker, no alcohol

iii. Differential Diagnosis:

Primary diagnosed with appearance of small hypodensity in left caudate nucleus in CT scan study of brain. In radiological examination atrophic alterations were seen (brain's CT scan). NCV study shows demyelinating polyradiculoneuropathy like AIDP.

iv. Examination:

CSF examination: CSF protein- 32.2mg/dl, sugar-60mg/dl

NCV examination: the sensory nerve action potential (SNAP) recorded from right sural nerve were of delayed onset latencies, reduced amplitude and conduction velocity. A Compound Muscle Action Potential (CMAP) significantly delayed distal latencies, reduced amplitude and conduction velocity. At 3-month follow-up, the patient showed significant recovery (MRC 4/5 in limbs) with outpatient physiotherapy, no IVIG-related adverse events, and full compliance to rehabilitation.This study

shows demyelinating polyradiculoneuropathy like AIDP [9-14]

3. DISCUSSION

This case highlights three critical aspects of GBS management: antecedent triggers, diagnostic challenges, and therapeutic implications. While *Campylobacter jejuni* and respiratory viruses are classic GBS precursors [15], our patient's Chikungunya infection (IgM +ve) and recent COVISHIELD vaccination represent underreported triggers, aligning with emerging evidence of arboviral/vaccine-associated GBS [16,17]. In addition to temporal proximity (2-week latency) and exclusion of other causes (e.g., metabolic/toxic), dual exposure complicates making attribution as to how the etiology came to be.

Diagnostic Failures & Strengths

The very first misdiagnosis (CT hypodensity mimicking stroke) informs two powerful lessons:

1. Early imaging overstated can delay NCS and lumbar puncture in GBS if it mimics CNS pathology [18].

2. Albuminocytologic dissociation (32.2 mg/dL CSF protein) and NCS (demyelination-specific) still remain the gold standard, as they did in our case.

Therapeutic justification

Although both are equally effective according to guidelines [19], IVIG (0.4 g/kg × 5 days) was preferred to plasmapheresis because of hospital constraints, which led to 3 months of recovery (MRC 4/5). It really solidifies the role of IVIG in axonal repair, but with weakness residual, it indicates a varying capability of regeneration following demyelination [20].

Strengths

- Comprehensive workup (CSF/NCS/imaging) excluding mimics (e.g., CIDP, vasculitis).
- Documentation of rare triggers (Chikungunya + COVID-19 vaccine).

Limitations

- No serial NCS to track remyelination.
- Unmeasured anti-ganglioside antibodies (e.g., GM1) further the diagnosis of molecular mimicry.

Takeaway: Early NCS is best applied in atypical paralysis, as demonstrated in the case, and arboviral/vaccine triggers should be considered in endemic areas.

Patient Perspective:

My treatment got postponed for a week due to misdiagnosis as a stroke. Though I regained some strength after IVIG, it is still very challenging for me to do daily activities after three months. An earlier diagnosis would have improved my outcome.

Declaration of patient consent

The authors confirm that they have acquired a patient consent form, in which the patient or caregiver has granted permission for the publication of the case, including accompanying images and other clinical details, in the journal. The patient or caregiver acknowledges that their name and initials will not be disclosed, and sincere attempts will be undertaken to safeguard their identity. However, complete anonymity cannot be assured.

4. CONCLUSION:

In patients with an atypical presentation, the diagnosis of GBS is often held up, usually because the diagnostic standards of GBS involve CSF investigation and

electrodiagnostic testing. We wish to point this out because this case report highlights the limitations of the existing diagnostic criteria, calls for prompt diagnosis by practitioners, and underscores the importance of maintaining vigilance for GBS symptoms.

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