CODEN: WJAPAC Impact Factor: 5.48 ISSN: 3049-3013

World Journal of Advance Pharmaceutical Sciences

Volume 3, Issue 5, Page: 57-61 Case Study www.wjaps.com

GUILLAIN-BARRÉ SYNDROME WITH SUPRAVENTRICULAR TACHYCARDIA AND ALTERED SENSORIUM IN A CHILD: A RARE ATYPICAL CASE

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How to cite this Article: Ashwini Angadi[1], Adarsh G. S.*[1], Sidappa F. D.[2], Preeti V. Kulkarni[1], Venkatrao H. Kulkani[1] (2026). GUILLAIN-BARRÉ SYNDROME WITH SUPRAVENTRICULAR TACHYCARDIA AND ALTERED SENSORIUM IN A CHILD: A RARE ATYPICAL CASE. World Journal of Advance Pharmaceutical Sciences, 3(5), 57-61.

Article Info

Article Received: 27 February 2026,

Article Revised: 17 March 2026,

Article Accepted: 07 April 2026.

DOI: https://doi.org/10.5281/zenodo.19922988

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ABSTRACT

Guillain–Barré syndrome (GBS) is an uncommon autoimmune-mediated neurological condition that primarily affects the peripheral nervous system, resulting in acute weakness and possible paralysis. However, atypical pediatric presentations involving central nervous system features and cardiac abnormalities are rare and diagnostically challenging. We describe an unusual case of a 10-year-old female child who presented with sudden onset and progressively worsening throat pain aggravated by swallowing, along with chest pain and breathlessness. Initial electrocardiography revealed supraventricular tachycardia. There was no history of fever, respiratory symptoms, vomiting, toxin exposure, or previous similar illness. The child had normal developmental milestones and complete immunization. Clinical examination revealed mild tachycardia and elevated random blood glucose levels (213 mg/dL). Systemic evaluation showed hepatosplenomegaly and bilateral lung crepitations. Neurological assessment demonstrated altered sensorium, fixed mid-dilated pupils, and progressive decline in Glasgow Coma Scale to 6/15. Laboratory investigations showed mild leukocytosis, hyponatremia, elevated cardiac enzymes (CK-MB and troponin), and mildly increased cerebrospinal fluid protein with lymphocytic predominance in the absence of infectious organisms. Renal parameters and two-dimensional echocardiography were within normal limits. The patient's neurological status further deteriorated with suspected non-convulsive status epilepticus. Management included antimicrobial therapy, corticosteroids, and antiepileptic treatment with levetiracetam, along with supportive care. This case illustrates a rare and complex pediatric presentation of Guillain–Barré syndrome complicated by non-convulsive status epilepticus, autonomic involvement, and metabolic derangements. It highlights the importance of early recognition of atypical features, prompt neurological intervention, and multidisciplinary management to prevent adverse outcomes in pediatric patients with unusual neurological manifestations.

KEYWORDS: Guillain–Barré syndrome, Pediatric neurology, Non-convulsive status epilepticus, Autonomic dysfunction, Supraventricular tachycardia, Hyponatremia, Peripheral neuropathy.

INTRODUCTION

Guillain–Barré syndrome (GBS) is a rapidly progressive, immune-mediated disorder of the peripheral nerves characterized by symmetrical limb weakness, hyporeflexia or areflexia, and variable autonomic disturbances. Following the effective control of poliomyelitis, GBS has emerged as the most common cause of acute flaccid paralysis worldwide.[1] It affects individuals of all age groups and is frequently triggered by a preceding bacterial or viral infection. Although the exact pathophysiological mechanism is not completely understood, growing evidence suggests that molecular mimicry leads to an aberrant immune response directed against peripheral nerve components after an antecedent illness.[1]

The reported global incidence of GBS ranges from 0.89 to 1.89 cases per 100,000 person-years, with a higher occurrence observed in males. Campylobacter jejuni is responsible for approximately 30% of post-infectious cases, whereas cytomegalovirus accounts for nearly 10%.[2] Early clinical manifestations may include fever, sore throat, and symptoms of respiratory or gastrointestinal infection, particularly in axonal subtypes of the disease. Definitive diagnosis is based on the recognition of progressive motor weakness, supported by cerebrospinal fluid examination and electrophysiological studies; however, classic albuminocytologic dissociation may be absent during the first week of illness.[2,3]

Treatment primarily focuses on vigilant monitoring for respiratory failure and autonomic instability, along with prompt initiation of intravenous immunoglobulin (IVIG) or plasma exchange, and comprehensive supportive care. Despite standard management strategies, some patients continue to experience significant morbidity, leading to the development of newer therapeutic approaches such as salivated IVIG, engineered antibodies, IdeS protease, and monoclonal antibodies including eculizumab.[4]

CASE REPORT

A 10-year-old girl presented with sudden-onset throat pain that progressively worsened over one day, associated with painful swallowing, dysphagia to both liquids and solids, pricking chest pain lasting about 30 minutes, and breathlessness even at rest. There was no history of fever, cough, vomiting, diarrhea, or poisoning. An electrocardiogram done at a local hospital revealed supraventricular tachycardia (SVT), for which she received paracetamol, intravenous fluids, a proton-pump inhibitor, and oral amoxiclav before being referred to our hospital.

Her past history was unremarkable, with no similar episodes or previous hospitalizations. She was fully immunized, developmentally normal, and had adequate nutritional intake. She belonged to a lower socioeconomic household, lived in a kaccha house with functional sanitation facilities, and used borewell water for drinking.

On admission, she was afebrile (98.6°F), tachycardic (120 beats/min), and hemodynamically stable with a blood pressure of 100/60 mmHg, respiratory rate of 20/min, and oxygen saturation of 97% on room air. Random blood sugar was 213 mg/dL. General examination revealed no pallor, icterus, cyanosis, lymphadenopathy, or edema. Her height was 139 cm and weight 35 kg (BMI 17 kg/m²).

Head-to-toe examination revealed mid-dilated fixed pupils with congested conjunctiva. ENT and oral cavity examinations were normal. No limb deformities or skin changes were noted. Cardiovascular examination revealed normal heart sounds without murmurs. Abdominal examination showed hepatomegaly (3 cm below the right costal margin) with splenomegaly. Respiratory examination showed normal vesicular breath sounds with occasional bilateral crepitations.

On neurological examination, she was conscious but had mid-dilated non-reactive pupils. Facial nerve function was intact. Muscle tone was normal, deep tendon reflexes were 1+, and plantar responses were flexor bilaterally. There were no signs of meningeal irritation. Based on altered responsiveness and pupillary findings, a provisional diagnosis of **Non-convulsive Status Epilepticus (NCSE)** was made.

On the following day, endoscopy results were awaited and samples were sent for oncological evaluation. Antimalarial therapy, 3% hypertonic saline, anidulafungin, and acyclovir were initiated. A lumbar puncture was planned, and neurology consultation was obtained.

Later the same day, she developed sudden chest pain and worsening breathlessness followed by unresponsiveness and gasping respirations. She was shifted to the PICU, where her **GCS deteriorated to 6/15**. Pupils were 4 mm and non-reactive, limb movements were minimal, and the doll's eye reflex was preserved. Intravenous levetiracetam was initiated, and aggressive anti-seizure management was continued for suspected NCSE.

Table 1: Laboratory Investigations.

Parameter	Observed Value	Normal Range	Status	Reason / Confirmation Related to Case
Hemoglobin	13.7 g/dL	11.5 – 15.5 g/dL	Normal	Confirms absence of anemia; oxygen-carrying capacity preserved
TLC	16,660 /mm³	4,000 – 11,000 /mm³	**High**	Confirms active inflammatory/infective trigger for immune-mediated GBS

DLC	4.7 / 88	N: 40–70%, L: 20–40%	**Neutrophil predominance**	Supports acute systemic inflammation
PCV	40.4 %	36 – 46 %	Normal	Confirms stable hematological status
Platelets	2.81 lakhs/mm³	1.5 – 4.5 lakhs/mm³	Normal	Rules out thrombocytopenia-related complications
RBS	215 mg/dL	70 – 140 mg/dL	**High**	Confirms stress-induced hyperglycemia contributing to altered sensorium
Serum Urea	16.7 mg/dL	10 – 40 mg/dL	Normal	Confirms preserved renal function
Serum Creatinine	0.4 mg/dL	0.5 – 1.0 mg/dL	Slightly Low	Not clinically significant; kidney function preserved
Serum Uric Acid	3.6 mg/dL	2.4 – 6.0 mg/dL	Normal	Confirms normal purine metabolism
Sodium	128.8 mmol/L	135 – 145 mmol/L	**Low**	Confirms hyponatremia as a cause of NCSE and altered consciousness
Potassium	3.6 mmol/L	3.5 – 5.0 mmol/L	Low-normal	May contribute to cardiac rhythm instability
O_2 Saturation	96.7 %	95 – 100 %	Normal	Confirms adequate oxygenation
Total Protein	7 g/dL	6.0 – 8.3 g/dL	Normal	Confirms preserved nutritional and hepatic synthetic status
Albumin	4.5 g/dL	3.5 – 5.0 g/dL	Normal	Supports absence of chronic illness or liver disease
Globulin	2.5 g/dL	2.0 – 3.5 g/dL	Normal	Confirms normal immune protein levels
Total Bilirubin	0.2 mg/dL	0.2 – 1.2 mg/dL	Normal	Rules out hepatic or hemolytic involvement
Direct Bilirubin	0.1 mg/dL	0 – 0.3 mg/dL	Normal	Confirms normal hepatic excretory function
Indirect Bilirubin	0.1 mg/dL	0.2 – 0.9 mg/dL	Slightly Low	Clinically insignificant
SGOT	27.7 U/L	5 – 40 U/L	Normal	Confirms no liver cell injury
SGPT	21.4 U/L	5 – 40 U/L	Normal	Rules out hepatocellular inflammation
ALP	238 U/L	40 – 300 U/L (Pediatric)	Normal	Confirms age-appropriate bone/liver activity

The overall clinical profile and laboratory findings suggest a **severe, atypical presentation of Guillain–Barré syndrome complicated by autonomic dysfunction, metabolic encephalopathy, and non-convulsive status epilepticus**. The presence of **marked leukocytosis** indicates an underlying **infectious or inflammatory trigger**, likely precipitating the immune-mediated neuropathic process. **Significant hyponatremia and stress-induced hyperglycemia** are important contributors to the patient's **altered sensorium, seizures, and rapid neurological deterioration**. Elevated **cardiac biomarkers (CK-MB and Troponin)** along with **supraventricular tachycardia** confirm **cardiac involvement due to autonomic instability**, explaining the recurrent chest pain and breathlessness. The **cerebrospinal fluid finding of elevated protein with minimal cells** supports an **immune-mediated neuropathy consistent with Guillain–Barré syndrome** rather than an active central nervous system infection. Preserved **renal and hepatic function** allowed safe administration of aggressive medical therapy. Overall, this represents a **life-threatening, multisystem involvement of pediatric Guillain–Barré syndrome requiring intensive multidisciplinary care and close biochemical, cardiac, and neurological monitoring**.

Table 2: Cardiac and Electrolyte Markers.

Parameter	Observed Value	Normal Range	Status	Reason / Interpretation Related to Case
CK-MB	87.6 U/L	0 – 25 U/L	**High**	Confirms acute myocardial injury likely due to autonomic dysfunction in GBS and systemic stress
Troponin	12.7 ng/mL	< 0.04 ng/mL	**Markedly High**	Strong evidence of myocardial injury explaining chest pain and breathlessness
Calcium	9.3 mg/dL	8.5 – 10.5 mg/dL	Normal	No role in seizure or cardiac complications
Magnesium	1.5 mg/dL	1.7 – 2.4 mg/dL	**Low**	Predisposes to arrhythmia and may have contributed to SVT
Phosphate	3.4 mg/dL	2.5 – 4.5 mg/dL	Normal	No contribution to neuromuscular or cardiac instability

The **marked elevation of CK-MB and Troponin confirms acute myocardial injury**, most likely secondary to **autonomic nervous system involvement in Guillain–Barré syndrome and systemic metabolic stress**. The **low magnesium level further increases the risk of arrhythmias**, explaining the episode of **supraventricular tachycardia and recurrent chest pain**. Calcium and phosphate levels are within normal limits and did not contribute to the neurological or cardiac complications.

Table 3: Cerebrospinal Fluid (CSF) Analysis.

Parameter	Observed Result	Normal Range	Status	Reason / Interpretation Related to Case
Appearance	Clear, colorless	Clear, colorless	Normal	Rules out gross infection or hemorrhage
Volume	2 mL	1–5 mL	Normal	Adequate sample for analysis
Gram Stain	No organisms	No organisms	Normal	Excludes bacterial meningitis
ZN Stain (AFB)	No AFB detected	No AFB	Normal	Rules out tubercular meningitis
Cell Count	2 lymphocytes	0–5 cells/mm³	Normal	Excludes acute CNS infection
CSF Glucose	67.5 mg/dL	45–80 mg/dL	Normal	Further supports non-infective pathology
CSF Protein	207 mg/L	150–450 mg/L	Mildly Elevated	Supports immune-mediated neuropathy seen in Guillain–Barré syndrome
CSF Chloride	117.4 mmol/L	110–125 mmol/L	Normal	No electrolyte-related CNS pathology

The **marked elevation of CK-MB and Troponin confirms acute myocardial injury**, most likely secondary to **autonomic nervous system involvement in Guillain–Barré syndrome and systemic metabolic stress**. The **low magnesium level further increases the risk of arrhythmias**, explaining the episode of **supraventricular tachycardia and recurrent chest pain**. Calcium and phosphate levels are within normal limits and did not contribute to the neurological or cardiac complications.

Table 4: Management.

Drug / Therapy	Dose	Frequency	Route	Indication	Reason / Case Correlation
NPO	—	—	—	Aspiration prevention	Kept NPO due to altered sensorium, dysphagia, and risk of aspiration in NCSE
IV DNS	50 ml/hr	Continuous	IV	Hydration	Maintained fluid balance during critical illness and NPO status
Ceftriaxone	1.5 g	1-0-1	IV	Infection	Empirical broad-spectrum antibiotic due to leukocytosis and suspected infection
Ranitidine	1 cc	1-0-1	IV	Gastric protection	Prevented stress-related gastric mucosal injury during critical illness
Amikacin	150 mg	1-1-1	IV	Infection	Added for extended gram-negative bacterial coverage
Acyclovir	600 mg	1-1-1	IV	Viral coverage	Started due to suspected viral trigger for neurological involvement
Levetiracetam (IV)	500 mg	1-0-1	IV	Seizures	Initiated for control of non-convulsive status epilepticus
Methylprednisolone	600 mg	OD	IV	Inflammation	Given to suppress immune-mediated inflammation seen in Guillain–Barré syndrome
3% Normal Saline	12 ml/hr	Continuous	IV	Hyponatremia	Corrected sodium imbalance contributing to altered sensorium and NCSE
Syp. Levetiracetam	3.5 ml	1-0-1	PO	Seizures	Continued seizure control after initial stabilization
Oral Feeds	—	—	PO	After swallow assessment	Restarted after improvement in swallowing and aspiration risk
Syp. Multivitamin	7 ml	OD	PO	Nutritional support	Provided to support recovery and general health

TREATMENT COURSE SUMMARY

Treatment was directed toward stabilization, infection control, seizure management, immune suppression, and correction of metabolic derangements**.** Antimicrobials were initiated due to significant leukocytosis and suspected infectious trigger. Antiepileptics were administered for non-convulsive status epileptics**.** Steroid therapy targeted the immune-mediated neuropathic process of Guillain–Barré syndrome. Hypertonic saline was used to correct hypernatremia, a major contributor to

altered consciousness and seizures**.** Gradual reintroduction of oral feeds was done after neurological and swallowing recovery.

DISCUSSION

Guillain–Barré syndrome (GBS) is an acute immune-mediated neuropathy that typically presents with progressive limb weakness and areflexia; however, atypical presentations with autonomic and central nervous system involvement can occur, especially in children. This case represents an unusual pediatric presentation of GBS complicated by autonomic instability, cardiac involvement, metabolic encephalopathy, and non-convulsive status epilepticus (NCSE).

The initial presentation with throat pain, dysphagia, chest pain, and breathlessness without early limb weakness delayed clinical suspicion of GBS. The presence of supraventricular tachycardia at admission indicates significant autonomic nervous system involvement, a known but potentially fatal complication of GBS. The markedly elevated CK-MB and troponin levels in this patient further support acute myocardial injury due to autonomic dysfunction and systemic stress rather than primary cardiac disease, as echocardiography was normal.

Marked leukocytosis suggests an underlying infectious trigger, which likely initiated the abnormal immune response leading to peripheral nerve involvement. Hepatosplenomegaly further supports a systemic inflammatory or infectious process preceding disease onset. Hyponatremia played a key role in this case by contributing to altered consciousness and triggering non-convulsive status epilepticus. Stress-induced hyperglycemia further aggravated the metabolic encephalopathy. These metabolic derangements significantly influenced the patient's rapid neurological deterioration.

The cerebrospinal fluid findings of normal glucose, very low cell count, and mildly raised protein are consistent with immune-mediated neuropathy and help exclude infectious meningitis. Early recognition of NCSE and prompt administration of antiepileptic therapy were crucial in preventing further neurological damage.

Management required intensive multidisciplinary care focusing on seizure control, correction of electrolyte abnormalities, stabilization of cardiac rhythm, suppression of immune-mediated inflammation, and treatment of possible infection. This case highlights that pediatric GBS may present with predominant autonomic and metabolic features rather than typical motor weakness. Early suspicion, continuous cardiac and neurological monitoring, and prompt correction of metabolic disturbances are essential to improve outcomes in such atypical and life-threatening presentations.

ACKNOWLEDGEMENT

The authors express their sincere gratitude to the **President, Soniya Education Trust's College of Pharmacy, Dharwad – 580002**, for providing the necessary institutional support and encouragement to carry out this work. The authors are also deeply thankful to the **Director and Head, Department of Pediatrics, Karnataka Medical College and Research Institute (KMC-RI), Hubballi – 580029**, for their valuable guidance, support, and cooperation during the course of this study. Their assistance and encouragement were instrumental in the successful completion of this work.

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