INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

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Case Study Article

Guillain–Barré Syndrome Presenting as Acute Flaccid Paralysis in a Child: A Case Report

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ARTICLE INFO

Published: 16 Jun. 2026
Keywords:
Guillain–Barré Syndrome (GBS), Acute Flaccid Paralysis, Pediatric Neuropathy, Intravenous Immunoglobulin (IVIG), Cerebrospinal Fluid Analysis, Motor Axonal Neuropathy
DOI:
10.5281/zenodo.20717261

ABSTRACT

Guillain–Barré syndrome is a rapidly evolving immune-mediated neuropathy and a leading cause of acute flaccid paralysis in children. We describe a twelve-year-old boy presenting with acute, progressive, symmetrical limb weakness following a recent febrile illness. Neurological examination revealed hypotonia, reduced muscle strength, and diminished deep tendon reflexes. Cerebrospinal fluid findings of elevated protein with normal cell count and electrophysiological evidence of motor axonal involvement supported the diagnosis. The patient received early treatment with intravenous immunoglobulin, accompanied by close clinical monitoring. Despite transient progression of weakness, there was no respiratory compromise or significant autonomic instability. Gradual neurological recovery was observed, with marked improvement by discharge and no requirement for ventilatory support. This case underscores the critical importance of early recognition, prompt immunotherapy, and vigilant monitoring in optimizing outcomes in pediatric Guillain–Barré syndrome.

INTRODUCTION

Guillain-Barré syndrome (GBS) is an uncommon, acute, inflammatory, immune-mediated disorder affecting the nerve roots and peripheral nerves, and it can be potentially life-threatening. It is recognized worldwide as a neuromuscular emergency and represents the most frequent cause of acute flaccid paralysis unrelated to poliovirus infection. Clinically, GBS commonly presents as a symmetrical, ascending sensorimotor weakness that begins with distal paraesthesia's, progresses to

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

weakness in the lower limbs, and may extend to the upper limbs and cranial nerves[1].

Acute flaccid paralysis (AFP) is a neurological condition marked by the sudden onset of weakness or paralysis in one or more limbs, and in some cases, the respiratory or bulbar muscles, along with reduced muscle tone, without any clear identifiable cause[2].

Most studies that estimate incidence rates of Guillain-Barré syndrome were done in Europe and North America, and showed a similar range of 0·8–1·9 (median 1·1) cases per 100 000 people per year.[15] The annual incidence rate of Guillain-Barré syndrome increases with age (0·6 per 100 000 per year in children and 2·7 per 100 000 per year in elderly people aged 80 years and over) and the disease is slightly more frequent in males than in females. Seasonal fluctuations, presumably related to variations in infectious antecedents, have been reported, but these observations are rarely statistically significant[3].

Guillain–Barré syndrome (GBS) is suspected in patients presenting with rapidly progressive bilateral limb weakness, often accompanied by sensory disturbances and diminished or absent deep tendon reflexes. Cranial nerve involvement, including facial weakness or bulbar palsy, as well as ophthalmoplegia and ataxia, may further support the clinical suspicion[4]

Guillain–Barré syndrome demonstrates considerable clinical variability in its presentation. In most patients, symptoms develop approximately 1–2 weeks following a preceding event, commonly an infectious illness, and progress to peak severity within two weeks. A very rapid progression within 24 hours or continued worsening beyond four weeks should prompt consideration of alternative diagnoses. Typically, the disease enters a plateau phase within one to four weeks after onset[1].

The diagnosis of GBS is primarily clinical and is established by fulfilling recognized diagnostic criteria while carefully excluding alternative causes of acute flaccid paralysis. Supportive investigations include routine laboratory tests to rule out other etiologies, cerebrospinal fluid (CSF) examination demonstrating characteristic findings, and electrophysiological studies to confirm peripheral nerve involvement and determine the subtype[4].

Intravenous immunoglobulin and plasma exchange are equally efficacious treatments for GBS[5]

Case Presentation

Patient Information

A 12-year-old male child was admitted to the Pediatrics Department at Karnataka Medical College and Research Institute (KMCRI), Hubballi on 2 January 2026 with complaints of weakness in both lower limbs for 2 days.

History of Presenting Illness

The child was apparently normal two days prior to admission. Subsequently, he developed sudden onset weakness in both lower limbs which gradually progressed. Initially, he experienced pain in both lower limbs, followed by difficulty in walking. By the day of admission, the child was unable to stand independently.

The weakness gradually progressed and involved the right upper limb, resulting in difficulty climbing stairs. There was a history of fever one week before the onset of weakness, which lasted for one day and subsided with symptomatic treatment. After the febrile episode, the child

experienced difficulty in getting up from a sitting position and climbing stairs.

There was no history of vomiting, loose stools, headache, convulsions, slurred speech, deviation of mouth angle, abnormal movements, loss of consciousness, head injury, or limb trauma. The child had no bowel or bladder incontinence, no recent vaccination, and no history of drug intake. There was no difficulty in breathing or performing fine motor activities such as writing or buttoning clothes. No diurnal variation of symptoms was reported.

Clinical Examination

Vital Signs

On admission, the patient's vital parameters were stable:

- Temperature: 36.8°C
- Pulse rate: 96 beats/min
- Respiratory rate: 24 breaths/min
- Blood pressure: 94/60 mmHg
- Oxygen saturation: 99% on room air
- Glasgow Coma Scale: E4 V5 M6 (15/15)

General Physical Examination

The child was conscious, cooperative, and oriented to time, place, and person. No pallor, icterus, cyanosis, clubbing, lymphadenopathy, or edema were observed.

Anthropometric measurements:

- Height: 130 cm
- Weight: 24.9 kg
- BMI: 14.7 kg/m²

No skeletal abnormalities were noted. Developmental milestones were appropriate for age, and immunization status was up to date.

Systemic Examination

Neurological Examination

- Cranial nerves: Normal
- Motor power:
  - Upper limbs: 4/5
  - Lower limbs: 2/5
- Muscle tone: Decreased
- Deep tendon reflexes: Diminished

Cardiovascular, respiratory, and abdominal examinations were within normal limits.

Investigations

Cerebrospinal Fluid Analysis

CSF examination revealed clear and colorless fluid with normal glucose levels, elevated protein (226 mg/L), and a normal cell count (3 cells/cumm), indicating albuminocytological dissociation, a characteristic finding in Guillain–Barré syndrome.

Gram stain, AFB stain, and CSF culture were negative, ruling out bacterial and tubercular meningitis.

Routine hematological and biochemical investigations were within normal limits except for mild hyponatremia, which may occur due to syndrome of inappropriate antidiuretic hormone secretion (SIADH) in Guillain–Barré syndrome.

Nerve Conduction Study

Nerve conduction study demonstrated reduced compound muscle action potential (CMAP) amplitude in the right peroneal nerve with preserved conduction velocity and distal latency. Sensory nerve conduction parameters were normal, and F-wave latencies were within normal limits. These findings suggested right peroneal

motor axonopathy without evidence of demyelination.

CT Cerebral and Neck Angiography

CT cerebral and neck angiography revealed patent extracranial and intracranial vessels without evidence of stenosis, occlusion, or aneurysm. No intracranial hemorrhage or acute infarction was observed, thereby excluding a vascular cause of acute limb weakness.

Management

The patient was treated with intravenous immunoglobulin (IVIG) at a dose of 400 mg/kg/day for 5 consecutive days. The infusion was administered over six hours with close monitoring of vital parameters. No infusion-related adverse reactions were observed.

During the first 48–72 hours of therapy, the patient showed progression of flaccid weakness predominantly affecting the lower limbs. However, there was no respiratory compromise, bulbar weakness, or significant autonomic instability.

Due to the risk of autonomic complications, the patient was monitored in the High Dependency Unit (HDU) but did not require ventilatory support.

Table 1: Management and Clinical Course of the Patient

Aspect	Details
Diagnosis	Guillain-Barré Syndrome with flaccid paralysis
Primary Treatment	Intravenous Immunoglobulin (IVIG)
IVIG Dose	400 mg/kg/day
Duration	5 consecutive days
Infusion Protocol	Administered over 6 hours daily
Monitoring During Infusion	Continuous monitoring of vital parameters
Infusion Reactions	No adverse reactions observed
Clinical Progress (48–72 hrs)	Progression of flaccid weakness (lower limbs predominance)
Respiratory Status	No respiratory compromise
Bulbar Involvement	Absent
Autonomic Instability	Not significant
Level of Care	Managed in High Dependency Unit (HDU)
Ventilatory Support	Not required
Supportive Care	Close neurological monitoring and observation for complications

Table 2: Clinical Course During Hospital Stay

Date	UL Power	LL Power	LL Tone	Reflexes	Key Events
03/01/2026	4/5	3/5	Decreased	Reduced	IVIG started (400mg/kg/day); in Ward
04/01/2026	4/5	3/5	Decreased	Reduced	IVIG day 2; in ward
05/01/2026	4/5	2/5	Decreased	Reduced	IVIG day 3;

					HDU admission for close monitoring
06/01/2026	4/5	2/5	Decreased	Reduced	IVIG day 4; Continued HDU monitoring
07/01/2026	4/5	3/5	Decreased	Reduced	IVIG completed; Shifted to Ward
08/01/2026	4/5	3/5	Improving	Improving	Motor recovery
09/01/2026	4/5	3+/5	Improving	Improving	Gradual Improvement
10/01/2026	4+/5	4/5	Improved	Improving	Marked neurological improvement following IVIG therapy

Outcome and Follow-Up

By the tenth day of hospitalization, the patient showed significant neurological improvement. Upper limb muscle strength improved to MRC grade 4+/5, while lower limb power improved to 3–4/5. Deep tendon reflexes gradually returned, and muscle tone normalized.

The patient remained hemodynamically stable throughout the hospital stay and did not require ventilatory support. He was discharged with advice for physiotherapy and regular follow-up.

Discharge medications included:

- Multivitamin syrup 5 mL orally twice daily
- Calcium syrup 8 mL orally three times daily

DISCUSSION:

Guillain–Barré syndrome (GBS) is an immune mediated disorder affecting the peripheral nerves, typically presenting with rapidly progressive symmetrical weakness of the limbs along with reduced or absent deep tendon reflexes[6]. GBS has emerged as one of the leading causes of acute flaccid paralysis (AFP) worldwide, especially after the eradication of poliomyelitis[7].

Acute flaccid paralysis is a clinical condition characterized by the sudden onset of weakness associated with decreased muscle tone without an obvious traumatic cause[8].

Clinically, the patient exhibited progressive weakness that began in the lower limbs and later involved the upper limbs, accompanied by hypotonia and diminished deep tendon reflexes. This pattern is characteristic of the classical ascending paralysis seen in GBS and fulfills the key diagnostic criteria[9]. the child developed neurological symptoms following a febrile episode one week prior, which is in line with the typical post-infectious pattern described in the literature[9].

Cerebrospinal fluid (CSF) examination revealed albuminocytological dissociation, evidenced by elevated protein levels with a normal cell count, which is a well-recognized diagnostic feature of GBS. This finding is consistent with previously reported literature and supports the clinical diagnosis in the present case[10].

Nerve conduction studies in this case showed motor axonopathy without evidence of demyelination, suggesting an axonal subtype of GBS, such as acute motor axonal neuropathy (AMAN). Axonal variants are increasingly recognized and are often linked to antecedent infections and immune-mediated axonal damage[11]

Early treatment with intravenous immunoglobulin (IVIG) is crucial for improving GBS outcomes. The patient received IVIG at a dose of 400 mg/kg/day for five days and showed gradual neurological improvement thereafter[12].

Hyponatremia is an clinical finding in Guillion-Barre syndrome, as autonomic dysfunction commonly seen and can lead to electrolyte imbalance such as SIADH. In our case, mild hyponatremia was observed, which is likely due to SIADH, a known association with GBS, This highlights the need for careful monitoring, preferably in a high-dependency setting, given the risk of autonomic instability[13].

CONCLUSION:

Acute flaccid paralysis in children is a serious neurological condition that requires prompt and accurate diagnosis to ensure effective treatment and prevent long-term disability. In this case, a thorough clinical assessment combined with targeted investigations enabled early identification of Guillain–Barré syndrome as the underlying cause. Early diagnosis is critical because it allows clinicians to initiate timely interventions that can halt disease progression and improve recovery outcomes.

The administration of intravenous immunoglobulin, along with vigilant clinical monitoring, played a pivotal role in the patient's favorable recovery without any complications. This case highlights the crucial role of early recognition and proactive management strategies in pediatric Guillain–Barré syndrome. By identifying the condition swiftly and starting appropriate treatment, healthcare providers can significantly reduce the risk of severe complications and enhance the overall prognosis for affected children.

ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to the **Director** and the **Head of the Department of Pediatrics** at **KMCRI (Karnataka Institute of Medical Sciences), Hubballi**, for their invaluable support, guidance, and for providing the necessary facilities to conduct this work.

Special thanks are also extended to the **President and Head of Department of Pharmacy Practice of SET's College of Pharmacy, Dharwad**, for his encouragement and for fostering an environment conducive to academic excellence and research

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HOW TO CITE: Megha Hiremath, Janaki Torvi, Adarsh G.S.*, Shivananda Illalu, Preeti Kulkarni, Venkatrao Kulkarni, Guillain–Barré Syndrome Presenting as Acute Flaccid Paralysis in a Child: A Case Report, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 6, 3912-3918. https://doi.org/ 10.5281/zenodo.20717261