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Case Series

Guillain-Barré syndrome in children: diverse clinical presentations and outcomes from a tertiary care hospital in central India

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ABSTRACT

Guillain-Barré syndrome (GBS) is an important cause of acute flaccid weakness in children, but its early manifestations may be atypical and may resemble muscle, nutritional, renal, or other systemic disorders. This retrospective observational case series describes eight children aged 4-16 years admitted with acute or subacute weakness to a tertiary care teaching hospital in Central India between January 2024 and June 2025. Demographic characteristics, clinical presentation, investigations, treatment, respiratory support, and outcome at discharge were reviewed. Six children (75%) were male and two (25%) were female; the median age was 12 years (range 4-16 years). Presentations included proximal or lower-limb weakness, gait disturbance, acute symmetrical weakness, and rapidly progressive quadriparesis or quadriplegia. The diagnostic evaluation identified electrophysiological or cerebrospinal-fluid evidence supporting GBS in four children, while other presentations included diagnostic mimics or cases without completed confirmation. Four children (50%) received intravenous immunoglobulin (IVIG), and two (25%) required respiratory support. Six children (75%) improved or recovered, one (12.5%) left against medical advice, and one (12.5%) died following severe disease complicated by ventilator-associated infection and sepsis. This series highlights the need to consider GBS in children with progressive weakness, while carefully distinguishing confirmed GBS from mimicking conditions. Neurological assessment, appropriate investigations, early recognition of respiratory compromise, and timely supportive and immunomodulatory management are important in optimizing outcomes.

Keywords: Guillain-Barré syndrome, Acute flaccid weakness, Childhood neuropathy, Intravenous immunoglobulin, Pediatric neurology, India

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute immune-mediated disorder of the peripheral nerves and nerve roots, classically characterized by progressive limb weakness, reduced or absent tendon reflexes, and variable sensory, cranial nerve, and autonomic involvement. It is an important cause of acute flaccid paralysis in the post-polio era. Population-based and clinical studies have described its epidemiology and broad clinical spectrum in children and adults.[1,2,11,14]

GBS often follows a respiratory or gastrointestinal infection. Molecular mimicry and cross-reactive immune responses may result in peripheral nerve injury, producing demyelinating or axonal forms of the disease. Although ascending weakness is a familiar pattern, children may initially present with limb pain, refusal to walk, gait disturbance, or nonspecific weakness, making early recognition difficult, particularly in younger children.[3-5,12,15]

The differential diagnosis of acute weakness in childhood is broad and includes myopathies, nutritional disorders,

and systemic illnesses. Clinical assessment, cerebrospinal fluid (CSF) analysis, electrophysiological studies, and, in selected cases, spinal magnetic resonance imaging (MRI) support diagnostic evaluation.[9,10,13]

The present case series describes eight children admitted with acute or subacute weakness at a tertiary care teaching hospital in Central India. It emphasizes the range of presentations encountered, the diagnostic challenges, management, and short-term outcomes, while distinguishing children with evidence supporting GBS from those included because of an alternative or unresolved diagnosis.

CASE SERIES

Study design and case selection

This retrospective observational case series was conducted in the Department of Pediatrics of a tertiary care teaching hospital in Central India. Medical records of children younger than 18 years admitted with acute or subacute weakness between January 2024 and June 2025 were reviewed. Eight children were included. Data extracted from the records comprised age, sex, antecedent illness, presenting symptoms, neurological examination, laboratory investigations, CSF findings, MRI, nerve conduction studies (NCS)/electromyography, treatment, respiratory support, and outcome at discharge. Children who initially raised a diagnostic possibility of GBS but were subsequently found to have a potential mimic, or in whom diagnostic confirmation was incomplete, were retained to reflect the diagnostic spectrum of acute weakness encountered in clinical practice.

Clinical presentation and investigations

The cohort comprised six boys (75%) and two girls (25%), with a median age of 12 years (range 4-16 years). The youngest child presented with proximal weakness and a waddling gait, with a markedly elevated creatine phosphokinase level (5,000 U/l), initially suggesting a myopathic process. A 15-year-old girl had lower-limb weakness with macrocytic anemia and nutritional deficiency. A 16-year-old boy developed acute symmetrical weakness following diarrhea but left the hospital against medical advice before diagnostic confirmation. A 12-year-old boy presented with fever, weakness, and cola-coloured urine; evaluation showed features of post-streptococcal glomerulonephritis.

Four children had findings supporting GBS on the available clinical, CSF, imaging, or electrophysiological assessment. A 10-year-old boy had acute quadriparesis with motor axonal polyneuropathy on NCS. A 12-year-old boy developed rapidly progressive quadriparesis and respiratory failure; CSF showed albuminocytological dissociation and NCS demonstrated a mixed axonal-demyelinating neuropathy.

A 9-year-old boy had lower-limb weakness and MRI evidence of thickened, enhancing cauda equina roots. A 13-year-old girl developed rapidly progressive quadriplegia; NCS showed an axonal neuropathy pattern.

Treatment and outcomes

Four children received IVIG. The child with severe disease and respiratory failure required prolonged invasive ventilation and tracheostomy; he subsequently developed ventilator-associated pneumonia and septicemia and died. Another child required non-invasive ventilatory support.

The remaining children received supportive care, supplementation where indicated, physiotherapy, and/or rehabilitation according to the clinical assessment. Six children improved or recovered by discharge, one left against medical advice, and one died.

Table 1: Aggregate demographic, clinical, management, and outcome characteristics (n=8).

Characteristic	Number (%) or summary
Age (years)	Median 12; range 4-16
4-7	1 (12.5)
8-11	2 (25.0)
12-15	4 (50.0)
16-17	1 (12.5)
Gender	
Male	6 (75.0)
Female	2 (25.0)
Clinical findings	
Proximal/lower-limb weakness or gait disturbance	3 (37.5)
Acute symmetrical limb weakness after diarrheal illness	1 (12.5)
Fever with weakness and renal presentation (PSGN features)	1 (12.5)
Acute quadriparesis/quadriplegia	3 (37.5)
Electrophysiological evidence of neuropathy (NCS)	3 (37.5)
CSF albuminocytological dissociation	1 (12.5)
MRI cauda equina root enhancement	1 (12.5)
Treatment and Outcome	
Received IVIG	4 (50.0)
Required respiratory support (invasive or non-invasive)	2 (25.0)
Improved/recovered by discharge	6 (75.0)
Left against medical advice	1 (12.5)
Death	1 (12.5)

IVIG: intravenous immunoglobulin; NCS: nerve conduction study; CSF: cerebrospinal fluid; MRI: magnetic resonance imaging; PSGN: post-streptococcal glomerulonephritis

DISCUSSION

This case series illustrates the heterogeneous presentation of acute weakness in children and the diagnostic difficulty of identifying GBS when the initial clinical picture is atypical. In this cohort, the median age was 12 years and 75% of patients were male. The male predominance is consistent with the recognized epidemiological pattern reported in population-based and pediatric studies, although the small sample size precludes meaningful comparison or inference about sex-specific risk.[11,14]

The clinical spectrum ranged from proximal or lower-limb weakness with gait disturbance to rapidly progressive quadriparesis or quadriplegia. Three children had severe generalized motor involvement, and two required respiratory support. Pediatric GBS may begin with pain, refusal to walk, or gait disturbance rather than the classic description of ascending paralysis; this is particularly relevant in younger children, in whom early symptoms may be difficult to characterize. The youngest child in our series had proximal weakness, waddling gait, and elevated creatine phosphokinase, initially suggesting myositis or myopathy. This observation underlines the importance of serial neurological examination and reconsideration of the diagnosis when weakness progresses.[4,15]

The cohort also included children with nutritional deficiency and post-streptococcal glomerulonephritis, as well as one child with a post-diarrheal acute weakness syndrome who left before diagnostic confirmation. These cases should be interpreted as part of the differential diagnosis of acute weakness rather than counted as confirmed GBS. This distinction is important because GBS diagnosis depends on the overall clinical pattern and supportive investigations, and alternative diagnoses must be considered. The Brighton diagnostic framework and contemporary diagnostic guidance emphasize clinical features, CSF findings, and electrophysiological evidence, while recognizing that investigations may be nondiagnostic early in the illness.[9,10,13]

NCS demonstrated motor axonal polyneuropathy in one child, mixed axonal–demyelinating neuropathy in another, and an axonal neuropathy pattern in a third. MRI showed cauda equina root enhancement in one child, while CSF albuminocytological dissociation was documented in the child with severe disease. GBS includes demyelinating and axonal subtypes, and electrophysiological classification can help characterize the neuropathy; however, early studies may require clinical correlation and, when indicated, repetition. The presence of axonal patterns in this small series is compatible with the recognized occurrence of axonal GBS variants, but this cohort is too small and selected to estimate their prevalence or compare it reliably with Western or Asian populations.[7,12]

IVIG was administered to four children (50% of the full acute-weakness cohort), corresponding to the four cases described as having findings supporting GBS. IVIG is an established immunomodulatory treatment for GBS, and evidence syntheses and clinical reviews support its use in patients with significant or progressive weakness. Treatment decisions should be based on clinical severity, progression, timing, and respiratory or bulbar involvement; corticosteroids alone are not an established effective treatment for typical GBS.[5,6,8,10]

Respiratory compromise occurred in two children (25%). One required non-invasive ventilation, whereas the other required prolonged invasive ventilation and tracheostomy. Respiratory failure is a major determinant of morbidity in GBS and warrants repeated assessment of respiratory muscle function, bulbar function, and autonomic status, with timely escalation of monitoring and ventilatory support when indicated. One child died after severe disease complicated by ventilator-associated pneumonia and septicaemia. Although the series cannot establish the contribution of individual complications to mortality, this outcome highlights the importance of preventing and promptly treating intensive-care-associated infections.[3,5,10]

Six children (75%) improved or recovered by discharge, one left against medical advice, and one died. These are short-term hospital outcomes and should not be interpreted as long-term neurological recovery. Previous clinical reviews describe variable recovery trajectories, with outcome influenced by disease severity, subtype, respiratory involvement, and complications.[2,4,12,14]

Limitations

The main limitations are the retrospective design, small sample size, incomplete diagnostic confirmation in some children, inclusion of acute weakness mimics within the clinical cohort, and absence of long-term follow-up. In addition, not all investigations were available for every child. Therefore, the aggregate percentages describe this eight-child acute-weakness cohort and should not be interpreted as estimates of GBS prevalence, subtype distribution, or treatment effectiveness.

CONCLUSION

Children with GBS may present with a wide range of symptoms, including gait disturbance, proximal weakness, and rapidly progressive quadriparesis with respiratory compromise. Acute weakness may also arise from nutritional or systemic conditions that mimic GBS, making careful clinical assessment and appropriate diagnostic confirmation essential. CSF analysis, electrophysiological studies, and selected imaging can support diagnosis. Early recognition of progression and respiratory involvement, timely IVIG in appropriate patients, and comprehensive supportive and critical care are important to optimize outcomes.

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