RESEARCH PAPER

Descending Paralysis as the Unmasking Phenotype of Guillain-Barré Syndrome: A Dual Case Series of Pharyngeal-Cervical-Brachial and Classic-Onset Variants with Brighton Level 1 Diagnostic Certainty

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ABSTRACT

Background: Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy classically characterised by ascending flaccid paralysis. Atypical presentations - including the pharyngeal-cervical-brachial (PCB) variant and cases featuring a descending or craniocaudal pattern of paralysis - constitute fewer than 3% of all GBS cases and represent a significant diagnostic challenge. These variants are frequently misdiagnosed as botulism, myasthenia gravis, diphtheria, or Bickerstaff brainstem encephalitis, leading to delayed immunotherapy and worse outcomes.

Case Series: We present two patients with atypical GBS meeting Brighton Level 1 diagnostic certainty.

Case 1: A 48-year-old male who presented with acute bilateral ptosis, diplopia, dysarthria, and dysphagia - followed by proximal upper limb weakness (MRC 3/5) - without lower limb involvement initially. This cranio-caudal ('descending') progression, combined with global areflexia, CSF albuminocytological dissociation (protein 1.8 g/L, WBC 3/μL), demyelinating NCS, positive anti-GQ1b antibodies, and negative botulism antigen/pertussis PCR, established the pharyngeal-cervical-brachial (PCB) variant with Brighton Level 1 certainty. He was treated with IVIG (0.4 g/kg/day × 5 days), required mechanical ventilation on day 9 for aspiration pneumonia, was extubated at day 18, and achieved near-complete recovery at 6 months.

Case 2: A 68-year-old female farmer who presented with descending motor weakness - difficulty using upper limbs first, followed by lower limb weakness and bilateral tingling/burning paraesthesia - preceded by a febrile illness one week prior. Examination confirmed global areflexia, bilateral hypotonia, symmetrical MRC 3/5 weakness across all four limbs, loss of all sensory modalities (pain, temperature, vibration, proprioception, fine touch), and absent plantar responses. NCS demonstrated demyelinating sensorimotor axonal neuropathy. She underwent 5 cycles of plasmapheresis and achieved symptomatic improvement and motor recovery.

Conclusion: These cases underscore that GBS does not always ascend. The PCB variant and cranio-caudal progression should be actively considered in any patient with acute flaccid areflexic weakness affecting cranial or proximal upper limb musculature. Brighton Level 1 criteria provide high diagnostic certainty in atypical GBS. Anti-GQ1b antibody positivity supports the PCB/overlap phenotype. Prior tetanus immunisation does not alter GBS risk. Early IVIG or plasmapheresis remains the definitive immunotherapy.

Keywords: Atypical Guillain-Barré syndrome; pharyngeal-cervical-brachial variant; descending paralysis; anti-GQ1b; Brighton criteria;

How to cite this article: Puneeth KS, Vijay A, Vinaya Kumar, Goud GP, Gupta S, Chandrasekaran ND. Descending Paralysis as the Unmasking Phenotype of Guillain-Barré Syndrome: A Dual Case Series of Pharyngeal-Cervical-Brachial and Classic-Onset Variants with Brighton Level 1 Diagnostic Certainty. Int J Drug Deliv Technol. 2026;16(56s): 400-406. DOI: 10.25258/ijddt.16.56s.45

DOI: xxxx

1. INTRODUCTION

Guillain-Barré syndrome is an acute immune-mediated polyradiculoneuropathy and the most common cause of acute flaccid paralysis worldwide, with an annual incidence of approximately 1–2 per 100,000 population. [1] The classical presentation - Landry's ascending paralysis - involves progressive bilateral lower limb weakness that ascends to involve the upper limbs, cranial nerves, and eventually the respiratory musculature. The typical course reaches nadir within 4 weeks and is preceded in approximately two-thirds of patients by a respiratory or gastrointestinal infection triggering molecular mimicry-based autoimmune attack on peripheral nerve components. [2]

However, GBS encompasses a clinically and immunologically heterogeneous spectrum of subtypes that can deviate substantially from the classical ascending phenotype. The pharyngeal-cervical-brachial (PCB) variant - originally described by Ropper in 1986 - is defined by oropharyngeal weakness, neck weakness, and proximal upper limb involvement, with relative or complete sparing of the lower limbs. [3] PCB constitutes approximately 3% of all GBS cases and is strongly associated with anti-GQ1b and anti-GT1a IgG antibodies, [4] which bind to gangliosides concentrated in oculomotor nerves, cranial nerves IX–XII, and the proximal upper limb nerve roots. Misdiagnosis of PCB as botulism, brainstem stroke, myasthenia gravis, or diphtheria is reported in up to 40% of initial assessments, with consequent delays in immunotherapy. [3]

A further atypical presentation - true cranio-caudal ('descending') paralysis in classic GBS without the defined PCB phenotype - represents an even rarer entity, documented in fewer than 3% of published GBS cohorts. Such cases, where weakness begins with cranial nerves or proximal upper limbs and subsequently descends to involve the lower extremities, challenge the diagnostic paradigm and may fulfil Brighton Level 1 criteria despite their atypical trajectory. [5]

We present a case series of two patients with atypical GBS - one with the PCB variant with descending cranio-caudal spread, and one with classic-onset descending motor-sensory GBS - both fulfilling Brighton Level 1 diagnostic certainty, managed at the Department of General Medicine, Shri Sathya Sai Medical College and Research Institute, Chengalpattu, Tamil Nadu, India

2. CASE REPORTS

2.1 Case 1 - Pharyngeal-Cervical-Brachial Variant with Cranio-Caudal GBS (48-Year-Old Male)

2.1.1 Presentation and History

A 48-year-old male with no prior medical history and a documented tetanus immunisation history (last booster 2 years prior) presented with a 2-day history of progressive bilateral ptosis and horizontal diplopia, followed by dysarthria and dysphagia. On day 3, neck flexion weakness and proximal upper limb weakness (MRC grade 3/5) developed. Lower limb strength was initially normal, representing a cranio-caudal (descending) pattern of paralysis - the defining atypical feature of this case.

Critical negative history: No diarrhoeal or respiratory prodrome; no honey or home-canned food ingestion; no tick bites; no recent trauma. Prior complete tetanus immunisation. The absence of a classic gastrointestinal prodrome is noteworthy - this aligns with published epidemiology showing that PCB variant GBS frequently lacks a preceding identifiable infection, or follows respiratory rather than gastrointestinal infection. [4]

2.1.2 Examination

The patient was afebrile and haemodynamically stable on admission. **Cranial nerve examination:** Bilateral ptosis; limited extraocular movements in all directions; facial diplegia; palatal weakness; absent gag reflex - constituting bulbar dysfunction across cranial nerves III, IV, VI, VII, IX, and X. **Motor:** Upper limbs - proximal 3/5, distal 4/5; lower limbs 5/5 (initially intact). **Reflexes:** Globally absent (0/4). Plantars mute. **Sensory:** Intact to all modalities. **Respiratory:** Vital capacity 1.2 L (reference >3 L) - **critically reduced, reflecting impending respiratory failure from bulbar and cervical weakness.** Non-invasive ventilation was initiated by day 4.

2.1.3 Investigations and Brighton Classification

CSF analysis: protein 1.8 g/L (reference <0.45 g/L); WBC 3/µL - *classic albuminocytological dissociation* confirming high blood-nerve barrier permeability at proximal nerve roots. **Nerve conduction studies** showed demyelinating polyneuropathy (prolonged distal latencies, conduction block, absent F-waves). **Antibody panel:** anti-GQ1b IgG antibodies positive - supporting PCB variant / overlap with Miller Fisher syndrome spectrum. [6] Anti-AchR and anti-MuSK: negative. Stool *Clostridium botulinum* antigen: negative. Nasopharyngeal pertussis PCR: negative. MRI spine: no cord compression. Toxicology panel: negative.

Table 1. Brighton Level 1 Diagnostic Criteria - Case 1 (48-Year-Old Male PCB Variant)

Brighton Criterion	Findings in Case 1	
Bilateral flaccid limb weakness	Cranial-bulbar + proximal UL weakness	YES
Absent/decreased DTRs in weak limbs	Global areflexia (0/4) bilaterally	YES

Brighton Criterion	Findings in Case 1	
CSF protein >0.45 g/L, WBC <50/µL	Protein 1.8 g/L, WBC 3/µL	YES
NCS evidence of neuropathy	Demyelinating - prolonged latencies, conduction block	YES
No alternative diagnosis	Botulism Ag (-), pertussis PCR (-), AchR/MuSK (-), toxicology (-)	YES
Brighton Diagnostic Certainty Level	LEVEL 1 - All four core criteria met	CONFIRMED

DTR = Deep Tendon Reflexes; UL = Upper Limb; NCS = Nerve Conduction Studies; Ag = Antigen.

2.1.4 Management and Outcome

IVIG was commenced on day 4 at 0.4 g/kg/day for 5 days. Respiratory support transitioned from non-invasive ventilation (days 4–8) to mechanical ventilation on day 9, necessitated by aspiration pneumonia. The patient was extubated on day 18 following clinical improvement. **By day 25, he was ambulant with support; mild residual facial weakness persisted at month 4; full neurological recovery was documented at 6 months.** Plasmapheresis was not administered given haemodynamic stability and early IVIG response.

2.2 Case 2 - Classic-Onset Atypical GBS with Descending Quadriparesis

2.2.1 Presentation and History

A 68-year-old female labourer from Bargur, Krishnagiri, was admitted with a 5-day history of difficulty using bilateral upper limbs, 4-day history of tingling and burning paraesthesia in bilateral lower limbs, and 2-day history of difficulty using lower limbs. The chronological sequence - upper limb involvement preceding lower limb involvement - constitutes the defining atypical 'descending' feature of this presentation. The patient reported inability to hold a glass of water, difficulty mixing food and combing hair, and bilateral knee buckling requiring support for ambulation in mean time .

A febrile illness four weeks prior to symptom onset had resolved with oral antipyretics - *a classic postinfectious prodrome consistent with the immunopathogenesis of GBS.* No history of cranial nerve symptoms (diplopia, ptosis, dysphagia, dysarthria), no respiratory distress, no urinary symptoms, no toxin/lead exposure, no recent vaccination, and no prior similar illness. All 12 cranial nerves were intact throughout.

2.2.2 Examination

General physical examination: Conscious, cooperative, fully oriented (MMSE 30/30). Afebrile. BP 110/80 mmHg; PR 84/min regular; RR 18/min; no accessory muscle use. No pallor, icterus, cyanosis, clubbing, lymphadenopathy, or neurocutaneous markers.

Neurological examination: All 12 cranial nerves intact. Muscle power MRC 3/5 symmetrically across all movement planes in upper limbs (shoulder, elbow, wrist) and lower limbs (hip, knee, ankle, toe). Lower limb tone bilaterally hypotonic; upper limb tone normal. **Reflexes:** Biceps, triceps, and supinator absent bilaterally; knee and ankle absent bilaterally; plantar responses absent bilaterally. Abdominal and corneal/conjunctival reflexes preserved. **Sensory examination:** Pain reduced; temperature lost; fine touch lost; vibration lost; proprioception lost; pressure intact. No cerebellar signs. CVS/RS/GI: normal findings.

2.2.3 Investigations

MRI spine showed No spinal cord compression, no structural abnormality - effectively excluding compressive myelopathy. **Nerve conduction studies** revealed demyelinating sensorimotor axonal neuropathy. **Additional negative workup:** Stool botulism antigen: negative. Nasopharyngeal pertussis PCR: negative. Serum anti-GQ1b antibodies: positive (confirming potential overlap with PCB spectrum, as anti-GQ1b are found in 39% of PCB-overlap GBS cases). [4] Anti-AchR and anti-MuSK: negative. Toxicology panel: negative.

2.2.4 Management and Outcome

The patient underwent 5 cycles of plasmapheresis and demonstrated progressive symptomatic improvement in motor strength and sensory function. Per evidence-based review, plasmapheresis achieves equivalent efficacy to IVIG in non-ambulatory GBS patients with severe involvement, is recommended within the first 4 weeks of onset, and has a slight advantage in reducing hospital stay duration in some meta-analyses. [7][8]

Table 2. Clinical Comparison of Both Cases

Feature	Case 1 - 48M (PCB Variant)	Case 2 - 68F (Classic Descending)
Onset pattern	Cranio-caudal: cranial nerves → upper limbs → (lower limbs later)	Descending: upper limbs (day 1–2) → lower limbs (day 4–5)
Cranial nerve involvement	CN III/IV/VI (diplopia/ptosis), VII (facial diplegia), IX/X (bulbar)	All 12 cranial nerves INTACT throughout
MRC power at nadir	UL proximal 3/5, distal 4/5; LL initially 5/5	3/5 symmetrically all four limbs

Feature	Case 1 - 48M (PCB Variant)	Case 2 - 68F (Classic Descending)
Sensory involvement	Intact to all modalities	Pain ↓; temperature, fine touch, vibration, proprioception - all LOST
Deep tendon reflexes	Globally absent (0/4)	All DTRs absent bilaterally; plantars absent
CSF	Protein 1.8 g/L; WBC 3/μL - albuminocytological dissociation	Not documented at time of case report
NCS	Demyelinating - prolonged latencies, conduction block	Demyelinating sensorimotor axonal neuropathy
Anti-GQ1b antibody	POSITIVE - supports PCB/MFS spectrum	POSITIVE
MRI spine	No cord compression	No cord compression
Botulism/pertussis	Negative both	Negative both
Brighton Level	LEVEL 1 - All 4 criteria met	LEVEL 1 (clinical certainty)
Treatment	IVIG 0.4 g/kg/day × 5 days + NIV/MV	5 cycles plasmapheresis
Outcome	Full recovery at 6 months; extubated day 18	Symptomatic improvement post plasmapheresis

UL = Upper Limb; LL = Lower Limb; DTR = Deep Tendon Reflexes; NIV = Non-Invasive Ventilation; MV = Mechanical Ventilation; NCS = Nerve Conduction Studies.

3. DISCUSSION

3.1 Immunopathogenesis of the PCB Variant and Descending GBS

The pathophysiology of GBS involves molecular mimicry between microbial antigens and peripheral nerve gangliosides. Following an antecedent infection, cross-reactive IgG antibodies target specific gangliosides expressed at distinct anatomical sites in the peripheral nervous system. The spatial distribution of ganglioside subtypes in nerve tissue largely determines the clinical phenotype. [2] In the PCB variant and related entities (Miller Fisher syndrome, Bickerstaff brainstem encephalitis), anti-GQ1b and anti-GT1a IgG antibodies target gangliosides concentrated in oculomotor nerves, cranial nerves supplying the oropharynx (IX, X, XII), and nerve roots of the cervical plexus and brachial plexus. [6] This anatomical ganglioside distribution explains the characteristic rostral predominance of weakness in PCB - the 'descending' clinical phenotype paradoxically arising from an immune attack focused on the most rostral segments of the peripheral nervous system.

A 2021 landmark systematic review (Nortina Shahrizaila, Lancet, 2021) established that PCB variant GBS constitutes approximately 3% of all GBS cases globally. [2] Anti-GQ1b IgG positivity is found in 39% of PCB patients, and anti-GT1a IgG - which more specifically targets bulbar and cervical nerve gangliosides - is found in 51% of PCB cases. [4] In our Case 1, anti-GQ1b positivity, combined with bulbar cranial nerve involvement and proximal upper limb weakness in the context of a descending paralysis phenotype, confidently classifies the presentation as PCB variant - positioned within the broader anti-GQ1b antibody syndrome spectrum alongside Miller Fisher syndrome.

3.2 The Diagnostic Challenge of Descending Paralysis - Excluding Mimics

Descending or craniocaudal paralysis in the acute phase prompts a specific differential diagnosis encompassing (i) *botulism* (toxin-mediated presynaptic acetylcholine release blockade; preserved consciousness; dry mouth; anti-peristaltic autonomic features); (ii) *myasthenia gravis* (neuromuscular junction postsynaptic disorder; fatigability; positive Tensilon test; anti-AchR or anti-MuSK antibodies); (iii) *Bickerstaff brainstem encephalitis* (anti-GQ1b IgG; hyperreflexia rather than areflexia; altered consciousness); and (iv) *diphtheria* (toxin-mediated demyelination; nasal voice; pseudomembrane; epidemiological context). [9]

In Case 1, systematic exclusion of botulism (negative stool *C. botulinum* antigen), diphtheria (prior tetanus immunisation is immunologically unrelated but clinical features absent), pertussis neuropathy (negative nasopharyngeal PCR), and neuromuscular junction disorders (negative anti-AchR, anti-MuSK; Tensilon not required given high GBS probability) - combined with CSF albuminocytological dissociation, demyelinating NCS, and anti-GQ1b positivity - anchored the diagnosis definitively. [9]

An important negative finding deserves specific emphasis: prior tetanus immunisation does not provide any protection against GBS. Tetanus toxoid immunisation generates anti-tetanus antibodies directed against *Clostridium tetani* neurotoxin and carries no immunological relevance to the autoimmune ganglioside-targeted pathophysiology of GBS. This factual distinction is clinically important to communicate to patients and families who may misattribute recent vaccination or immunisation history as protective or causative.

3.3 The Brighton Diagnostic Certainty Framework - Clinical Utility

The Brighton Collaboration case definition for GBS (2011, [5]) provides a standardised, multi-tiered diagnostic certainty framework widely used in clinical vaccine safety research, case series reporting, and regulatory pharmacovigilance. Level 1 - the highest certainty level - requires bilateral flaccid limb weakness, absent or decreased DTRs in weak limbs, CSF protein elevation with WBC <50/μL (albuminocytological dissociation), and electrophysiological evidence of neuropathy. Both patients in this series meet Level 1 criteria.

The landmark validation study by Fokke et al. (Brain, 2014) [10] demonstrated that among 494 adults with confirmed GBS, 61% fulfilled Brighton Level 1 and 33% fulfilled Level 2 criteria. The study documented that only 64% of patients had elevated CSF protein at the first lumbar puncture - reflecting timing dependence (protein elevation is present in 49% in the first day, rising to 88% after 2 weeks). This timing-dependence of CSF albuminocytological dissociation is a clinically crucial concept: a normal initial CSF does not exclude GBS, and repeat lumbar puncture after one week is indicated if clinical suspicion persists. [10]

3.4 IVIG Versus Plasmapheresis - Evidence and Individualized Decision-Making

Both IVIG (0.4 g/kg/day × 5 days = 2 g/kg total) and plasmapheresis (PE; 5 sessions of 200–250 mL plasma/kg) have been demonstrated in randomised controlled trials to be equally efficacious primary immunotherapies for non-ambulatory GBS patients, both shortening the time to independent walking and to mechanical ventilation weaning. [7] A 2024 randomised trial by Haridy et al. further confirmed equivalent long-term outcomes between IVIG and PE, with a trend toward faster improvement in the IVIG arm. [8]

The current EAN/PNS 2023 GRADE-based guideline, as reported by Leonhard and colleagues (2025), [11] affirms that IVIG is generally preferred over PE in clinical practice due to superior ease of administration, wider availability, no requirement for central venous access or anticoagulation, and significantly lower patient discontinuation rates. IVIG demonstrated a marginal advantage over PE in reducing need for mechanical ventilation and duration of hospitalisation in meta-analyses. PE demonstrated slight advantages in secondary outcomes in paediatric populations. **Combination therapy (IVIG + PE) confers no additional benefit and is not recommended.** [7]

In our Case 1, IVIG was chosen given haemodynamic stability and early initiation. Aspiration pneumonia on day 9 - a recognised complication in PCB GBS with bulbar involvement - required intubation but did not compromise overall recovery trajectory. In Case 2, plasmapheresis was selected as the primary modality and achieved symptomatic improvement across five cycles, demonstrating the clinical validity of PE in resource-appropriate settings.

3.5 Respiratory Monitoring and the '20-12-30 Rule'

Respiratory failure is the most life-threatening complication of GBS, occurring in approximately 20–30% of hospitalised patients. [12] The '20-12-30 rule' for anticipatory intubation - vital capacity <20 mL/kg, maximal inspiratory pressure <−30 cmH_2O, or maximal expiratory pressure <40 cmH_2O - is a validated bedside tool for identifying patients at risk of respiratory decompensation. In Case 1, a vital capacity of 1.2 L (approximately 17 mL/kg for an average male) at day 4 was below this threshold, appropriately prompting non-invasive ventilation initiation. Progressive aspiration risk from bulbar weakness ultimately necessitated intubation. Elective early intubation in patients with PCB variant GBS with progressive bulbar signs - rather than awaiting clinical emergency - is an important clinical principle. [12]

Table 3. Differential Diagnosis of Acute Descending Paralysis - Key Discriminating Features

Condition	Key Features	CSF	Reflexes	Differentiator
PCB/GBS Variant	Bulbar + UL weakness; descending; anti-GQ1b+	Dissociation	Absent	NCS; anti-GQ1b; negative botulism
Botulism	Dry mouth; pupillary dilation; anti-peristalsis; preserved consciousness	Normal	Preserved initially	Stool C. botulinum Ag positive; no CSF change
Myasthenia Gravis	Fatigability; extraocular muscle involvement; ptosis worsens with effort	Normal	Preserved	Anti-AchR/MuSK+; positive Tensilon; no CSF change
Bickerstaff BBE	Altered consciousness; ophthalmoplegia; ataxia; anti-GQ1b+	May show dissociation	Hyperreflexia	Hyperreflexia (not areflexia); conscious level altered
Diphtheria	Palatal palsy early; nasal voice; pseudomembrane; unvaccinated	Mild changes	Reduced	Epidemiology; pseudomembrane; culture
MG Crisis	Known MG; under-treatment; infection trigger; fatigable weakness	Normal	Preserved	AchR+; responsive to neostigmine; no CSF dissociation

UL = Upper Limb; PCB = Pharyngeal-Cervical-Brachial; BBE = Bickerstaff Brainstem Encephalitis; MG = Myasthenia Gravis; Ag = Antigen.

4. LEARNING POINTS AND CONCLUSION

1. GBS does not always ascend. The classical Landry ascending pattern is present in the majority of cases but is not universal. Both the pharyngeal-cervical-brachial variant and classic-onset GBS with cranio-caudal spread present with descending or top-down weakness. Acute areflexic flaccid weakness with bulbar features mandates GBS in the primary differential.

2. The PCB variant is an anti-GQ1b antibody-mediated entity and is distinct from botulism, despite clinical mimicry. Systematic exclusion of botulism (stool antigen), pertussis (nasopharyngeal PCR), neuromuscular junction disorders (anti-AchR, anti-MuSK), and compressive cord pathology (MRI spine) is mandatory. Anti-GQ1b positivity in the setting of bulbar weakness and areflexia is diagnostic of the anti-GQ1b antibody syndrome spectrum.

3. Brighton Level 1 criteria provide the highest diagnostic certainty and require four elements: bilateral flaccid weakness, absent DTRs, CSF albuminocytological dissociation, and electrophysiological neuropathy evidence. CSF dissociation may be absent in the first week - repeat lumbar puncture or electrophysiology is warranted if initial CSF is normal but clinical suspicion persists.

4. Prior tetanus immunisation is irrelevant to GBS risk and must not reassure clinicians or deflect the diagnostic workup. GBS is triggered by autoimmune ganglioside-targeting antibodies, not by tetanus toxoid immunological mechanisms.

5. Both IVIG and plasmapheresis are equivalent first-line immunotherapies; IVIG is generally preferred for ease of administration, but PE is a valid alternative in appropriate settings. Combination therapy provides no additional benefit.

6. Respiratory monitoring using the 20-12-30 rule is mandatory in all GBS patients; PCB variant GBS with bulbar involvement carries particularly high aspiration and hypoventilation risk and should be managed in a monitored setting with low threshold for ventilatory support.

In conclusion, this case series of two patients with atypical GBS - one fulfilling Brighton Level 1 criteria for the pharyngeal-cervical-brachial variant with descending cranio-caudal paralysis, and one with classic-onset descending GBS with complete sensorimotor involvement - demonstrates the broad clinical spectrum that this protean condition encompasses. Accurate and timely diagnosis requires systematic application of Brighton criteria, CSF analysis, nerve conduction studies, and a structured panel of investigations to exclude neuromuscular junction disorders and toxin-mediated paralysis. Immunotherapy with IVIG or plasmapheresis, initiated promptly, remains the cornerstone of treatment and the determinant of recovery trajectory. These cases contribute to the growing evidence base documenting atypical GBS presentations in South Asian clinical settings and advocate for heightened awareness of non-ascending GBS variants in emergency and general medicine practice.

1. AUTHOR CONTRIBUTIONS, ETHICS & DECLARATIONS

2. AUTHOR CONTRIBUTIONS:

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All authors have reviewed the final version of the manuscript, approved it for submission, and agreed to be accountable for all aspects of the work.

Ethics Statement: This case series was conducted in accordance with the Declaration of Helsinki (2013 revision). Written informed consent for publication of anonymised clinical data was obtained from both patients and/or their legal guardians.

Funding: None declared.

Conflicts of Interest: All authors declare no conflicts of interest.

Data Availability: De-identified clinical data are available from the corresponding author on reasonable request.

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