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Preserved Deep Tendon Reflexes with Prolonged Ventilator Dependence in a Child with Guillain-Barré Syndrome and an Acute Motor Axonal Neuropathy Pattern: A Case Report

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Abstract

Preserved or exaggerated deep tendon reflexes in acute motor axonal neuropathy (AMAN) have been associated with milder disease. A previously healthy 9-year-old boy presented two days after symptom onset with ascending quadriparesis, dysphagia and preserved deep tendon reflexes in all four limbs. Cerebrospinal fluid protein was 1.695 g/L with 3 cells × 10^6/L, and a single nerve conduction study on illness day 26 showed reduced compound muscle action potential amplitudes with normal sensory responses, consistent with an AMAN pattern on single-study criteria. His Erasmus Guillain-Barré Syndrome Respiratory Insufficiency Score (EGRIS) at admission was 6 (highest-risk category), and his paediatric EGRIS-Kids score was 8 of a possible 9. Despite intravenous immunoglobulin, he was intubated two days after admission and required 47 days of ventilatory support, a tracheostomy and drainage of a ventilator-associated pneumothorax, and was decannulated 67 days after intubation. At review 100 days after admission, reflexes were present and he was walking short distances unaided. Preserved reflexes neither exclude Guillain-Barré syndrome nor signal a mild course; in children with rapidly progressive weakness and bulbar signs, structured respiratory risk assessment rather than reflex status should guide monitoring, escalation and prompt immunotherapy.

Keywords

Guillain-Barré Syndrome, Acute Motor Axonal Neuropathy, Preserved Reflexes, Respiratory Insufficiency, Child

1. Introduction

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy and, since the near-eradication of poliomyelitis, the most common cause of acute flaccid paralysis in children [1]. It typically presents with rapidly progressive, symmetrical limb weakness and reduced or absent tendon reflexes, and remains a neurological emergency because of the risk of respiratory failure and autonomic dysfunction [1] [2]. Its annual incidence is approximately 0.81 - 1.89 per 100,000 [1], and a prospective electrophysiological study of 312 Bangladeshi patients confirmed that axonal subtypes are common in this region [3].

Reduced or absent reflexes in weak limbs form a core clinical feature of GBS and a required element of the Brighton case definition [4]. Nevertheless, preserved or exaggerated reflexes are well recognised, particularly in acute motor axonal neuropathy (AMAN). Kuwabara and colleagues found hyperreflexia in 7 of 54 consecutive Japanese patients (13%), mostly in the early recovery phase, and associated it with AMAN, anti-GM1 antibodies and milder peak disability [5]. A systematic review of 45 patients with hyperreflexic GBS reported that none required mechanical ventilation or died, and that most patients with limb weakness walked unaided within six months [6]. Proposed mechanisms include spinal motoneuron hyperexcitability and dysfunction of inhibitory interneuronal circuits [5] [6]. Reflex preservation has therefore come to be associated with a relatively benign phenotype.

Whether this association should influence bedside risk assessment in children is uncertain. The Erasmus GBS Respiratory Insufficiency Score (EGRIS) was developed in adults [7], and its paediatric counterpart, EGRIS-Kids, was derived because the adult model performs less accurately in children [8]; neither incorporates reflex status. We report a child with GBS, an AMAN pattern on a single nerve conduction study and preserved deep tendon reflexes whose course, comprising 47 days of ventilatory support, tracheostomy and a ventilator-associated pneumothorax, contrasts with the benign phenotype described above, and we interpret the case against validated respiratory risk tools and previous paediatric reports. The report follows the CARE guidelines.

2. Case Presentation

2.1. History and Examination

A previously healthy 9-year-old boy was admitted to the National Institute of Neurosciences and Hospital, Dhaka, on 30 November 2025 with a two-day history

of progressive bilateral lower-limb weakness that ascended to the upper limbs within 24 hours. He then developed dysphagia and a weak cough. There was no preceding febrile, diarrhoeal or respiratory illness, recent vaccination, sensory symptoms, or bowel or bladder dysfunction. Growth and development were age-appropriate, and there was no relevant family history.

He was conscious, oriented and haemodynamically stable, but tachypnoeic and required supplemental oxygen. The single-breath count was 10 - 12. Muscle tone was normal. Power was Medical Research Council (MRC) grade 3/5 in the upper limbs and 2/5 in the lower limbs (MRC sum score 30/60). Knee and ankle reflexes were present bilaterally (2+), and upper-limb reflexes were also present. There was no clonus or reflex spread, and plantar responses were equivocal. Sensory examination was normal. Bulbar dysfunction was present, without facial weakness or ophthalmoplegia. The GBS disability score was 4 (unable to walk).

At admission the EGRIS was 6 (days from onset to admission ≤ 3, 2 points; facial and/or bulbar weakness, 1 point; MRC sum score 21 - 30, 3 points), placing him in the highest-risk category (5 - 7), which corresponds to a predicted probability of about 65% of requiring mechanical ventilation within the first week of admission [7]. The EGRIS-Kids score, calculated retrospectively for this report, was 8 of a possible 9 (age 6 - 10 years, 1 point; cranial nerve involvement, 3 points; GBS disability score 4 at admission, 4 points) [8]. EGRIS-Kids has no published risk categories; in its derivation study, the predicted risk of respiratory failure during the disease course rose from 4% at the lowest score (1) to 50% at the highest (9) [8], so a score of 8 lies in the upper part of the predicted-risk range.

2.2. Cerebrospinal Fluid and Blood Tests

Lumbar puncture on 6 December 2025 (six days after admission; illness day 8) showed albuminocytological dissociation, with a protein concentration of 1.695 g/L and a leucocyte count of 3×10^6/L (**Table 1**), a finding supportive of GBS [1] [2]. Serum potassium was 3.58 mmol/L.

Table 1. Cerebrospinal fluid analysis.

Parameter	Result	Reference range
Appearance	Clear, colourless, no sediment	-
Red blood cells	0×10^6/L	0×10^6/L
Total leucocytes	3×10^6/L (100% lymphocytes)	0 - 5×10^6/L
Protein	1.695 g/L	0.15 - 0.45 g/L
Glucose	3.40 mmol/L	2.50 - 4.44 mmol/L
Paired blood glucose	5.24 mmol/L	-

2.3. Neuroimaging

Magnetic resonance imaging (MRI) of the brain and whole spine was performed on the same day (illness day 8) to evaluate central causes of acute flaccid weakness,

particularly acute transverse myelitis, acute flaccid myelitis and brainstem pathology. Brain MRI, including diffusion-weighted and contrast-enhanced sequences, showed no infarction, haemorrhage, demyelination, abnormal enhancement or structural lesion (Figure 1). Sagittal T2-weighted images of the whole spine showed normal cord signal without compression, disc herniation or epidural pathology (Figure 2). Nerve-root enhancement could not be assessed because

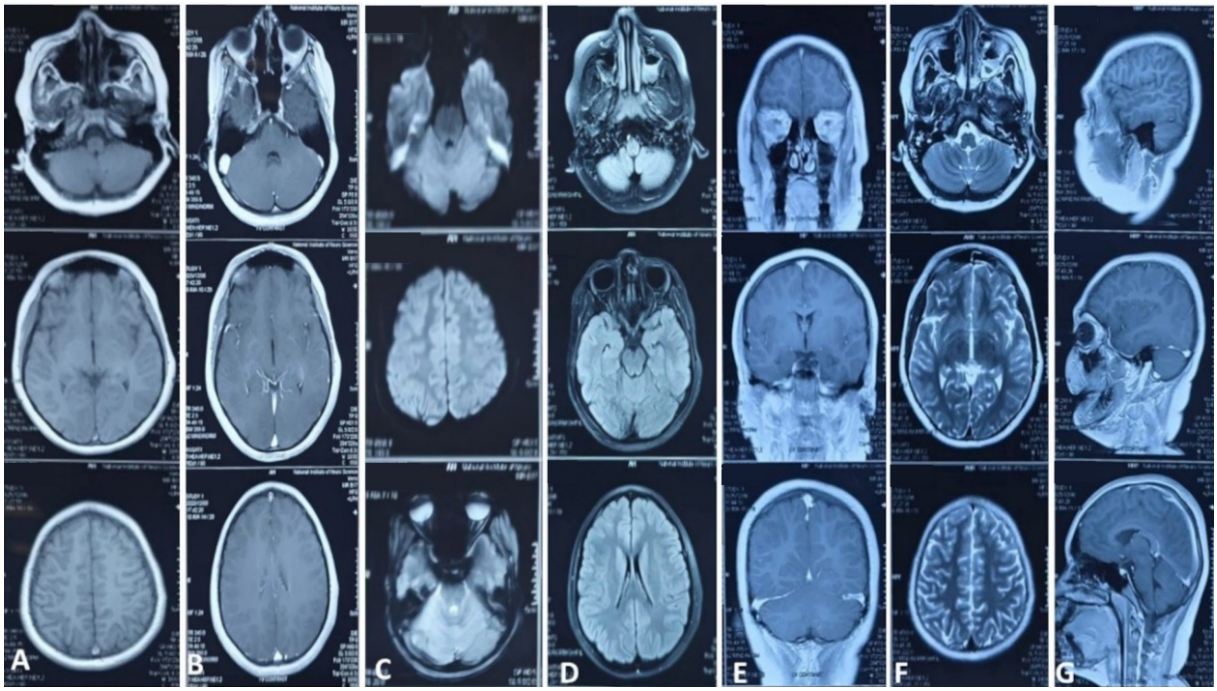


Figure 1. Brain MRI. Each column shows three representative slices of one sequence: (A) axial T1-weighted; (B) axial contrast-enhanced T1-weighted; (C) axial diffusion-weighted imaging (upper two rows) and apparent diffusion coefficient map (lower row); (D) axial T2 fluid-attenuated inversion recovery; (E) coronal contrast-enhanced T1-weighted; (F) axial T2-weighted; (G) sagittal contrast-enhanced T1-weighted. No restricted diffusion, parenchymal signal abnormality, abnormal parenchymal or leptomeningeal enhancement, or structural lesion is seen.

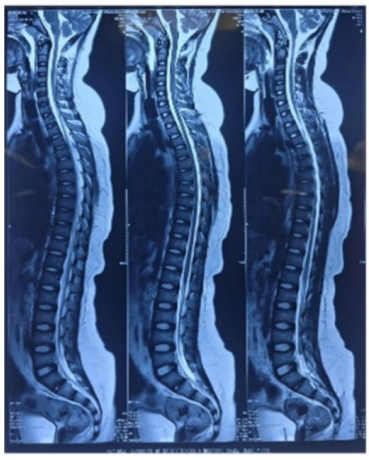


Figure 2. Sagittal T2-weighted MRI of the whole spine (three adjacent sagittal slices, cervical to sacral levels) showing normal vertebral alignment and normal cord signal, with no cord compression, disc herniation or epidural collection. Contrast-enhanced spinal images were not obtained.

post-contrast spinal images were not obtained. These findings made a structural brain or spinal cord cause unlikely but do not by themselves exclude all central nervous system disorders; normal early imaging has been described in acute flaccid myelitis, and the diagnosis rested on the combined clinical, cerebrospinal fluid and electrophysiological findings.

2.4. Nerve Conduction Studies

A single study was performed on 24 December 2025 (illness day 26) using a Nihon Kohden Neuropack system at a limb skin temperature of ≥32˚C, and was interpreted against the in-house paediatric reference limits of the neurophysiology laboratory of the National Institute of Neurosciences and Hospital, matched to the patient's age (9 years). Compound muscle action potential (CMAP) amplitudes were reduced in the left median, both ulnar and the right peroneal nerves; distal motor latencies and conduction velocities were within reference limits (Table 2). No nerve showed conduction block or abnormal temporal dispersion, defined, respectively, as a proximal-to-distal CMAP amplitude ratio < 0.50 and an increase in proximal CMAP duration > 30%. Minimum F-wave latencies in the upper limbs were mildly prolonged (115% - 118% of the upper reference limit, below the 120% threshold used as a demyelinating criterion), F-wave persistence was markedly reduced, and only an M response was obtained from the peroneal nerve. Sensory nerve action potential amplitudes and sensory conduction velocities were normal in all nerves tested (Table 3). The findings fulfilled the single-study criteria of Rajabally *et al.* for axonal GBS [9], with an AMAN pattern. Because no follow-up study was performed, reversible conduction failure could not be distinguished from axonal degeneration [10]; the classification therefore rests on a single study and is referred to as an AMAN pattern throughout this report.

Table 2. Motor nerve conduction studies (illness day 26). Abnormal values are shown in bold.

Nerve (recording site)	Stimulation sites	DML (ms)	CMAP amplitude (mV), distal/ proximal	CMAP dur. (ms)	CV (m/s)	F-wave min. latency (ms)/persistence (%)	CB/TD
L median (APB)	Wrist/elbow	2.8	**1.2/1.1**	4.2	52	28.4/30	Absent/absent
L ulnar (ADM)	Wrist/below elbow	2.2	**0.9/0.8**	3.9	55	27.6/20	Absent/absent
R ulnar (ADM)	Wrist/below elbow	2.1	**0.8/0.7**	4.0	54	27.9/20	Absent/absent
R peroneal (EDB)	Ankle/fibular head	3.5	**0.4/0.3**	4.9	46	M response only/0	Absent/absent
Reference limits	-	<3.8	>4.0 (upper limb)/>2.0 (lower limb)	<5.5	>50 (upper limb)/>45 (lower limb)	<24.0 (upper limb)/<32.0 (lower limb); persistence > 80	-

DML, distal motor latency; CMAP, compound muscle action potential; dur., duration; CV, conduction velocity; CB, conduction block; TD, temporal dispersion; APB, abductor pollicis brevis; ADM, abductor digiti minimi; EDB, extensor digitorum brevis; L, left; R, right. Reference limits: in-house age-matched paediatric values of the neurophysiology laboratory, National Institute of Neurosciences and Hospital (Section 2.4).

Table 3. Sensory nerve conduction studies (illness day 26).

Nerve	Onset latency (ms)	SNAP amplitude (μV)	CV (m/s)
L median	1.8	24.5	56
L ulnar	1.6	21.0	58
R sural	2.1	18.2	50
Reference limits	<2.5	>15.0	>45.0

SNAP, sensory nerve action potential; CV, conduction velocity; L, left; R, right. Reference limits: in-house age-matched paediatric values of the neurophysiology laboratory, National Institute of Neurosciences and Hospital (Section 2.4).

Anti-ganglioside antibody testing and stool culture for *Campylobacter jejuni* were not available.

2.5. Differential Diagnosis

Rapidly progressive ascending weakness with bulbar involvement and albuminocytological dissociation strongly suggested GBS, but other causes of acute flaccid weakness in a child were considered. Acute transverse myelitis and acute flaccid myelitis were made unlikely by the absence of a sensory level, sphincter involvement or cerebrospinal fluid pleocytosis, and by normal cord signal on MRI. Hypokalaemic paralysis was made unlikely by a serum potassium within the reference range. Myasthenic crisis was considered because of bulbar weakness, but the ascending pattern, absence of ptosis or ophthalmoplegia, raised cerebrospinal fluid protein and reduced CMAP amplitudes with normal conduction pointed away from a neuromuscular junction disorder; repetitive nerve stimulation was not performed. Botulism was unlikely because weakness ascended rather than descended, there was no ptosis, ophthalmoplegia or pupillary involvement, there was no food or wound exposure, and cerebrospinal fluid protein is typically normal in botulism. Poliomyelitis and other enterovirus-associated acute flaccid paralysis were considered because of their regional importance, but the absence of fever, the symmetrical weakness, the normal cerebrospinal fluid cell count and the electrophysiological pattern argued against anterior horn cell disease; stool samples for poliovirus under national acute flaccid paralysis surveillance were not collected. Diphtheritic polyneuropathy was unlikely without preceding pharyngitis, pharyngeal membrane or myocarditis. The final diagnosis was GBS with an AMAN pattern on a single nerve conduction study.

2.6. Treatment and Clinical Course

Intravenous immunoglobulin (IVIG) 0.4 g/kg/day (12 g/day for a body weight of 30 kg) for five days (total 2 g/kg; 60 g) was started on clinical grounds on 1 December 2025 (illness day 3), before cerebrospinal fluid and electrophysiological confirmation, in line with current guideline recommendations [11]. Respiratory function nevertheless deteriorated, with increasing respiratory distress and a fall-

ing single-breath count. The decision to intubate was based on this clinical deterioration. The serial single-breath counts, respiratory rate, oxygen saturation and oxygen requirement immediately before intubation were not documented in the case records, and no arterial blood gas, vital capacity or maximal respiratory pressure measurements were recorded. He was intubated and mechanically ventilated on 2 December 2025 (ventilation day 1), two days after admission. Because weaning was not possible, a tracheostomy was performed on 1 January 2026 (ventilation day 31).

On 17 December 2025 (ventilation day 16), a right-sided pneumothorax was identified during positive-pressure ventilation. At that time, he was ventilated in pressure control-assist/control mode with a delivered tidal volume of approximately 210 mL (7 mL/kg for a body weight of 30 kg), a positive end-expiratory pressure of 6 cmH_2O and a peak inspiratory pressure of 22 cmH_2O. No right-sided central venous cannulation had been performed in the preceding 48 hours, and the pneumothorax was attributed to barotrauma. A right intercostal chest drain was inserted, and the next day's radiograph showed re-expansion of the right lung (Figure 3). The drain was removed on 22 December 2025.

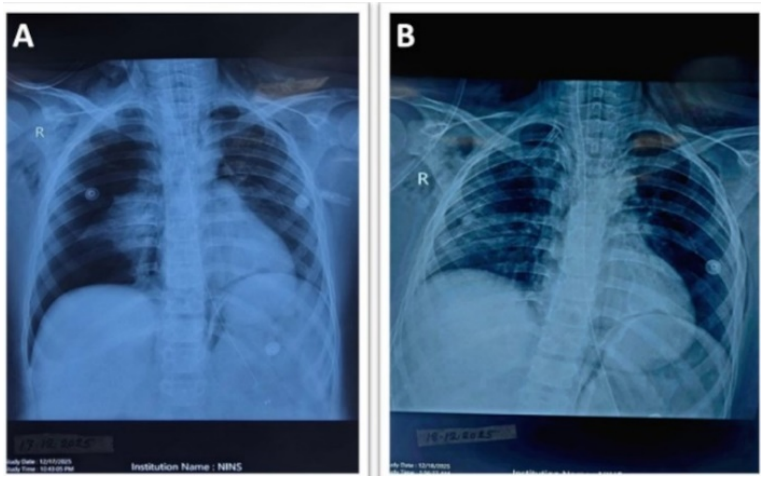


Figure 3. Portable anteroposterior chest radiographs. (A) 17 December 2025 (ventilation day 16): right-sided pneumothorax with a visible visceral pleural line and absent peripheral lung markings in the right hemithorax. (B) 18 December 2025, after insertion of a right intercostal chest drain: re-expansion of the right lung with the drain in situ and no residual pneumothorax.

2.7. Outcome and Follow-Up

He received 47 days of ventilatory support: mechanical ventilation in the intensive care unit from 2 December 2025 to 10 January 2026 (39 days), followed by ventilation through the tracheostomy on a portable ventilator in the ward from 10 to 18 January 2026 (8 days), after which he was weaned successfully. Recovery of bulbar function allowed nasogastric feeding to stop on 22 January 2026. At discharge on 25 January 2026, he was breathing spontaneously through the tracheostomy without ventilatory support, feeding orally, and had improved limb strength

(MRC sum score 42/60; GBS disability score 4); physiotherapy continued. He was decannulated without respiratory compromise on 7 February 2026, 67 days after intubation. At the latest review on 10 March 2026 (100 days after admission) the stoma had healed, respiratory function was normal and neurological recovery was continuing: the MRC sum score was 54/60, he was able to walk 5 m unaided but not 10 m (GBS disability score 3), and deep tendon reflexes were present (2+) bilaterally. The clinical course is summarised in Table 4.

Table 4. Timeline of the clinical course.

Date (day)	Event
28 Nov 2025 (illness day 0)	Onset of bilateral lower-limb weakness
30 Nov 2025 (illness day 2)	Admission: ascending quadriparesis, dysphagia, single-breath count 10 - 12, preserved deep tendon reflexes; EGRIS 6; EGRIS-Kids 8
1 Dec 2025 (illness day 3)	IVIG 2 g/kg over 5 days started
2 Dec 2025 (ventilation day 1)	Intubation and mechanical ventilation in the intensive care unit
6 Dec 2025 (illness day 8)	Lumbar puncture (protein 1.695 g/L, 3 cells × 10^6/L); MRI brain and whole spine
17 Dec 2025 (ventilation day 16)	Right pneumothorax; right intercostal chest drain inserted
22 Dec 2025	Chest drain removed
24 Dec 2025 (illness day 26)	Nerve conduction study (AMAN pattern)
1 Jan 2026 (ventilation day 31)	Tracheostomy
10 Jan 2026	Transferred to ward on portable ventilator via tracheostomy
18 Jan 2026	Weaned from ventilation (47 days of ventilatory support in total)
22 Jan 2026	Nasogastric feeding discontinued
25 Jan 2026	Discharged home with tracheostomy
7 Feb 2026	Decannulation (67 days after intubation)
10 Mar 2026	Follow-up: stoma healed; continued neurological recovery; reflexes present

Illness days are counted from symptom onset (28 November 2025 = day 0) and ventilation days from intubation (2 December 2025 = day 1); durations are expressed as elapsed days. EGRIS, Erasmus GBS Respiratory Insufficiency Score; IVIG, intravenous immunoglobulin; MRI, magnetic resonance imaging; AMAN, acute motor axonal neuropathy.

3. Discussion

Preserved reflexes in AMAN are not new, and this report does not claim otherwise. Its contribution is narrower: it documents a severe, prolonged respiratory course in a child whose reflexes were preserved at presentation, a combination that runs against the benign phenotype described in the literature, and it shows that the established, adult-derived respiratory risk score placed him in its highest-risk category for early mechanical ventilation even though reflex status argued for reassurance.

In the original series, hyperreflexic patients had milder peak disability than those without hyperreflexia [5], and none of the 45 patients in the later systematic

review required mechanical ventilation [6]. Among the paediatric reports we identified (**Table 5**) [12]-[15], one described intubation in a 3-year-old with exaggerated reflexes [15], confirming that serious respiratory involvement can occur; however, courses were generally short, and none described ventilation lasting several weeks or tracheostomy. Two qualifications apply. First, our patient had preserved rather than exaggerated reflexes, and the benign association is best documented for hyperreflexia. Second, **Table 5** is based on a targeted rather than a systematic search, so earlier similar cases cannot be excluded.

Table 5. Selected paediatric reports of Guillain-Barré syndrome with preserved or exaggerated reflexes.

Report	Age, sex	Subtype	Reflexes	Treatment	Respiratory course/outcome
Singhal and Bhat, 2011 [12]	10 y, M	Acute motor conduction block neuropathy	Brisk throughout; extensor plantars	IVIG + IV methylprednisolone	Ventilation not reported; walking with support at discharge
Tellier *et al.*, 2012 [13]	10 y, M	AMAN; anti-GM1, anti-GD1b IgG	Preserved	IVIG	Ventilation not reported; favourable
Zouiri *et al.*, 2016 [14]	2.5 y, F	AMAN	Preserved	IVIG	Ventilation not reported; complete motor recovery
Abuhalima *et al.*, 2025 [15]	3 y, F	AMAN	Exaggerated	IVIG	Intubated; walking with assistance by day 9
Present case	9 y, M	AMAN pattern (single study)	Preserved (all four limbs)	IVIG	47 days of ventilatory support, tracheostomy, pneumothorax; decannulated 67 days after intubation

"Ventilation not reported" indicates that this information was not available in the sources consulted. AMAN, acute motor axonal neuropathy; IVIG, intravenous immunoglobulin; IV, intravenous; y, years; M, male; F, female.

The practical lesson concerns risk assessment. At admission this child scored on all three EGRIS components (rapid onset, bulbar weakness and severe limb weakness), giving a score of 6, in the category with a predicted probability of about 65% of mechanical ventilation within the first week [7]; he was intubated two days after admission. His EGRIS-Kids score, calculated retrospectively, was 8 of a possible 9, reflecting cranial nerve involvement and inability to walk. EGRIS-Kids has no formal risk categories, but its predicted risk of respiratory failure rose from 4% to 50% across the score range, against an overall rate of 19% in its derivation cohort of 421 children [8]. Both scores estimate the risk of needing mechanical ventilation, EGRIS within the first week of admission [7] and EGRIS-Kids during the disease course [8]; neither predicts the duration of ventilation, the need for tracheostomy or ventilator-associated complications, so this child's prolonged and complicated course lies outside what either score was designed to anticipate. Neither tool includes reflex status, and this case suggests they are right not to. A clinician who reads preserved reflexes as reassuring, or as a reason to question the diagnosis and defer treatment, risks under-monitoring precisely the patient who needs escalation. Serial single-breath counts, and vital capacity where it can be

measured in children, should guide the timing of intubation regardless of reflex findings.

The case also bears on diagnostic criteria. The Brighton case definition requires decreased or absent reflexes in weak limbs for diagnostic certainty levels 1 - 3 [4]. Our patient did not meet this element at presentation, and reflexes were again present at follow-up; on the basis of these documented examinations, he would not have reached Brighton levels 1 - 3. Uncini *et al.* have argued that the possibility of hyperreflexia should be written into future diagnostic criteria [6]. This matters beyond bedside diagnosis, because such case definitions are used in surveillance and in trial eligibility.

Several aspects of pathophysiology cannot be resolved from this case. AMAN encompasses both reversible conduction failure at the nodes of Ranvier, which usually recovers quickly, and axonal degeneration, which recovers slowly; the two can be separated only by serial studies [10], which is why the case is described as an AMAN pattern rather than a definitive subtype. The need for 47 days of ventilatory support and the gradual bulbar recovery are more in keeping with axonal degeneration, but this remains an inference. Hyperreflexia has been linked to anti-GM1 antibodies [5], and neither antibody testing nor H-reflex studies were available, so the proposed mechanisms of motoneuron hyperexcitability and interneuronal dysfunction [6] were not tested here.

Tracheostomy was performed on ventilation day 31, after an unsuccessful weaning period. In adults, the inability to lift the arms from the bed one week after intubation predicts prolonged ventilation and supports earlier tracheostomy [16]; no equivalent validated tool exists for children. The pneumothorax occurred with a tidal volume of approximately 7 mL/kg, a peak inspiratory pressure of 22 cmH_2O and a positive end-expiratory pressure of 6 cmH_2O, settings within the ranges generally regarded as lung-protective. Barotrauma can nonetheless occur with apparently protective settings in patients who require prolonged positive-pressure ventilation, and close monitoring of respiratory mechanics remains important in this group.

Limitations

This report has important limitations. Only a single nerve conduction study was performed, so the evolution of electrophysiological abnormalities and the distinction between reversible conduction failure and axonal degeneration could not be assessed; the AMAN classification is therefore provisional. Anti-ganglioside antibodies and *C. jejuni* testing were unavailable, so the case adds no molecular information to the established association between reflex preservation, AMAN and anti-GM1 antibodies. Reflexes were examined at admission and at follow-up but were not graded serially during the ventilated period. EGRIS-Kids was calculated retrospectively. The respiratory findings immediately before intubation, including serial single-breath counts, oxygen requirement, arterial blood gas values and vital capacity, were not documented, so the timing of intubation cannot be related to

objective respiratory measurements. Contrast-enhanced spinal imaging was not performed to evaluate nerve-root enhancement, and clinical follow-up extends to only 100 days after admission. As a single case, it cannot establish how often reflex preservation accompanies severe respiratory involvement.

4. Conclusion

Preserved deep tendon reflexes neither exclude GBS nor indicate a mild course. They should not delay the diagnosis or treatment of GBS in a child with rapidly progressive weakness and bulbar signs. In this child with an AMAN pattern, respiratory risk scores that disregard reflex status placed him in the highest EGRIS category and near the top of the EGRIS-Kids range for mechanical ventilation, and he was intubated two days after admission; these scores estimate the risk of needing ventilation, not its duration or complications. Respiratory risk, not reflex status, should drive monitoring, escalation and prompt immunotherapy.

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Author Contributions

Conceptualization: Abu Naser Md. Faiaz, Nitisha Najiat. Investigation: Abu Naser Md. Faiaz. Data curation: Emtiazur Rahman. Visualization: Mohiuddin Ahmad Nasid. Supervision: Nitisha Najiat. Writing - original draft: Abu Naser Md. Faiaz. Writing - review and editing: Mohiuddin Ahmad Nasid, Emtiazur Rahman. All authors read and approved the final manuscript.

Ethics Statement

Formal institutional ethics committee approval was not required for this single-patient case report, in accordance with the policy of the National Institute of Neurosciences and Hospital, Dhaka, Bangladesh. The report was prepared in line with the principles of the Declaration of Helsinki.

Consent for Publication

Written informed consent was obtained from the patient's parents for publication of this case report and the accompanying images.

Data Availability Statement

Data sharing is not applicable to this article because no datasets were generated or

analysed.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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