

CASE REPORT

Rapid Ventilator Weaning and Neuromuscular Recovery After a Single Large-Volume Therapeutic Plasma Exchange in Acute Motor Axonal Neuropathy: A Case Report

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ABSTRACT

Background. Acute motor axonal neuropathy (AMAN) is an antibody-mediated variant of Guillain-Barré syndrome (GBS) that can cause respiratory failure. Guidelines recommend a course of several therapeutic plasma exchange (TPE) sessions. Ventilator weaning after one session has rarely been reported.

Objective. To describe the clinical course, ventilator weaning, and recovery of one patient with AMAN after a single large-volume TPE session.

Methods. This case report follows the CARE guideline. A 68-year-old, previously independent man had three weeks of ascending quadriparesis. Cerebrospinal fluid showed albuminocytologic dissociation, and nerve conduction studies showed absent motor responses with preserved sensory responses. On Day 4 his Medical Research Council sum score reached a nadir of 12/60. He was intubated. On Day 5 a single TPE session exchanged 2,500 mL of plasma (1.08 plasma volumes) with albumin and saline.

Results. Dynamic lung compliance rose by 81.4% (31.7 to 57.5 mL/cmH₂O) by Day 6. He was extubated on Day 7. He left the intensive care unit on Day 8, and his GBS disability score fell from 5 to 4 at extubation and to 3 at hospital discharge on Day 21. No antibody, complement, or repeat nerve-conduction data were available.

Conclusion. In this patient, ventilator weaning and strength recovery followed a single large-volume TPE session, but one case cannot show cause or support replacing multi-session TPE. The mechanism remains hypothetical.

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1. Introduction

Guillain-Barré syndrome (GBS) is a diverse group of acute, post-infectious, immune-mediated polyradiculoneuropathies, and it is the leading cause of non-traumatic acute flaccid paralysis worldwide.¹ The estimated annual incidence ranges from 0.4 to 4.0 cases per 100,000 individuals. The disorder crosses demographic boundaries. Its age distribution is bimodal, with one peak in young adulthood and a steady rise among older people, and a modest male predominance has been documented.^{2,3} Many patients regain meaningful function. Recovery takes several months to years. Even so, the syndrome still causes severe morbidity, long stays in the intensive care unit (ICU), and death.^{1,4} Up to 30% of hospitalized patients develop acute respiratory failure that requires endotracheal intubation and invasive mechanical ventilation. Overall mortality stays between 5% and 10% in high-resource tertiary centers, but it exceeds 15% in resource-limited developing countries.^{5,6}

The European Academy of Neurology (EAN) and the Peripheral Nerve Society (PNS) recognize several electrophysiological and pathological variants of GBS.^{7,8} Acute inflammatory demyelinating polyneuropathy (AIDP) predominates in North America and Europe. Its classic

model is T-cell-mediated, macrophage-driven stripping of myelin sheaths.^{9,10} Asian, Latin American, and tropical regions differ. There, primary axonal variants, namely acute motor axonal neuropathy (AMAN) and acute motor-sensory axonal neuropathy, make up as many as 30% to 65% of documented cases.^{9,11}

AMAN is an antibody-mediated autoimmune disorder. High-affinity immunoglobulin G (IgG) autoantibodies target specific gangliosides, including GM1 and GD1a, which cluster densely along the motor axolemma at the nodes of Ranvier and the paranodal junctions.^{10,12} Antecedent microbial pathogens carry surface lipo-oligosaccharides that mimic these gangliosides, and the mimicry yields cross-reactive antibodies. They recruit C1q. The classical complement cascade then starts on the nodal axolemmal membrane.^{5,10} The membrane attack complex (MAC; C5b-9) then assembles in its terminal form and disrupts the clustering of voltage-gated sodium channels (Nav1.6). Conduction fails, and rapid motor paralysis follows.^{10,13}

International consensus guidelines list therapeutic plasma exchange (TPE) and high-dose intravenous immunoglobulin (IVIg) as first-line immunomodulatory therapies with proven efficacy, and the American Society for Apheresis (ASFA) assigns TPE to Category I.^{7,14}

Standard plasmapheresis regimens call for a course of five to six separate exchanges over 10 to 14 days. Each session exchanges 1.0 to 1.5 plasma volumes against an albumin or plasma replacement colloid.^{5,7} The rationale for such a prolonged schedule comes from classical IgG redistribution pharmacokinetics. Extravascular antibodies re-equilibrate into the circulation over 24 to 48 hours, and plasma IgG concentrations then return to 60% to 80% of pre-exchange baselines. Single-session or shortened exchange regimens are therefore not recommended in current guidelines. The evidence for them is limited. To our knowledge, rapid clinical stabilization after one apheresis session in ventilator-dependent AMAN has rarely been reported.^{5,7}

The authors have previously reported the same patient, a ventilator-dependent man with electrophysiologically confirmed AMAN who was liberated from the ventilator after a single, abbreviated, large-volume TPE session.¹⁵ The present report describes the course again in the structure of the CARE guideline. The recovery may reflect the natural history of AMAN rather than the exchange. One patient cannot separate the two. What a single case can offer is a detailed, day-by-day record of neuromuscular recovery, ventilatory mechanics, and gas exchange around one large-volume exchange. The objective is to describe the clinical course, ventilator weaning, and recovery of one patient with AMAN after a single large-volume TPE session. The report follows the CARE guideline.¹⁶ Mechanistic explanations are offered in the Discussion only as hypotheses.

2. Methods

2.1. Report design and reporting guideline

This is a single-patient case report prepared according to the CARE guideline.¹⁶ Its aim was to describe the physiological course, neuromuscular recovery, and weaning from intensive care after an abbreviated single-session TPE in fulminant AMAN, with closely spaced serial physiological parameters recorded through the acute organ failure and its resolution. These data are reported descriptively.

2.2. Setting

The setting was Dr. Moewardi Regional General Hospital, a tertiary-care academic referral center in Surakarta, Central Java, Indonesia, within its Department of Anesthesiology and Intensive Therapy. Care was delivered in three units. They were the acute neurology ward, the ICU, and the general neurology ward.

2.3. Patient information and case selection

Eligibility was established with the international consensus criteria for GBS defined by the National Institute of Neurological Disorders and Stroke (NINDS) and validated by the Brighton Collaboration GBS Working Group.^{17,18} The case met Brighton Collaboration Level 1 criteria. This is the highest tier of diagnostic certainty. The findings were bilateral, symmetrical flaccid limb weakness, progressive ascending evolution over less than 28 days, generalized hyporeflexia progressing to areflexia, and no alternative diagnosis. Classic cerebrospinal fluid (CSF) albuminocytologic dissociation was also present. So was electrophysiological confirmation of AMAN.^{18,19} Subtype differentiation followed the electrodiagnostic criteria for AMAN formulated by Ho and colleagues and endorsed by the Peripheral Nerve Society.^{11,20} Laboratory screening and clinical examination excluded alternative toxic, metabolic, structural, and infectious etiologies. Demographic and baseline clinical information on the patient is given in the Results.

2.4. Case timeline

The patient was followed through four consecutive clinical environments. These were community onset before admission (Weeks 1 to 3), evaluation on the acute neurology ward (Days 1 to 3), and life support and apheresis in the ICU (Days 4 to 8). Subacute neurorehabilitation on the general neurology ward continued until discharge (Days 9 to 21). Table 1 lists the sequence of events. Days are counted relative to hospital admission (Day 1), and the list runs from symptom onset through intubation, TPE, extubation, ICU discharge, and hospital discharge.

Table 1. Case timeline from symptom onset to hospital discharge, with time expressed in days relative to admission.

Time point	Clinical event	Investigation and result	Intervention
About Day –31 (about 10 days before onset)	Dry, non-productive cough; no diarrheal illness or fever	None recorded	None recorded
About Day –21 (onset)	Onset of weakness in the distal lower extremities with glove-and-stocking paresthesias and moderate, dull lower back pain; ascent to the proximal muscles and upper extremities over about 3 weeks	None recorded	None recorded
Day 1 (admission to neurology ward)	Flaccid quadriplegia; GCS 15/15; cranial nerves intact; MRC sum score 20/60; GBS disability score 4	CSF protein 142 mg/dL, 2 cells/ μ L, glucose 68 mg/dL; nerve conduction: no motor responses, preserved SNAPs; blood culture: <i>Micrococcus luteus</i> in 1 bottle (48 h); chest radiograph clear	Admission to inpatient neurology ward; no immunotherapy before Day 5
Days 2 to 3	Progressive decline of motor function on the ward	None recorded	None recorded
Day 4 (nadir; ICU admission)	Respiratory distress, tachypnea 28 to 32 breaths/min, paradoxical abdominal breathing; GCS 14/15; MRC sum score 12/60; areflexia; GBS disability score 5	Arterial blood gas: pH 7.31, PaCO ₂ 49.0 mmHg, PaO ₂ 88.0 mmHg, PaO ₂ /FiO ₂ 146.7 mmHg; NIF –12 cmH ₂ O; vital capacity 8.5 mL/kg; RSBI 68.4 breaths/min/L	Rapid sequence intubation (7.5 mm cuffed tube); transfer to ICU; SIMV with pressure support, PEEP 10 cmH ₂ O, FiO ₂ 0.60
Day 5 (TPE)	Pre-TPE: MRC sum score 12/60; GBS disability score 5; GCS 14/15	Hour 12 after TPE: pH 7.38, PaO ₂ /FiO ₂ 226.7 mmHg	Single TPE session (140 min, 2,500 mL exchanged; 2,000 mL 5% albumin and 500 mL 0.9% saline; ACD-A anticoagulation, calcium gluconate infusion)
Day 6 (24 h after TPE)	Alert; MRC sum score 20/60; GBS disability score 5	pH 7.42, PaCO ₂ 38.0 mmHg, PaO ₂ 115.0 mmHg, PaO ₂ /FiO ₂ 328.6 mmHg; compliance 57.5 mL/cmH ₂ O	SIMV changed to CPAP with pressure support; FiO ₂ 0.35, PEEP 6 cmH ₂ O, pressure support 8 cmH ₂ O
Day 7 (48 h after TPE)	MRC sum score 28/60; patellar reflexes +1; GBS disability score 4	Spontaneous breathing trial: RSBI 31.3 breaths/min/L, NIF –32 cmH ₂ O, vital capacity 18.5 mL/kg, PaO ₂ /FiO ₂ 360.0 mmHg	Extubation at 14:00; nasal cannula 2 L/min
Day 8 (72 h after TPE)	Clinically stable on room air; MRC sum score 30/60	None recorded	Catheter removed; discharge from ICU to neurology ward; neurorehabilitation continued
Day 14	MRC sum score 38/60; GBS disability score 3; stood with assistance and walked 10 m with two therapists	None recorded	Neurorehabilitation
Day 21 (hospital discharge)	MRC sum score 44/60; GBS disability score 3; walked more than 20 m with a rolling walker	None recorded	Discharge home; outpatient physical therapy.

Notes: Day 1 is the day of admission to the neurology ward; negative days precede admission. Time points before admission are approximate and taken from the history. ACD-A, acid citrate dextrose formula A; CPAP, continuous positive airway pressure; CSF, cerebrospinal fluid; FiO₂, fraction of inspired oxygen; GBS, Guillain-Barré syndrome; GCS, Glasgow Coma Scale; ICU, intensive care unit; MRC, Medical Research Council; NIF, negative inspiratory force; PaCO₂, arterial carbon dioxide tension; PaO₂, arterial oxygen tension; PEEP, positive end-expiratory pressure; RSBI, rapid shallow breathing index; SIMV, synchronized intermittent mandatory ventilation; SNAP, sensory nerve action potential; TPE, therapeutic plasma exchange. MRC sum scores and GCS values are as printed in Table 3.

2.5. Clinical findings and diagnostic assessment

CSF was collected by atraumatic lumbar puncture at the L3–L4 intervertebral space under sterile conditions. Analysis followed within two hours. Tests covered total protein concentration, total and differential leukocyte counts, glucose concentration, Gram staining, and bacterial culture. Nerve conduction studies (NCS) used a calibrated computerized electromyography system for bilateral motor and sensory recordings on major peripheral nerve trunks, and the motor studies covered the median, ulnar, common peroneal, and tibial nerves. The motor studies measured distal motor latencies, compound muscle action potential (CMAP) peak-to-peak amplitudes, conduction velocities, and late F-wave responses. Sensory studies covered the median, ulnar, and sural nerves. They measured sensory nerve action potential (SNAP) amplitudes and peak latencies. AMAN was confirmed when motor CMAPs were reduced or inexcitable across multiple nerve distributions. SNAPs had to be normal. Primary demyelinating features had to be absent. Examples were prolonged distal motor latencies >130% of normal, conduction velocity slowing <70% of normal, or temporal dispersion.^{11,20}

2.6. Therapeutic intervention

On Day 4 the patient developed acute neuromuscular respiratory failure. He was intubated emergently. A 7.5 mm internal diameter cuffed endotracheal tube was placed and

secured at 22 cm at the incisors, and an advanced intensive care ventilator delivered synchronized intermittent-mandatory ventilation (SIMV) with pressure support (PS). Initial settings were a positive end-expiratory pressure (PEEP) of 10 cmH₂O and a fraction of inspired oxygen (FiO₂) of 0.60 (60%). Inspiratory pressure above PEEP was 10 cmH₂O. The PS was 12 cmH₂O. Monitoring covered exhaled tidal volume, peak inspiratory pressure, plateau airway pressure, total respiratory frequency, and dynamic thoracic compliance. Compliance was calculated as exhaled tidal volume divided by the difference between peak inspiratory pressure and PEEP.

An indwelling 20-gauge radial arterial catheter supplied samples for arterial blood gas analysis. Samples were taken right after intubation, 12 hours after apheresis, during daily spontaneous breathing evaluations, and two hours after extubation. We recorded arterial oxygen tension (PaO₂), carbon dioxide tension, arterial pH, bicarbonate concentration, and base excess. We also recorded the ratio of PaO₂ to FiO₂ (PaO₂/FiO₂). Weaning readiness was judged daily against objective extubation criteria. These were the rapid shallow breathing index (RSBI; spontaneous breathing frequency divided by tidal volume in liters), negative inspiratory force (NIF), and vital capacity.²¹ The thresholds used were an RSBI below 105 breaths/min/L, an NIF more negative than –25 cmH₂O, and a vital capacity above 15 mL/kg. All three were expected to be met before extubation.

TPE took place on Day 5 on an automated membrane-based apheresis platform. Access was a temporary 12-French double-lumen hemodialysis catheter placed in the right internal jugular vein under sterile ultrasound guidance. Estimated plasma volume was calculated with the standard Kaplan formula. It uses body weight and baseline hematocrit: $0.07 \times \text{body weight (kg)} \times (1 - \text{hematocrit})$. Acid citrate dextrose formula A (ACD-A) provided anticoagulation at a whole blood-to-citrate ratio of 1:12 to 1:14, and calcium gluconate (10%) was infused continuously as prophylaxis against systemic hypocalcemia. The team exchanged 2,500 mL of plasma. This equals 1.08 plasma volumes. Replacement used 2,000 mL of 5% human albumin plus 500 mL of 0.9% normal saline, which maintained intravascular oncotic pressure and prevented hypovolemia. Monitoring was continuous. Throughout the procedure we recorded invasive arterial pressure, electrocardiographic rhythm, pulse oximetry, and hourly urine output.

2.7. Outcome measures and follow-up

Serial parameters were recorded from admission through the hospital course in four domains: neurological motor status, pulmonary-ventilatory mechanics, blood gas values, and apheresis parameters. Motor strength was scored with the Medical Research Council (MRC) sum score. It covers six paired muscle groups tested bilaterally: shoulder abductors (deltoid), elbow flexors (biceps), wrist extensors, hip flexors (iliopsoas), knee extensors (quadriceps), and ankle dorsiflexors (tibialis anterior).²² Each group received a score from 0 (no contraction) to 5 (normal power against full resistance), so the sum score ranged from 0 to 60. Functional disability was assessed alongside it with the Hughes GBS disability score. Grade 0: healthy. Grade 1: minor symptoms, fully capable of running. Grade 2: able to walk 10 meters independently without support. Grade 3: able to walk 10 meters with a walker or assistance. Grade 4: bed- or chair-bound, unable to walk. Grade 5: requiring mechanical ventilation for at least part of the day or night. Grade 6: death.²³ The Glasgow Coma Scale (GCS) and detailed cranial nerve examinations tracked consciousness and brainstem integrity. Ventilator mechanics were followed with PEEP, PS, FiO₂, peak inspiratory pressure, exhaled tidal volume, dynamic compliance, RSBI, NIF, vital capacity, and the blood gas values described above. The patient was followed until hospital discharge on Day 21.

This is single-subject research (n = 1). Inferential testing, such as p-values, Student’s t-tests, analysis of variance, or chi-square tests, was therefore avoided, because

computing population-level hypothesis probabilities from one subject is invalid and misleading. The analysis was descriptive: serial values are tabulated by day. Percentage change across sequential clinical milestones was calculated with one formula: $(\text{value at time } n - \text{value at the baseline nadir}) \div \text{value at the baseline nadir} \times 100\%$. This percentage is a descriptive summary only, and absolute values are given with it. Clinical resolution rates were the absolute change in functional scores divided by elapsed observation time in days.

2.8. Ethics and consent

The CMHC Research Center Ethics Committee, Indonesia, reviewed and approved the study protocol, data collection methods, and biological sampling procedures (Protocol Approval Number: 2025/0418). Institutional governance at Dr. Moewardi Regional General Hospital and the Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia, took part in the review. Every clinical intervention, diagnostic procedure, and analytical evaluation followed the World Medical Association Declaration of Helsinki of 1975, as revised in Fortaleza, Brazil in 2013.²⁴ It also followed the international ethical guidelines for health-related research involving humans issued by the Council for International Organizations of Medical Sciences (CIOMS).²⁵ The patient and his legal surrogate gave written informed consent directly. They did so before data extraction and manuscript preparation began. Consent covered publication of all anonymized clinical details, physiological data, laboratory metrics, electrophysiological recordings, and associated clinical infographics. Direct identifiers were removed. Every diagnostic image was fully anonymized. The full statement is given in the Declarations.

3. Results

3.1. Presentation and baseline findings

The patient was a 68-year-old man who presented with subacute weakness of all four limbs that had worsened steadily over the 21 days before admission (Table 1; Figure 1; Table 2). Before the neurological symptoms began, he was active and fully independent, with a modified Rankin Scale (mRS) score of 0 and a GBS disability score of 0. He had no chronic metabolic, autoimmune, cardiorespiratory, or renal disease. Diabetes mellitus, chronic kidney disease, and preexisting peripheral polyneuropathy were absent from his history. He did not smoke or drink alcohol. He had no industrial neurotoxic exposure.

Case profile: 68-year-old man with Guillain-Barré syndrome (AMAN variant)		
Initial assessment data on admission to the neurology service.		
Age 68 years	Sex Male	Pre-illness functional status Fully independent and active
Presenting complaint Progressive weakness of all four limbs for 3 weeks	Associated symptoms Paresthesias in hands and feet; lower back pain	Past medical history Unremarkable; no known chronic illnesses
Antecedent events Dry cough about 10 days before onset; no recent diarrheal illness	Social history Non-smoker, non-drinker	

AMAN, acute motor axonal neuropathy; GBS, Guillain-Barré syndrome.

Figure 1. Patient demographics and baseline characteristics at presentation.

Table 2. Baseline demographic, clinical, and laboratory findings compared with reference intervals.

Biological parameter / clinical metric	Observed baseline value	Reference interval	Comment
Chronological Age	68 years	N/A	Age group with higher reported GBS incidence; vulnerability to axonal damage may be greater
Sex	Male	N/A	Male sex is slightly more frequent in GBS epidemiology
Pre-Illness Functional Status	mRS 0; GBS-DS 0	Fully independent (0)	Consistent with a previously independent patient
Symptom Duration to Admission	21 days (3 weeks)	Subacute onset	Course consistent with ascending flaccid polyradiculoneuropathy
Antecedent Infection Symptoms	Dry non-productive cough	Negative	Possible upper respiratory prodrome; no history of enteritis (<i>C. jejuni</i>)
Comorbid Health Profile	Unremarkable	Healthy baseline	Argues against diabetic, uremic, or toxic-metabolic polyneuropathy
Estimated Body Weight / Height	65.0 kg / 168 cm	BMI 23.0 kg/m ²	Normal body mass index; estimated plasma volume (EPV) of 2,310–2,450 mL
CSF Total Protein Concentration	142 mg/dL	15–45 mg/dL	Albuminocytologic dissociation; compatible with blood-nerve barrier disruption
CSF Total Leukocyte Count	2 cells/μL	0–5 cells/μL	Normal cell count; argues against bacterial or viral meningitis
CSF Glucose / Serum Glucose Ratio	68 mg/dL / 104 mg/dL (0.65)	0.60–0.70	Normal glucose ratio; no finding suggesting central infection
Peripheral White Blood Cell Count	8.4×10^3 /μL	$4.5\text{--}11.0 \times 10^3$ /μL	No leukocytosis
Hemoglobin / Hematocrit	13.8 g/dL / 41.5%	13.0–17.5 g/dL / 40–52%	Values within the reference interval
Platelet Count	245×10^3 /μL	$150\text{--}450 \times 10^3$ /μL	Normal count before vascular catheterization and TPE
Serum Sodium / Potassium	138 mEq/L / 4.1 mEq/L	135–145 / 3.5–5.0 mEq/L	Normal sodium and potassium
Serum Urea / Creatinine	16 mg/dL / 0.92 mg/dL	7–20 / 0.70–1.20 mg/dL	Normal renal function
Serum Albumin Concentration	3.8 g/dL	3.5–5.2 g/dL	Normal serum albumin before colloid replacement
Blood Microbial Culture	<i>Micrococcus luteus</i> (1 bottle)	Negative / Sterile	Interpreted as a skin contaminant from venipuncture; no clinical sepsis
Plain Chest Radiography	Clear lung fields; no infiltrate	Normal aerated lungs	No pneumonia or ARDS pattern; pulmonary cause of ventilatory failure not identified

Notes: ARDS, acute respiratory distress syndrome; BMI, body mass index; *C. jejuni*, *Campylobacter jejuni*; CSF, cerebrospinal fluid; EPV, estimated plasma volume; GBS, Guillain-Barré syndrome; GBS-DS, GBS disability score; mRS, modified Rankin Scale; TPE, therapeutic plasma exchange. Reference intervals are the standard institutional laboratory values at Dr. Moewardi Regional General Hospital.

Weakness was ascending and symmetrical. It started in the distal lower extremities and spread to the proximal muscles and the upper extremities over three weeks (Figure 2). Bilateral distal paresthesias of the hands and feet followed a glove-and-stocking pattern, and he also had moderate, dull lower back pain that was considered compatible with acute inflammatory radiculitis. On review for an antecedent infection, he recalled a dry, non-productive cough about ten days before the weakness

began, which was considered compatible with a mild viral upper respiratory tract infection. He reported no diarrheal illness, fever, or gastrointestinal distress. The history did not suggest *Campylobacter jejuni* enteritis. Body weight was 65.0 kg. Height was 168 cm, and body mass index was 23.0 kg/m². With a baseline hematocrit of 41.5%, total circulating plasma volume was estimated at 2,310 to 2,450 mL (Table 2; Figure 1).



ICU, intensive care unit; TPE, therapeutic plasma exchange. Day 1 is the day of admission to the neurology ward.

Figure 2. Chronological timeline of key clinical milestones from symptom onset to ICU discharge.

On admission to the inpatient neurology ward (Day 1), cognition and orientation were intact. The GCS score was 15/15 (E4V5M6) (Figure 3). Cranial nerves II through XII

were intact: extraocular movements were normal, the face was symmetrical, the palate elevated, and the tongue protruded normally. There was no cranial or bulbar

involvement at presentation. Motor examination showed flaccid quadriparesis. On the MRC scale, strength was 3/5 bilaterally in the upper extremities (deltoid, biceps, and wrist extensors) and 2/5 bilaterally in the lower extremities (iliopsoas, quadriceps, and tibialis anterior). The admission MRC sum score was recorded as 20/60 (Table 3). Deep tendon reflexes were diminished (+1) at

the biceps and brachioradialis. They were absent (0) at both patellar and Achilles tendons. Sensory testing found subjective glove-and-stocking paresthesias, but pinprick, light touch, and vibration sense were normal on examination. The patient could not walk independently or stand without help. This gave a GBS disability score of 4 (bed- or chair-bound) (Table 3; Figure 3).

On admission (Day 1) Initial assessment on the neurology ward Consciousness Alert and oriented (GCS 15/15) Cranial nerves Unremarkable Motor strength (MRC scale) Upper extremities: 3/5 (bilateral). Lower extremities: 2/5 (bilateral). Deep tendon reflexes Upper extremities: +1 (diminished). Lower extremities: 0 (absent). GBS disability score 4 (bed- or chair-bound)	At ICU admission (Day 4) Status on critical deterioration Consciousness Restless, anxious (GCS 14/15) Cranial nerves Unremarkable Motor strength (MRC scale) Upper extremities: 2/5 (bilateral). Lower extremities: 1/5 (bilateral). Deep tendon reflexes All limbs: 0 (areflexia) GBS disability score 5 (requires mechanical ventilation)
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GBS, Guillain-Barré syndrome; GCS, Glasgow Coma Scale; ICU, intensive care unit; MRC, Medical Research Council.

Figure 3. Neurological examination findings on ward admission (Day 1) and at the ICU admission nadir (Day 4).

Table 3. Neurological trajectory: motor strength, reflexes, and disability scores from admission to discharge.

Time point / event	GCS score	Upper extremity MRC (bilateral)	Lower extremity MRC (bilateral)	MRC sum score (0–60)	Deep tendon reflexes	GBS disability score	Change in MRC sum score (Δ%)
Day 1: Ward Admission	15/15	3/5 (Deltoid, Biceps, Wrist)	2/5 (Iliopsoas, Quads, Tib. Ant.)	20/60	Upper: +1; Lower: 0	4 (Bed/chair-bound)	Baseline reference
Day 4: Clinical Nadir & ICU Transfer	14/15	2/5 (Gravity-eliminated movement)	1/5 (Trace muscular flicker)	12/60	All limbs: 0 (Areflexia)	5 (Ventilator-dependent)	Δ –40.0% vs. Day 1
Day 5: Pre-TPE Assessment	14/15	2/5 (Gravity-eliminated movement)	1/5 (Trace muscular flicker)	12/60	All limbs: 0 (Areflexia)	5 (Ventilator-dependent)	Clinical nadir plateau
Day 6: 24 h Post-TPE (PS Weaning)	15/15	3/5 (Anti-gravity movement)	2/5 (Gravity-eliminated movement)	20/60	Upper: +1; Lower: 0	5 (Ventilator-assisted)	Δ +66.7% vs. Nadir
Day 7: 48 h Post-TPE (Extubation)	15/15	4/5 (Moderate resistance)	3/5 (Anti-gravity movement)	28/60	Upper: +1; Lower: +1	4 (Bed- or chair-bound; extubated)	Δ +133.3% vs. Nadir
Day 8: 72 h Post-TPE (ICU Discharge)	15/15	4/5 (Moderate resistance)	3/5 (Anti-gravity movement)	30/60	Upper: +2; Lower: +1	4 (Transferred to ward)	Δ +150.0% vs. Nadir
Day 14: Subacute Follow-Up (Ward)	15/15	4+/5 (Overcoming resistance)	4/5 (Overcoming resistance)	38/60	Upper: +2; Lower: +2	3 (Assisted ambulation)	Δ +216.7% vs. Nadir
Day 21: Hospital Discharge	15/15	5/5 (Full normal strength)	4+/5 (Overcoming resistance)	44/60	Upper: +2; Lower: +2	3 (Walks with walker)	Δ +266.7% vs. Nadir

Notes: GBS, Guillain-Barré syndrome; GCS, Glasgow Coma Scale; ICU, intensive care unit; MRC, Medical Research Council muscle scale (graded 0 to 5; sum score maximum 60); PS, pressure support; TPE, therapeutic plasma exchange. Δ% = $[(T_n - T_0) / T_0] \times 100\%$, where T_0 is the nadir score on Day 4 (12/60); the Day 4 row compares the nadir with Day 1 (20/60).

3.2. Diagnostic findings

Investigations began at once on admission, with the aim of confirming the diagnosis and excluding other central or peripheral causes (Figure 4). Lumbar puncture, done under sterile conditions, returned clear cerebrospinal fluid (CSF). Total protein was 142 mg/dL (reference interval 15 to 45 mg/dL), which is 3.2 times the upper limit of normal.

The total leukocyte count was 2 cells/ μ L (reference interval 0 to 5 cells/ μ L), and glucose was 68 mg/dL (simultaneous serum glucose 104 mg/dL; ratio 0.65). This pattern is albuminocytologic dissociation. It combines raised protein with a normal cell count and no central nervous system pleocytosis. It is compatible with blood-nerve barrier disruption and inflammatory polyradiculopathy (Table 2; Figure 4).

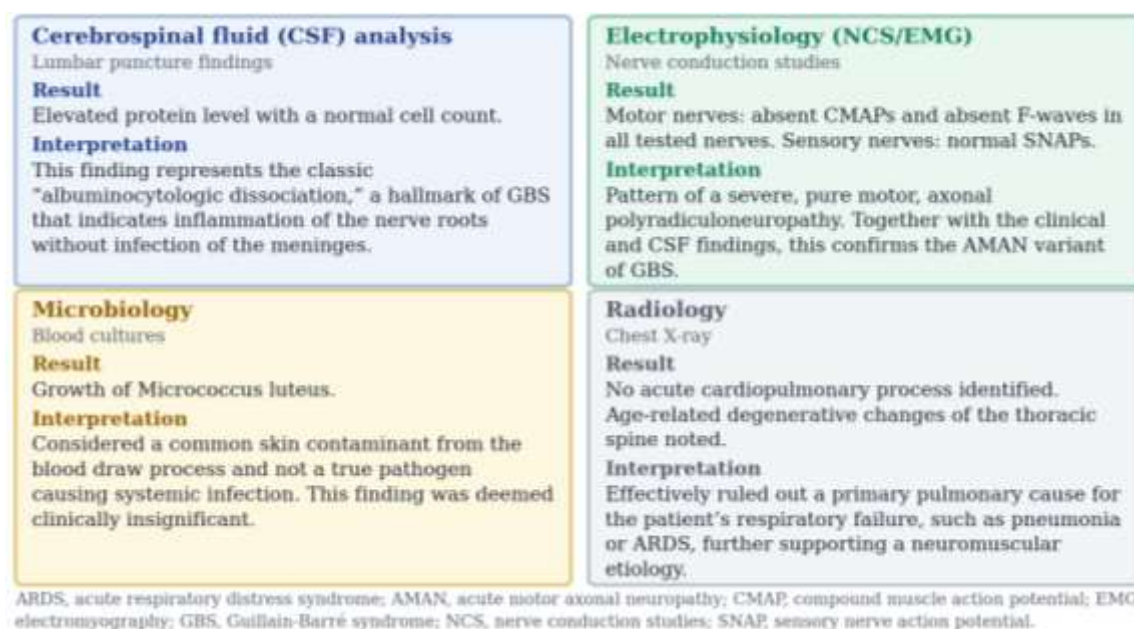


Figure 4. Diagnostic investigations: CSF analysis, nerve conduction studies, blood culture, and chest radiograph.

Bilateral nerve conduction studies were performed (Figure 4). Motor conduction studies found no excitable response in any tested nerve, including the bilateral median, ulnar, common peroneal, and posterior tibial nerves. Compound muscle action potentials (CMAPs) could not be obtained (0 mV peak-to-peak amplitude), and late F-wave responses were absent in every motor distribution. Sensory nerve action potentials (SNAPs) were present bilaterally, with normal peak latencies and amplitudes in the median (18.2 μ V), ulnar (14.6 μ V), and sural (12.4 μ V) nerves. Absent motor responses with preserved sensory conduction were in keeping with the AMAN variant of GBS. No feature of primary demyelination was found (Table 2; Figure 4). Anti-ganglioside antibody titers were not measured. No repeat nerve conduction study was done.

Microbiological and radiological studies were done to look for systemic infection and lung disease (Figure 4). Automated blood cultures drawn on admission grew *Micrococcus luteus* in one aerobic bottle after 48 hours of incubation. The patient remained afebrile. His peripheral white blood cell count was $8.4 \times 10^3/\mu\text{L}$ (reference 4.5 to $11.0 \times 10^3/\mu\text{L}$), serum procalcitonin was 0.08 ng/mL, and hemodynamics were stable. The isolate was judged a skin commensal contaminant from venipuncture. No antibacterial treatment was given, and sepsis was not diagnosed. Plain chest radiography showed clear lung fields bilaterally. There were no alveolar or interstitial infiltrates. The costophrenic angles were sharp, and the cardiothoracic ratio was 0.46. Minor age-related degenerative thoracic spondylosis was present. The radiograph showed no pneumonia, pneumothorax, pulmonary edema, or pattern of acute respiratory distress syndrome, and no pulmonary or septic cause of ventilatory failure was identified at this stage (Table 2; Figure 4).

3.3. Treatment course and outcome

Motor function declined steadily over the first 72 hours on the neurology ward (Figure 3). Strength reached its lowest point on the morning of Day 4. Severe dyspnea followed. His tachypnea ran at a spontaneous rate of 28 to 32 breaths/min, with paradoxical abdominal breathing and

intercostal retractions. He showed air hunger, sweating, and restless anxiety. His GCS score fell slightly to 14/15 (E4V4M6), which was attributed to acute hypercapnic-hypoxic distress. Extraocular and facial muscle power remained preserved. Repeat motor testing showed a worse flaccid quadriparesis. Upper extremity strength had fallen to 2/5 bilaterally. Lower extremity power was down to a trace flicker of movement, graded 1/5 bilaterally. The MRC sum score fell from 20/60 on Day 1 to a nadir of 12/60 on Day 4, a change of -40.0% (Table 3). Reflexes had progressed to global areflexia (0 in all four limbs). The GBS disability score rose from grade 4 to grade 5. This grade is defined by the need for mechanical ventilatory support (Table 3; Figure 3).

The airway was secured at once (Figure 5). After preoxygenation and rapid sequence induction, the trachea was intubated without trauma. A 7.5 mm internal diameter cuffed endotracheal tube was used, fixed at 22 cm at the teeth. The patient was moved urgently to the ICU. There he was placed on synchronized intermittent mandatory ventilation (SIMV) with pressure support (Table 4). Baseline settings were a PEEP of 10 cmH₂O, a FiO₂ of 0.60, an inspiratory pressure above PEEP of 10 cmH₂O, and a pressure support of 12 cmH₂O. The first arterial blood gas showed acute respiratory acidosis with hypoxemia: pH 7.31, arterial carbon dioxide tension (PaCO₂) 49.0 mmHg, arterial oxygen tension (PaO₂) 88.0 mmHg, serum bicarbonate 24.2 mEq/L, base excess -2.1 mEq/L, and a PaO₂/FiO₂ ratio of 146.7 mmHg. Gas exchange was impaired. These values were in keeping with hypoventilation and microatelectasis. Peak inspiratory airway pressure (PIP) was 22 cmH₂O. The exhaled tidal volume was 380 mL (5.9 mL/kg predicted body weight [PBW]; PBW 64.2 kg for a man 168 cm tall), and baseline dynamic thoracic compliance was 31.7 mL/cmH₂O. The rapid shallow breathing index (RSBI) was 68.4 breaths/min/L, below the cut-off of 105 breaths/min/L. NIF was -12 cmH₂O. Vital capacity was 8.5 mL/kg (Table 4; Figure 5). The respiratory rate of 26 breaths/min listed for Day 4 in Table 4 was recorded after intubation. The 28 to 32 breaths/min above preceded it.

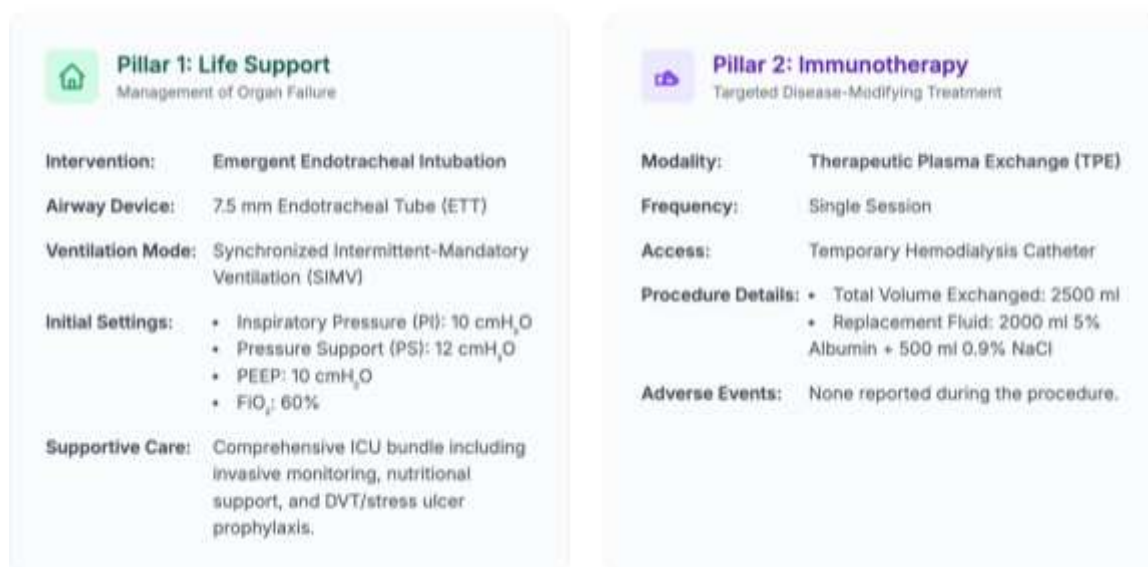


Figure 5. ICU life support and plasma exchange interventions.

Table 4. Serial ventilator settings, arterial blood gases, and respiratory mechanics from intubation to extubation.

Parameter	Day 4: emergent intubation	Day 5: post-TPE (hour 12)	Day 6: pressure support weaning	Day 7: pre-extubation SBT	Day 8: post-extubation (room air)	Delta ($\Delta\%$), Day 4 to Day 7 (Cdyn and PIP rows: Day 4 to Day 6)
Ventilator Mode	SIMV + PS	SIMV + PS	CPAP + PS	T-piece Spontaneous	Unassisted/NC 2 L/min	Extubated on Day 7
Fraction of Inspired Oxygen (FiO_2)	0.60 (60%)	0.45 (45%)	0.35 (35%)	0.30 (30%)	0.21 (Room air)	Δ -50.0%
Positive End-Expiratory Pressure (PEEP)	10 cmH ₂ O	8 cmH ₂ O	6 cmH ₂ O	0 cmH ₂ O	0 cmH ₂ O	Δ -100.0%
Inspiratory Pressure Support (PS)	12 cmH ₂ O	10 cmH ₂ O	8 cmH ₂ O	0 cmH ₂ O	0 cmH ₂ O	Δ -100.0%
Peak Inspiratory Pressure (PIP)	22 cmH ₂ O	19 cmH ₂ O	14 cmH ₂ O	N/A	N/A	Δ -36.4%
Expiratory Tidal Volume (Vte)	380 mL	420 mL	460 mL	480 mL	490 mL	Δ +26.3%
Dynamic Lung Compliance (Cdyn)	31.7 mL/cmH ₂ O	38.2 mL/cmH ₂ O	57.5 mL/cmH ₂ O	N/A	N/A	Δ +81.4%
Spontaneous Respiratory Rate (f)	26 breaths/min	20 breaths/min	16 breaths/min	15 breaths/min	16 breaths/min	Δ -42.3%
Rapid Shallow Breathing Index (RSBI)	68.4 breaths/min/L	47.6 breaths/min/L	34.8 breaths/min/L	31.3 breaths/min/L	N/A	Δ -54.2%
Negative Inspiratory Force (NIF)	-12 cmH ₂ O	-18 cmH ₂ O	-26 cmH ₂ O	-32 cmH ₂ O	N/A	Δ +166.7%
Vital Capacity (VC)	8.5 mL/kg	11.2 mL/kg	14.8 mL/kg	18.5 mL/kg	19.8 mL/kg	Δ +117.6%
Arterial pH (pH units)	7.31	7.38	7.42	7.41	7.42	+0.10 pH units
Arterial Carbon Dioxide Tension (PaCO_2)	49.0 mmHg	41.0 mmHg	38.0 mmHg	39.0 mmHg	38.5 mmHg	Δ -20.4%
Arterial Oxygen Tension (PaO_2)	88.0 mmHg	102.0 mmHg	115.0 mmHg	108.0 mmHg	96.0 mmHg	Δ +22.7%
$\text{PaO}_2/\text{FiO}_2$ ratio	146.7 mmHg	226.7 mmHg	328.6 mmHg	360.0 mmHg	457.1 mmHg	Δ +145.4%

Notes: Cdyn, dynamic thoracic compliance [Vte / (PIP - PEEP)]; CPAP, continuous positive airway pressure; FiO_2 , fraction of inspired oxygen; NC, nasal cannula; NIF, negative inspiratory force; PaCO_2 , arterial carbon dioxide tension; PaO_2 , arterial oxygen tension; PEEP, positive end-expiratory pressure; PBW, predicted body weight; PIP, peak inspiratory pressure; PS, pressure support; RSBI, rapid shallow breathing index (respiratory frequency / tidal volume in liters; cut-off for weaning failure 105 breaths/min/L, all listed values are below it); SBT, spontaneous breathing trial; SIMV, synchronized intermittent mandatory ventilation; TPE, therapeutic plasma exchange; Vte, exhaled tidal volume. The Cdyn and PIP changes (+81.4% and -36.4%) compare Day 4 with Day 6 because these values were not available on Day 7 (T-piece). The Δ for NIF refers to an increase in the magnitude of inspiratory force. The Day 4 tidal volume of 380 mL is 5.9 mL/kg PBW (PBW 64.2 kg for a man 168 cm tall; 5.8 mL/kg actual weight of 65.0 kg). The respiratory rate of 26 breaths/min on Day 4 was recorded after intubation; the 28 to 32 breaths/min in the text preceded it.

Immunotherapy began on Day 5, once hemodynamics had stabilized and the baseline diagnostic work-up was complete (Figure 5). Resources were limited and the case was urgent. One large-volume session of TPE was the only immunotherapeutic intervention. Vascular access was a temporary 12-French dual-lumen hemodialysis catheter. It was placed in the right internal jugular vein under real-time ultrasound guidance.

Automated membrane plasma separation ran for 140 minutes (Table 5). Blood flow averaged 90 mL/min. A total

of 2,500 mL of plasma was removed. This equaled about 1.08 calculated plasma volumes against an estimated plasma volume of 2,310 mL. Replacement fluid combined colloid and crystalloid. It comprised 2,000 mL of 5% human albumin (80% of the exchange volume) and 500 mL of 0.9% normal saline (20%). Anticoagulation used acid citrate dextrose formula A (ACD-A). A continuous intravenous infusion of 10% calcium gluconate ran alongside it.

Table 5. Plasma exchange procedure and modeled clearance.

Procedural / pharmacokinetic metric	Observed or modeled value / setting	Reference, target, or detail	Comment
Exchange modality	Membrane plasma separation	Automated TPE platform	Automated procedure of 140 min; blood flow averaged 90 mL/min
Procedural frequency	Single session	Reference regimen: 5 sessions over 10–14 days	-
Vascular access route	Right internal jugular vein	12-French dual-lumen catheter	Placed under real-time ultrasound guidance; blood flow 80–100 mL/min
Anticoagulation protocol	Regional citrate (ACD-A)	ACD-A infusion (1:12 to 1:14 ratio)	Continuous 10% calcium gluconate infusion given alongside
Patient estimated plasma volume (EPV)	2,310 mL	Weight 65 kg; hematocrit 41.5%	Kaplan formula as printed: $EPV = 0.07 \times \text{weight (kg)} \times (1 - \text{hematocrit})$
Total volume exchanged	2,500 mL	1.08 plasma volumes (calculated)	Volume exchanged relative to the EPV of 2,310 mL
Replacement colloid	2,000 mL 5% human albumin	80% of exchange volume	Replacement fluid consisted of albumin and saline
Replacement crystalloid	500 mL 0.9% normal saline	20% of exchange volume	Replacement fluid consisted of albumin and saline
Intravascular IgG removal	66.0%	Expected 60–70% per 1.0 EPV	-
Circulating anti-GM1/GD1a	About 65–70%	Reduction below a binding threshold	-
Complement components C1q, C3, C5	More than 75%	Temporary complement depletion	-
Systemic inflammatory cytokines	Acute clearance, no value given	TNF- α , IL-1, IL-6	-
Procedural hemodynamics	Mean arterial pressure 74–86 mmHg	No vasopressors	-
Post-procedural ionized calcium	1.18 mmol/L (normal 1.15–1.33)	Citrate-related hypocalcemia avoided	No tetany or QTc prolongation reported; calcium gluconate infused during the procedure
Post-procedural serum albumin	4.2 g/dL (pre-procedure 3.8 g/dL)	Reference: above 3.5 g/dL	Time of the post-procedure value not stated

Notes: ACD-A, acid citrate dextrose formula A; EPV, estimated plasma volume; GM1 and GD1a, gangliosides; IgG, immunoglobulin G; IL, interleukin; QTc, corrected QT interval; TNF- α , tumor necrosis factor- α ; TPE, therapeutic plasma exchange. Calcium gluconate was given as 10% solution.

A first-order apheresis clearance model was applied to the single pass of 1.08 plasma volumes. It gave a theoretical intravascular immunoglobulin G (IgG) removal of 66.0% ($C_{\text{post}} = C_{\text{pre}} \times e^{(-1.08)}$; Table 5). As a theoretical estimate only, a non-selective colloid exchange of this volume would also be expected to lower several targets by more than 75%. These are the circulating complement components (C1q, C3, and C5) and the major pro-inflammatory cytokines (tumor necrosis factor- α , interleukin-1, and interleukin-6). Anti-ganglioside antibody, complement, and cytokine levels were not

measured. Mean arterial pressure stayed between 74 and 86 mmHg with no vasopressor infusion. Heart rate ranged from 68 to 78 beats/min. Post-procedural ionized calcium was 1.18 mmol/L. Serum albumin rose from 3.8 g/dL to 4.2 g/dL. No apheresis-related adverse events occurred. There were no allergic reactions, hypocalcemic tetany, catheter-related thrombosis, or secondary infections (Table 5; Figure 5). After the single TPE session on Day 5, motor and respiratory measures improved over the next 72 hours (Figure 2; Figure 3; Figure 6; Table 3; Table 4).

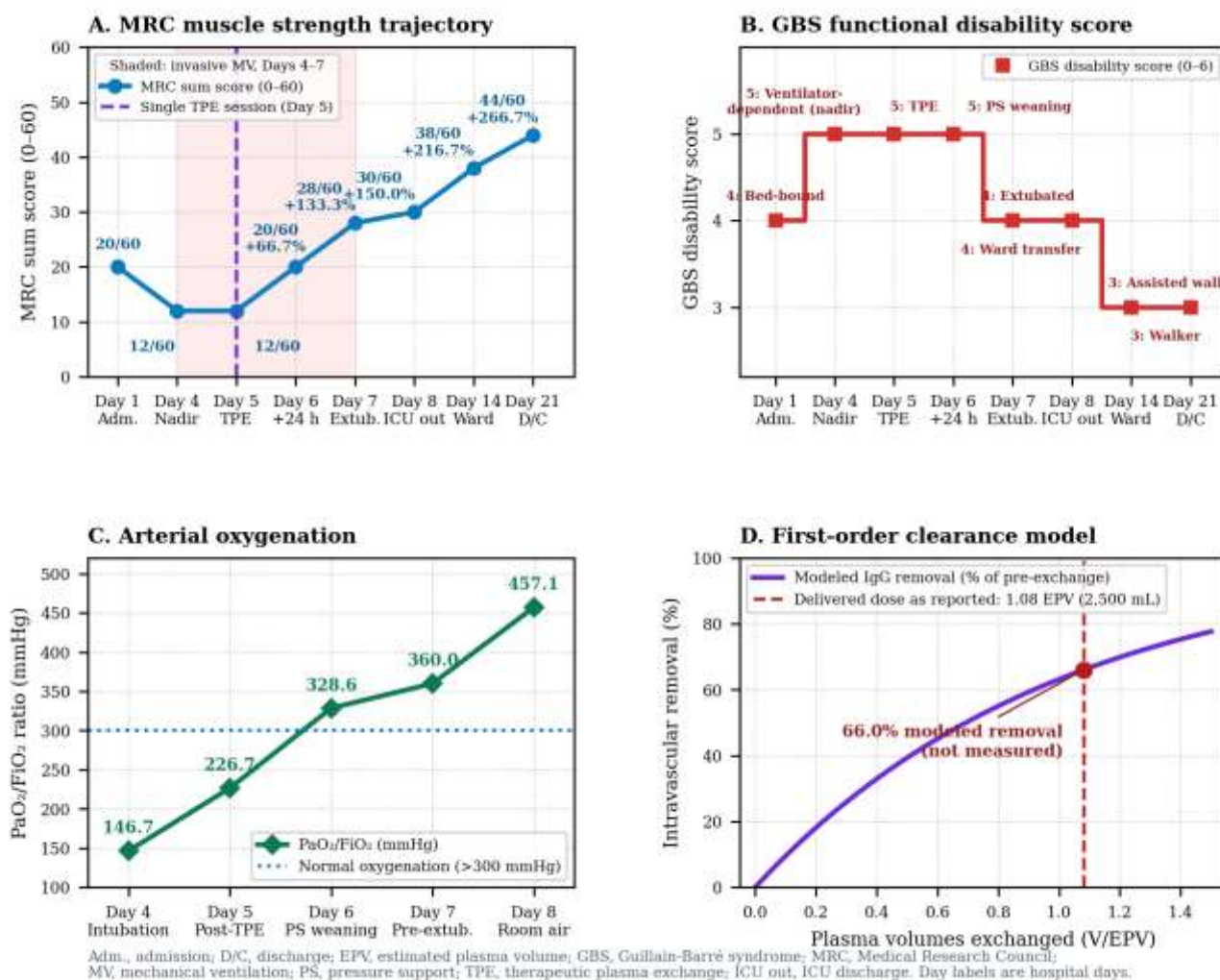


Figure 6. Trajectories after plasma exchange: (A) MRC sum score, (B) GBS disability score, (C) PaO₂/FiO₂ ratio, and (D) modeled first-order IgG removal (modeled, not measured)

By 24 hours after TPE (Day 6), spontaneous respiratory effort was present. The patient was fully alert. He opened his eyes spontaneously and communicated by nods and hand gestures (GCS score 15/15 on the ventilator). Support was changed from SIMV to continuous positive airway pressure (CPAP) with pressure support (Table 4). FiO₂ was weaned from 0.60 to 0.35, PEEP was lowered to 6 cmH₂O, and pressure support to 8 cmH₂O, while PIP fell from 22 to 14 cmH₂O and exhaled tidal volume rose to 460 mL. Dynamic thoracic compliance rose by 81.4%, from 31.7 mL/cmH₂O at baseline to 57.5 mL/cmH₂O. Arterial blood gas values on Day 6 were pH 7.42, PaCO₂ 38.0 mmHg, and PaO₂ 115.0 mmHg. The PaO₂/FiO₂ ratio was 328.6 mmHg (+124.0% relative to Day 4) (Table 4). Upper extremity strength was 3/5 bilaterally. Lower extremity power was 2/5 bilaterally. The MRC sum score was 20/60 (Table 3).

On Day 7 (48 hours after TPE), the patient met the extubation criteria defined in the Methods (Table 4). A spontaneous breathing trial followed on a T-piece circuit with supplemental oxygen (3 L/min). Respiratory frequency was 15 breaths/min. Average tidal volume was 480 mL. The RSBI was 31.3 breaths/min/L, below the cut-off of 105 breaths/min/L. NIF was -32 cmH₂O (-12 cmH₂O on Day 4). Vital capacity was 18.5 mL/kg (8.5 mL/kg on Day 4). Arterial blood gas values on the T-piece gave a PaO₂/FiO₂ ratio of 360.0 mmHg. Extubation took place at 14:00 on Day 7. Laryngeal stridor, respiratory fatigue, and hemodynamic instability did not follow. Oxygen was given

by low-flow nasal cannula at 2 L/min. A repeat arterial blood gas on room air after extubation gave a PaO₂/FiO₂ ratio of 457.1 mmHg (full values for both samples in Table 4). Motor strength was 4/5 in the upper extremities and 3/5 in the lower extremities, and the MRC sum score was 28/60 (Table 3). Bilateral patellar deep tendon reflexes returned (+1). The GBS disability score fell from 5 to 4 (Table 3).

On Day 8 (72 hours after TPE and the fifth calendar day in the ICU), the patient remained clinically stable. He breathed room air with stable hemodynamics. The MRC sum score was 30/60. Biceps and triceps reflexes were graded +2 (Table 3). The temporary hemodialysis catheter was removed. The patient then left the ICU for the general neurology ward. The ICU stay ran from Day 4 to Day 8 (4 days by date). Invasive mechanical ventilation lasted about 3 days (intubation on Day 4, extubation at 14:00 on Day 7) (Table 3; Table 4; Figure 2).

On the general neurology ward, the patient followed a daily structured neurorehabilitation protocol. It consisted of bedside passive and active range-of-motion exercises, respiratory muscle training, and progressive neuromuscular re-education. No secondary neurological fluctuations appeared. Nor did any apheresis rebounds or autonomic instabilities.

The subacute follow-up took place on Day 14 (nine days after TPE). Motor strength was 4+/5 in the upper limbs and

4/5 in the lower limbs. The MRC sum score was 38/60 (Table 3). Deep tendon reflexes were brisk and symmetrical (+2) in all four limbs. With assistance he could stand. Two therapists physically supported him as he walked 10 meters. His GBS disability score was grade 3.

By Day 21 (hospital discharge), motor power was 5/5 in both upper extremities and 4+/5 in both lower extremities. The MRC sum score was 44/60 (Table 3). He stood independently and transferred safely. He walked unassisted for more than 20 meters with a standard rolling walker. His GBS disability score remained 3 (Figure 6). He was discharged home in stable condition with an outpatient physical therapy regimen.

4. Discussion

4.1. Principal lesson of the case

The main lesson of this case is that rapid recovery can follow a single large-volume TPE session in fulminant AMAN, although guidelines recommend a multi-session course. This report describes that course. It does not show that the exchange caused the recovery.

An elderly patient with fulminant AMAN, dependent on a ventilator, recovered neuromuscular function and came off the ventilator after one abbreviated, large-volume session of TPE (quantitative trajectory in Figure 6). This course differs from the multi-session treatment recommended in international consensus guidelines. They call for five to six plasma exchanges spread over 10 to 14 days, or a five-day course of high-dose IVIg (2 g/kg).^{5,7} Neurocritical care teaching describes AMAN as a severe and aggressive phenotype of GBS. Prolonged mechanical ventilation is expected (often beyond 30 to 60 days), with tracheostomy in 30% to 50% of patients, a long ICU stay, and slow,

incomplete axonal regeneration at about 1 mm per day.^{25,26} Our patient was extubated within 48 hours after apheresis and left the ICU within 72 hours. By Day 21 he walked independently with an assistive device. Section 4.3 sets out possible explanations, all of which are hypotheses.

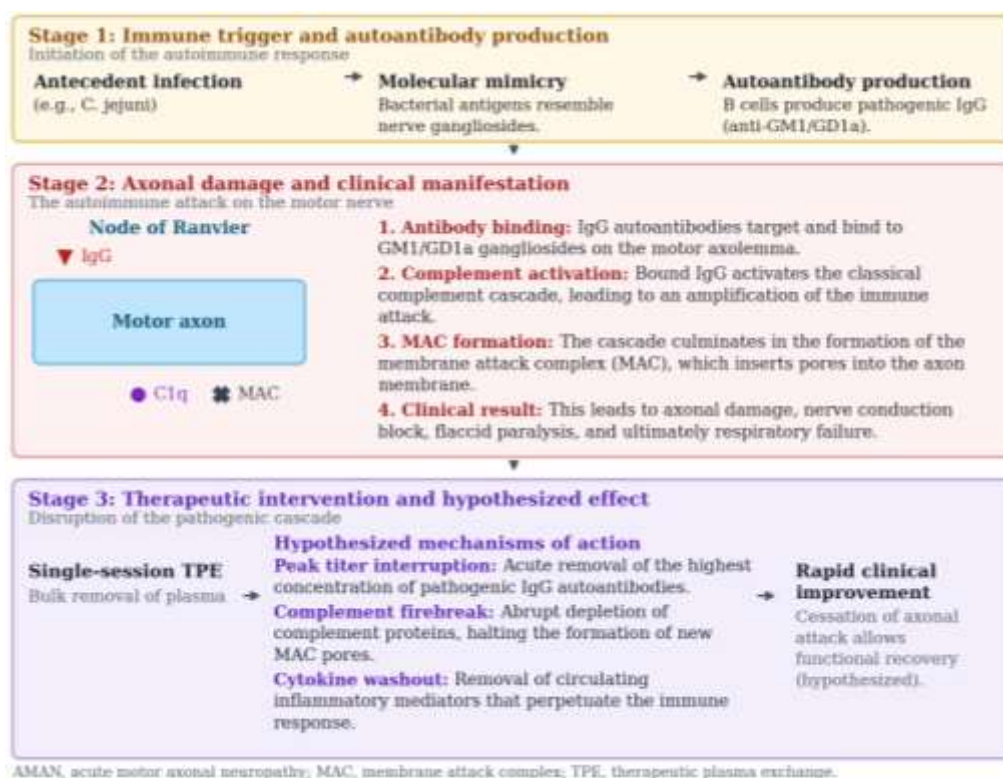
4.2. Comparison with the published literature

Set against the international literature, the course seen here differs from that of established prognostic cohorts.^{27,28} Multicenter randomized trials found that patients with severe GBS did significantly worse with two plasma exchange sessions than with four. That established the multi-session standard of care.^{29,30} Prospective data from the International Guillain-Barré Syndrome Outcome Study (IGOS) point the same way. Patients with severe AMAN on mechanical ventilation needed a median of 28 to 42 days of ventilatory support, and fewer than 15% were extubated in the first week of ICU admission.

Our patient was ventilated for about 3 days, from intubation on Day 4 to extubation on Day 7. The total ICU stay was 4.0 days. This brevity contrasts with the reports above. It also contrasts with the cohort data. It is compatible with the idea that interrupting the autoimmune cascade at an early point can shorten ventilation, but one patient cannot show this (Figure 6).^{27,28}

4.3. Pathophysiological interpretation

The mechanisms discussed below are hypotheses. What was observed in this patient was the timing of recovery, the improvement in dynamic lung compliance, and the normalization of arterial blood gases (Figure 6).²⁹⁻³³ Figure 7 shows the proposed three-stage pathophysiology of AMAN and the proposed effect of a single TPE session.



AMAN, acute motor axonal neuropathy; MAC, membrane attack complex; TPE, therapeutic plasma exchange.

Figure 7. Hypothesized three-stage pathophysiology of AMAN and proposed effect of single-session TPE.

A possible explanation starts from the molecular targets of AMAN, laid out in the multi-tier cascade of Figure 8.^{5,34-36}

AIDP has been interpreted mainly as a cellular demyelinating process, largely through T-cell-mediated

models. AMAN differs. It is better described as a humoral, antibody-mediated autoimmune nodopathy.⁵ Circulating IgG autoantibodies drive the injury by targeting specific gangliosides, mainly GM1 and GD1a, which cluster densely along the unmyelinated axolemma at the nodes of Ranvier and paranodal junctions of motor axons.^{12,36} In our patient, the antecedent dry cough may have marked an initial sensitizing immune event. B lymphocytes could then have

expanded clonally and produced cross-reactive IgG autoantibodies through molecular mimicry. The nodes of Ranvier have no dense myelin covering, and the blood-nerve barrier is described as physiologically deficient at motor nerve terminals and spinal roots. Circulating autoantibodies may therefore reach these nodal gangliosides directly.

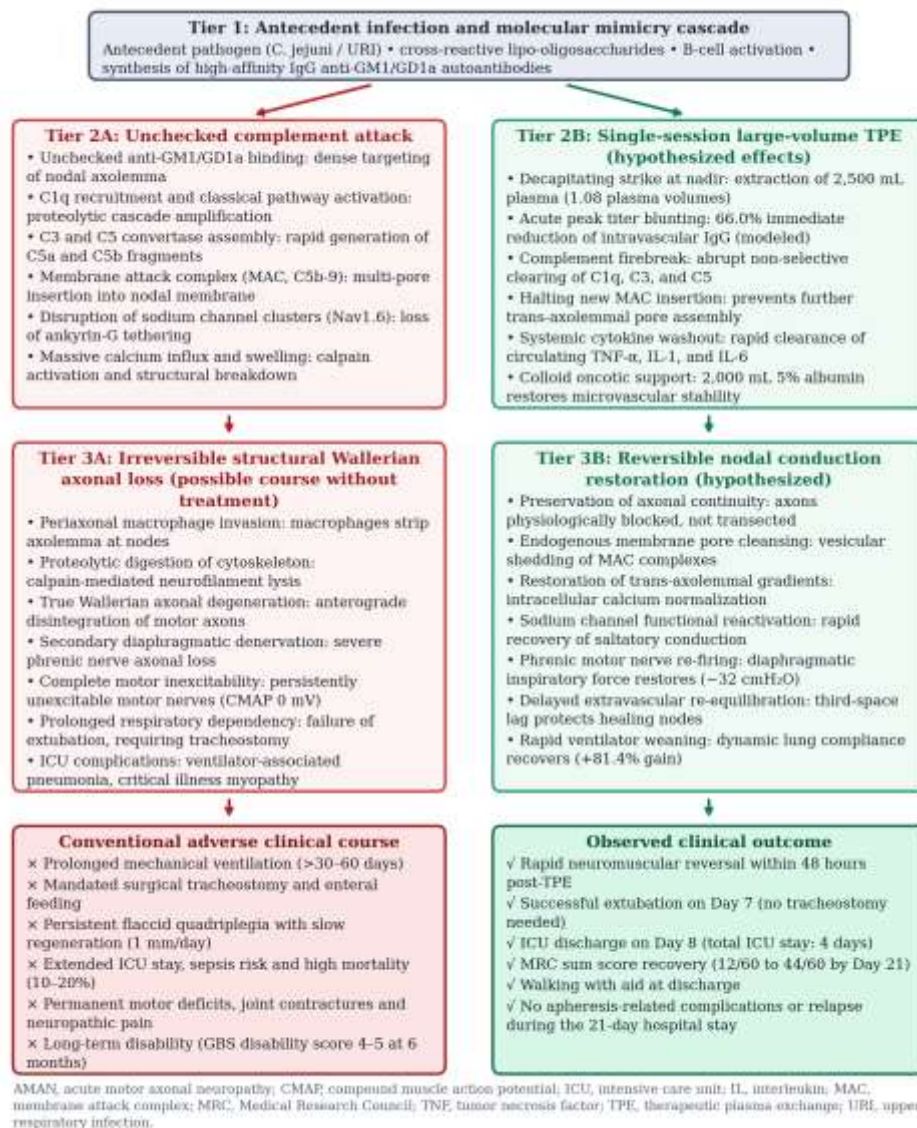


Figure 8. Hypothesized multi-tier cascade illustrating molecular mimicry, nodal ganglioside autoantibody attack, complement-mediated axolemmal pore formation, and the proposed interruption by single-session large-volume TPE.

Autoantibody binding to nodal gangliosides triggers the classical complement cascade (Figure 8).^{5,36} C1q is recruited and attaches to the Fc domains of clustered IgG molecules. A proteolytic amplification chain follows through C4, C2, and C3. It ends in activation of C5 convertase and formation of C5b-9 membrane attack complex (MAC) pores in the axolemmal membrane.^{5,37} The pores damage the nodal membrane, and extracellular calcium and sodium flow uncontrolled into the axoplasm.³⁷ Rising intracellular calcium activates calcium-dependent calpains, which disrupt the cytoskeletal scaffold (ankyrin-G and beta-IV spectrin among its parts) that holds voltage-gated sodium channels (Nav1.6) together at the nodal gap.^{12,37} Saltatory conduction fails. Paralysis is flaccid. Respiratory failure follows.^{12,36} Autoantibody synthesis, complement activation, and MAC assembly all occur within

the intravascular and interstitial fluid compartments, so the bloodstream is a rational target for extracorporeal macromolecular clearance, such as TPE (Figure 8).^{4,5}

We propose five interlocking hypotheses. They address how one well-timed, large-volume plasma exchange could change the natural history of fulminant AMAN if the proposed mechanisms operate. They are named 'peak titer interruption', 'complement firebreak', 'reversible nodal conduction block', 'altered IgG pharmacokinetics in critical illness', and 'cytokine washout and oncotic stabilization' (Figure 8).

The first hypothesis, 'peak titer interruption', proposes that the exchange interrupted the humoral cascade near its peak (Figure 8). In acute GBS, pathogenic autoantibody titers are thought not to stay constant. They may follow a dynamic, bell-shaped curve over time. After the initial

antigen exposure, cross-reactive IgG titers would climb for two to three weeks. They would peak when immune activation and clonal plasma cell output are greatest, then fall as endogenous clearance and regulatory immune circuits take over. The clinical nadir (maximal weakness, respiratory failure) is proposed to coincide often with this peak antibody concentration. Our patient reached respiratory collapse on Day 4 after a three-week subacute progression. This would place his disease near the proposed immunological peak. Day 5 brought a 2,500 mL plasma exchange (1.08 plasma volumes). By the authors' calculation, that removed about 66.0% of the total intravascular IgG pool in one session (Figure 6); this figure is an estimate and depends on the plasma volume used (Section 4.6).^{38,39} The exchange fell near the proposed peak of autoantibody production, so it would be expected to clear a large absolute mass of circulating anti-ganglioside antibodies. Timing could have helped further. The patient was entering the fourth week of illness, when endogenous antibody synthesis may have begun to wane, and a 66.0% extraction could then have pushed the total-body antibody burden below the level needed to sustain axolemmal binding and complement recruitment (Figure 6; Figure 8).

The second hypothesis, 'complement firebreak', rests on transient systemic complement depletion (Figure 8). The autoantibody does not by itself complete the injury at the nodal axolemma. The complement cascade it activates does.^{5,12,36} Classical complement is a strong enzymatic amplifier. One antibody molecule bound to the nodal membrane can seed the deposition of hundreds of C5b-9 MAC pores.^{5,40} Plasma exchange removes plasma in bulk, without selectivity, so exchanging 1.08 plasma volumes with an albumin-saline replacement would be expected to deplete circulating complement factors, among them C1q, C3, and C5, by an estimated figure of over 75% (not measured in this patient).^{5,41,42} Complement depletion would have been brief. It would have removed a substrate the destructive process needs.^{5,41} Some autoantibodies may have stayed bound to the axolemma or shifted back from the interstitial space, yet without circulating complement new MAC pores could not form as readily.^{12,43} A therapeutic window of 48 to 72 hours may have opened. Throughout it, the motor nerve axolemma could have escaped continuing lytic attack, and cellular repair could have shifted the balance toward structural preservation (Figure 8). The window is an inference, because no complement level was measured.

The third hypothesis, 'reversible nodal conduction block', is the one the authors consider most relevant to this patient's course (Figure 8).^{36,44,45} Early in AMAN, motor nerves may be completely inexcitable on the first electromyography. That does not necessarily mean irreversible axonal transection or Wallerian degeneration, and reversible conduction failure of this kind is described in AMAN.^{44,45} Rapid recovery can also occur in AMAN with reversible conduction failure without exchange. The pace of recovery alone therefore cannot separate a treatment effect from the natural course. Experimental models show that anti-ganglioside antibodies and MAC pores first cause a functional conduction block. It is thought to involve axolemmal depolarization and sodium channel inactivation, while the axonal core stays intact for the time being.^{12,36} Nodal architecture could still be saved if complement activation stops before periaxonal macrophages enter the nodal space and phagocytose the axon.^{12,43} Schwann cells and motor axons may use their own repair mechanisms. They include endocytosis and

vesicular shedding of MAC pores, repolarization by Na⁺/K⁺-ATPase pumps, and re-clustering of Nav1.6 channels.^{12,46} Once the lytic pores are cleared and intracellular calcium normalizes, saltatory conduction could resume, in the authors' estimate within 24 to 48 hours.^{45,47} Recovery in our patient was rapid. Dynamic lung compliance improved by 81.4%, arterial blood gases normalized, and extubation followed at 48 hours after apheresis (Figure 6). Axonal regrowth cannot account for this. It would need months to span the distance from the cervical spinal cord to the diaphragm. The rapid functional recovery is compatible with reversible nodal conduction block, rather than completed axonal necrosis, as the cause of his respiratory failure. No repeat nerve conduction study was done to test this (Figure 6; Figure 8).

The fourth hypothesis, 'altered IgG pharmacokinetics in critical illness', concerns the behavior of IgG during life-threatening illness. Classical apheresis models assume that intravascular IgG rebounds to 60% to 80% of baseline within 48 hours, because IgG redistributes quickly from the extravascular space.^{42,48} In the authors' view, that model came from stable volunteers who were not severely ill, and it may not fit ICU patients. Our patient had acute respiratory distress and severe physiological stress. Such conditions are known to change microvascular endothelial permeability, cause systemic capillary leak, and expand the third space.⁴⁹ Under these conditions, much extravascular immunoglobulin may be held in interstitial tissue. Sluggish microcirculatory perfusion and impaired lymphatic drainage could slow its return to the intravascular compartment further. The usual 48-hour redistribution curve might then be flattened and stretched to 72 or 96 hours. That would lengthen the post-apheresis antibody nadir. The motor nerves would gain a longer protected period to finish membrane repair, with no sharp rebound in circulating antibody titers (Figure 6).

The fifth hypothesis, 'cytokine washout and oncotic stabilization', covers both the removal of inflammatory mediators and the effect of the replacement fluid on oncotic pressure and hemodynamics. GBS comes with raised circulating levels of pro-inflammatory cytokines, such as tumor necrosis factor- α , interleukin-1, and interleukin-6. These may worsen blood-nerve barrier permeability and sustain B-cell activation.⁵⁰ Removing plasma in bulk would also remove these inflammatory mediators, which could dampen secondary microvascular inflammation.⁴¹ Replacement fluid may have mattered as well. Albumin at 5% (2,000 mL) replaced 80% of the extracted plasma volume and raised serum albumin from 3.8 g/dL to 4.2 g/dL. This may have helped to maintain intravascular oncotic pressure and to limit microvascular fluid shifts. End-organ perfusion and hemodynamic stability were maintained through the weaning window (Table 5).

These five hypotheses are not mutually exclusive. This report cannot test any of them, because the antibody, complement, and cytokine measurements and the repeat electrophysiology that would be needed are missing.

4.4. Implications for practice

These observations raise a question for ICU stewardship and global health, especially in low- and middle-income countries (LMICs), that could be examined in further study.⁵² The authors state that across much of Southeast Asia, South Asia, Africa, and Latin America a full five-session TPE course often costs several thousand United States dollars. A full course of IVIg costs similarly.

Uninsured patients cannot afford either, and both strain municipal healthcare budgets.^{7,51} The authors also state that shortages of replacement human albumin, specialized apheresis tubing sets, and central venous hemodialysis catheters force clinicians to cut therapy short.⁵¹ Where a full course cannot be given, it is worth asking whether one large-volume exchange timed near the clinical nadir could reduce prolonged mechanical ventilation, ventilator-associated pneumonia, and surgical tracheostomy. This single case cannot answer that question.

We emphasize that this report does not argue for routinely scaling back the standard, evidence-based multi-session TPE protocols in GBS. Nothing in it supports replacing the multi-session protocol.^{7,30} It is a hypothesis-generating report. It raises questions about static pharmacokinetic assumptions and about whether neuroimmunological recovery follows a dynamic, non-linear course. The timing of apheresis relative to the immunological curve may matter more than the cumulative number of sessions, but this idea needs testing.

4.5. Strengths

The diagnostic work-up was complete (Table 2; Figure 4). Ventilator settings and arterial blood gases were recorded serially (Table 4), and neurological status was followed with objective scales (Table 3). Ethics approval and consent are documented (Section 2.8). The report states plainly that antibody and complement data are missing.

4.6. Limitations

Several methodological and scientific limitations must be acknowledged. First, design. This is a single-patient case report ($n = 1$). It cannot establish efficacy or a causal relationship, and a single patient cannot match the inferential power of randomized controlled clinical trials. It also cannot exclude that the recovery happened to coincide with a spontaneous, natural plateau in the disease. The natural history of AMAN and reversible conduction failure were not excluded, and rapid recovery can occur in AMAN without exchange.

Second, our patient presented after three weeks of symptoms. This is a mature immune phase in which antibody synthesis may naturally have been plateauing. The same single-session intervention during the hyperacute inductive phase (Days 1 to 7) might have produced a fast rebound, as continuing B-cell synthesis quickly replaced the cleared antibodies.

Further limitations apply. No repeat electrophysiology was performed, so the proposed nodal conduction block was not tested. Follow-up was limited to 21 days. Depending on the formula used to estimate plasma volume, the single exchange of 2,500 mL may have corresponded to 0.94 to 1.01 plasma volumes rather than 1.08. The estimated IgG removal of 66.0% would then be lower.

4.7. Recommendations for future work

Future translational research should test biomarker-guided, dynamic apheresis protocols in prospective multicenter evaluations. These would use point-of-care autoantibody titers, complement activation markers, and advanced neurophysiological monitoring to tailor the intensity and timing of TPE in acute neurocritical care.

5. Conclusion

This case report describes the course of a ventilator-dependent patient with fulminant AMAN after one large-

volume TPE session on Day 5. Respiratory mechanics improved at once. He was extubated within 48 hours after apheresis, left the ICU within 72 hours, and walked independently with an assistive device by Day 21. No secondary neurological fluctuations or apheresis-related complications occurred. This course differs from the multi-session treatment recommended in guidelines. One patient cannot show that the exchange caused the recovery, and the report does not support abandoning multi-session protocols. The transferable lesson is practical. When a patient recovers quickly after an exchange, the timing of recovery, respiratory mechanics, antibody and complement levels, and nerve conduction should be recorded in detail. Future cases can then test the hypotheses raised here.

6. Declarations

Ethics approval and consent to participate The study was conducted in accordance with the principles of the Declaration of Helsinki (2013 revision) and the guidelines of the Council for International Organizations of Medical Sciences (CIOMS). Ethical approval was granted by the Central Medical & Health Committee (CMHC) Ethics Committee, Indonesia (Protocol Approval Number: 2025/0418), in collaboration with institutional authorities at Dr. Moewardi Regional General Hospital and the Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia. Written informed consent was obtained from the patient and his legal surrogate before data analysis.

Consent for publication Written informed consent was obtained from the patient and his legal surrogate for publication of this case report, including all accompanying clinical data, physiological metrics, and diagnostic infographics. A copy of the written consent form is available for review by the journal editor.

Availability of data and materials All data generated or analyzed during this study are included within this published article and its accompanying tables and figures. De-identified physiological datasets are available from the corresponding author upon reasonable academic request.

Artificial intelligence (AI) use statement None. The authors declare that no generative AI tool was used in the conception, analysis, writing, or editing of this manuscript.

Author contribution statement W.P.: Conceptualization; Supervision; Writing – original draft. S.A.P.: Investigation; Data curation. E.J.H.: Investigation (electrophysiological review); Writing – review & editing. All authors (W.P., S.A.P., E.J.H.) contributed to data interpretation, reviewed and revised the manuscript, and approved the final version for submission.

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Conflict of interest None. The authors declare that they have no competing financial, personal, or professional interests that could have influenced the work reported in this paper.

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