CASE REPORT

From Decision to Delivery: A Twelve- to Fourteen-Day Interval Before Plasma Exchange in a Child with Ventilator-Dependent Axonal Guillain-Barré Syndrome

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ABSTRACT

Background: Guillain–Barré syndrome is the commonest cause of acute flaccid paralysis in children. Plasma exchange is accepted when immunoglobulin fails, but the interval from a treatment decision to its delivery is rarely reported.

Objective: To measure the interval between the written decision to escalate to plasma exchange and the first session, and to re-score instruments left uncomputed during admission.

Methods: This CARE-guided case report audited the de-identified clinical record, preserved stated discordances, and recomputed clinical scores, treatment doses, nutritional estimates, and dated intervals.

Results: An 8-year-old boy presented after limb weakness following a diarrhoeal illness, with motor strength graded 1 in all four limbs. Immunoglobulin 0.4 g/kg/day was given for 5 days. Respiratory failure occurred on hospital day 4 and mechanical ventilation continued for 21 days. Plasma exchange was planned on day 10; the first session was completed between days 22 and 24, giving a decision-to-delivery interval of 12–14 days. Three exchanges were completed and a fourth was cancelled. Re-scoring yielded an Erasmus respiratory insufficiency score of 5 or 6 of 7 and Brighton level 2 rather than 1. Weight fell from 77.8% to 70.4% of the documented ideal weight.

Conclusion: At the last review, the disability score was unchanged from admission. Motor recovery was documented before the earliest possible first exchange, so no treatment effect can be claimed. A 10-item minimum data set is proposed.

1. Introduction

Guillain-Barre syndrome is the commonest cause of acute flaccid paralysis in children now that poliomyelitis has been eliminated from most of the world, and it is one of the few paediatric neurological emergencies in which the clock, rather than the diagnosis, governs the outcome. Nationwide and population-based studies place its incidence in childhood between 0.62 and roughly 1 per 100,000 per year: sixty-three paediatric cases were identified in Hong Kong over the study period, giving an estimated annual incidence of 0.62 per 100,000 population, with Miller Fisher syndrome in 16 of them (25.4%)[1]; a nationwide Japanese survey identified 87 children with Guillain-Barre syndrome, 10 with Fisher syndrome and 6 with Bickerstaff brainstem encephalitis over a three-year window, and every child in that survey was able to walk within one year[2]; and a Thai national analysis of 4,521 patients of all ages found the incidence rate rising from 0.48 to 0.93 per 100,000 population over thirteen years with a mortality of 3.5%[3]; and a Korean registry of 11,146 patients found the age-adjusted incidence rising from 0.84 per 100,000 in 2002 to 1.68 in 2018, with 1,221 (11.0%) dying during follow-up[4]. Against those reassuring aggregate figures sit the children who deteriorate. Among 224 critically ill children with Guillain-Barre syndrome, the acute motor axonal neuropathy subtype accounted for 61.6%, mechanical ventilation was required in 54.9% and tracheostomy in 24.2%[5]; in a combined European cohort of 421 children, 79 (19%) required mechanical ventilation[6]; and in 102 Ethiopian children, thirteen (12.7%) died, with respiratory failure the only determinant of mortality (adjusted odds ratio 11.40, 95% confidence interval 1.818 to 71.52, p = 0.009)[7].

Two immunotherapies change the course of the disease and no third has yet replaced them. A double-blind randomised trial that assigned 81 adults 2:1 to plasma exchange (n = 54) or immunoglobulin (n = 27) found significantly more improvement in the immunoglobulin arm at one and three months and no difference at one year[8]. Newer agents have not displaced either: a phase 3 trial of eculizumab in 57 participants with severe disease (37 eculizumab, 20 placebo) did not achieve its primary endpoint of time to a functional grade of 1 or less (hazard ratio 0.9, 95% confidence interval 0.45 to 1.97, p = 0.89)[9], and an observational comparison of efgartigimod with immunoglobulin in 22 patients, eleven per group, reported week-2 response rates of 90.9% and 81.8% respectively[10]. What happens when the first course of immunoglobulin does not work is, by contrast, almost entirely unstudied. A randomised trial of a second immunoglobulin dose in 93 patients selected for poor prognosis returned an adjusted common odds ratio for improvement on the disability score at four weeks of 1.4 (95% confidence interval 0.6 to 3.3, p = 0.45)[11], and a systematic review of the alternative found 37 articles – one randomised trial, three observational studies and 33 case reports or series – comprising 422 patients, with no superiority of either repeat immunoglobulin or plasma exchange on any clinical outcome measure[12]. That review is the single most directly relevant piece of evidence to the situation described below, and its conclusion is that the evidence does not decide between the two options.

Underneath that unresolved question sits a more practical one that the literature almost never quantifies: whether plasma exchange can be delivered at all, and how long it takes to arrive. The procedure needs an apheresis machine, a trained operator, replacement colloid and reliable large-bore vascular access, and in most of the world at least one of those belongs to a department other than the one caring for the child. The evidence that it works in children is now reasonably good and comes largely from resource-constrained settings: in 36 children with severe disease receiving 122 exchange sessions, upper-extremity power on the Medical Research Council scale rose from 1.7 ± 1.1 to 3.7 ± 0.9, an increase of 2.0 ± 1.1 (95% confidence interval 1.6 to 2.4, $p < 0.001$)[13]; among 23 children with acute transverse myelitis, Guillain-Barre syndrome or acute flaccid myelitis treated by apheresis, 17 (74%) improved significantly by the end of treatment[14]. The access question is measured differently and less often. A survey of 328 responses from 238 hospitals in 60 countries found that only 77 paediatric intensive care units (52.7%) had a paediatric intensivist or anaesthetist present around the clock and 71 (49.7%) had nurse-to-patient ratios below one to two[15]. Where access has been measured directly it has been found unequal: among 45,515 hospitalisations for Guillain-Barre syndrome, Black patients were less likely to receive plasmapheresis (odds ratio 0.75, 95% confidence interval 0.59 to 0.95), and each higher quartile of postcode income reduced inpatient mortality (odds ratio 0.80)[16].

Delay before the first treatment has been studied only at the front door. Of 100 adults with Guillain-Barre syndrome at a single centre, 50 were treated within 24 hours of arrival and 50 later, and 41 of those 50 were not initially given a clear-cut diagnosis, orthopaedic (11 of 41), vascular (7 of 41) and nutritional (6 of 41) alternatives being considered first[17]. In children the same problem is larger: among 21 children with the syndrome there were 40 separate misdiagnoses among 17 patients and a median diagnostic delay of 9 days (interquartile range 6 to 17)[18]. But a delay before the FIRST treatment is a diagnostic delay, and it is not the same quantity as the delay between a decision to escalate that has already been written in the notes and the delivery of the treatment that decision names. The second interval is a property of the health system rather than of the clinician's reasoning, it is measurable to the day from any set of ward notes, and we have found no paediatric report that states it.

This report sets out that interval in a single child. An 8-year-old boy with axonal Guillain-Barre syndrome who became ventilator dependent on hospital day 4 completed a five-day course of immunoglobulin without recovering, and plasma exchange was written into the plan on hospital day 10. The first exchange was completed between hospital days 22 and 24, an interval of 12 to 14 days, and the record names the reasons in its own words: on hospital day 17 the anaesthesiology service had been contacted but the referral had not yet been connected, and a fourth exchange was cancelled outright because the central dialysis line access was blocked. The novelty of this report is twofold. First, it quantifies the escalation interval as a distinct, dated and modifiable quantity, separate from diagnostic delay, and shows that in this child it consumed more time than the entire immunoglobulin course. Second, it re-scores the record against the instruments the disease already has – the Brighton case definition, the Erasmus respiratory insufficiency score, the disability score, the Medical Research Council sum score and a nutritional index – and shows that the information needed to compute every one of them was present in the notes on the day of admission and that none of them was computed. Our aim is therefore not to claim that plasma exchange rescued this child; the record itself forbids that claim, and we set out why. It is to measure what the record will support – an interval, a set of scores that were computable and were not computed, and 18 internal disagreements that we declare rather than smooth over – and to close with a 10-item minimum data set that any unit treating a child with this syndrome can adopt without buying equipment.

2. Methods

Case design and reporting framework

This CARE-guided, single-patient retrospective case report was prepared from routine clinical records at a tertiary paediatric referral centre in Padang, West Sumatera, Indonesia. It describes the illness from symptom onset through the second outpatient review.

Patient information and consent

Clinical history, examination findings, laboratory and imaging results, respiratory support, medications, nutrition, motor assessments, and daily progress were abstracted from the de-identified clinical record. Written informed consent for publication, including the radiographic and magnetic resonance images, was obtained from both parents as the child's legal guardians, together with age-appropriate assent from the child. Direct identifiers were removed before analysis and publication.

Clinical record audit and analysis

Recorded values were transcribed without silently reconciling discordances. Absolute blood-cell counts, the neutrophil-to-lymphocyte ratio, prognostic nutritional index, Medical Research Council sum scores, Erasmus scores, Brighton diagnostic level, weight-for-height percentages, body mass index, per-kilogram treatment exposures, energy estimates, endotracheal-tube depth, plasma-exchange volume, and dated intervals were recomputed from source values. Each calculation was checked against the cited definition or comparator.

Ethical considerations

Ethical approval for the preparation and publication of this case report was granted by the Research Ethics Committee of CMHC Research Center, Indonesia (reference no. CMHC/VI/2026/020). The report was prepared in accordance with applicable ethical standards, and all clinical information was de-identified before analysis and publication.

3. Results

Presentation, antecedent illness and the record's own arithmetic

An 8-year 9-month-old boy was referred to a tertiary paediatric centre in Padang, West Sumatera, with weakness of all four limbs. The demographic and clinical characteristics of the patient, together with every place at which the source record states the same quantity a second time, are set out in Table 1. The chief complaint dates the weakness to 4 days before admission; the history of present illness in the same document dates the weakness of the hands to 3 days before admission and the pain in both legs to 6 days before. We carry both readings forward rather than choosing between them, and every interval computed from the onset of weakness in this report is therefore stated as a range.

The antecedent illness is the classical one. 9 days before admission the child passed loose stools 3–4 times a day for 4 days, each stool estimated at 20 to 100 mL, without mucus or blood and without fever. That places the interval from the

onset of diarrhoea to the onset of weakness at 5 to 6 days. No stool culture, no stool antigen test and no serology for any enteric pathogen appears anywhere in the record, and no anti-ganglioside antibody was requested at any point. A fall in the sitting position is recorded, 3 days before admission in the history of present illness and 4 days before admission in the opening narrative of the same document; cramping pain in both legs is said to have begun three hours afterwards, and it is this sequence that produced vertebral trauma as a differential diagnosis on admission. There had been contact with a person with coronavirus disease 2019 and had received 2 doses of a SARS-CoV-2 vaccine, the last in March 2022, with a measles-rubella immunisation in July 2022. There had been no defecation for 4 days before admission, while micturition was normal in colour and frequency and the child could still feel and defer it. Medication before referral comprised a B-vitamin preparation, methylprednisolone 50 mg intravenously four times a day, mecobalamin, ketoprofen and ranitidine. There was no family history of similar illness; birth, immunisation, growth and development were recorded as normal, and the household's sanitation and food quality were described as good.

On admission the child appeared moderately ill and was fully conscious. Blood pressure was 100/60 mmHg, heart rate 90 beats per minute, respiratory rate 20 breaths per minute and temperature 37.0 °C. Weight was 21.0 kg and height 132.0 cm; the nutritional assessment form in the same record derives an ideal weight for height of 27.0 kg and a height-age of 8 years 10 months. Pupils were 2 mm and equal with normal light reflexes, the tonsils were T1-T1 and not injected, the chest was clear on auscultation and the abdomen soft with normal bowel sounds; puberty was A1P1G1. There were no signs of meningeal irritation: no neck stiffness, negative Brudzinski I and II and a negative Kernig sign. Physiological reflexes were reduced and pathological reflexes absent. Motor strength was graded 1 in each of the four digits recorded for the upper limbs on both sides and 1 in each of the four recorded for the lower limbs on both sides, and there was hyperaesthesia in all four extremities. Swallowing was normal, there was no facial weakness and there was no shortness of breath.

Table 1. Demographic and clinical characteristics of the patient, and every quantity that the source record states more than once.

Item	As the record states it	Where the record states it differently	Recomputed here, or the resolution adopted
Sex and age	male, 8 years 9 months	**9 years in the narrative**	the nutritional assessment form and the imaging header agree on 8 years; every paediatric reference used here is indexed by age
Weight and height on admission	21.0 kg, 132.0 cm	stated once	body mass index 12.05 kg/m^2
Ideal weight for height, and height-age	27.0 kg; height-age 8 years 10 months	stated once	derived by the record from the growth reference; used here unchanged
Weight for height	77%	stated once	77.8% of the record's own ideal weight
Chief complaint	weakness of all four limbs	stated once	-
Antecedent illness	diarrhoea 9 days before admission, 3–4 times a day for 4 days, 20–100 mL per stool, no mucus or blood, no fever	stated twice in identical terms	5 to 6 days from the onset of diarrhoea to the onset of weakness
Investigation of that antecedent	*no stool culture, no stool antigen, no enteric serology*	*no anti-ganglioside antibody at any point*	**the single most informative pair of tests, both obtainable on the day of admission, and neither requested**
Bowel and bladder function	no defecation for 4 days before admission; micturition normal in colour and frequency, sensation and continence preserved	stated once	constipation without urinary involvement, compatible with autonomic involvement of the gut
Vital signs on admission	100/60 mmHg, 90 beats/min, 20 breaths/min, 37.0 °C	stated once	-
Cranial nerves and bulbar function	pupils 2 mm and equal with normal light reflexes; tonsils T1-T1 and not injected; swallowing normal	no facial weakness and no dysphagia are recorded at any point	the absence of cranial nerve involvement is one of the three items of the paediatric respiratory score[6]
Meningeal signs	no neck stiffness, Brudzinski I and II negative, Kernig negative	stated once	-
Reflexes	physiological reflexes reduced, pathological reflexes absent on admission	patellar and Achilles reflexes absent from hospital day 14	reduced or absent reflexes in the weak limbs satisfies item 2 of the case definition[19]
Sensation	hyperaesthesia in all four extremities, cramping pain in both legs	**the nerve conduction report states no sensory involvement**	both are reported; no sensory examination is recorded after admission and no pain score appears on any day
Immunisation and exposure	2 SARS-CoV-2 vaccine doses, the last in March 2022; measles-rubella in July 2022; household contact with coronavirus disease 2019	stated once	the last dose precedes the onset of weakness by about nine months, outside the risk windows used in the pharmacoepidemiological literature[20]
Medication before referral	a B-vitamin preparation, methylprednisolone 50 mg intravenously four times daily, mecobalamin, ketoprofen, ranitidine	stated once	**corticosteroid, which has no established benefit in this syndrome, had been given before referral and is not mentioned again**

Item	As the record states it	Where the record states it differently	Recomputed here, or the resolution adopted
Age of the patient	9 years, in the narrative	**8 years 9 months on the nutritional assessment form and 8 years in the header of the radiographic study**	8 years 9 months is adopted, the two independent sources agreeing
Onset of limb weakness	4 days before admission, in the chief complaint	**weakness of both hands 3 days before admission, in the history of present illness**	both readings are carried and every interval from onset is stated as a range
Timing of the fall	4 days before admission, in the opening narrative	**3 days before admission, in the history of present illness**	declared; the fall bears only on the differential diagnosis, which the imaging settled
Weight for age	77% on the nutritional assessment form	**70% in the admission physical examination**	no pair of weights in the record reproduces 70%; the form's figure is consistent with the recorded weight and the record's own ideal weight
Label given to the nutritional state	malnutrition, in the physical examination	**underweight, on the nutritional assessment form**	neither is accompanied by a z-score or a centile; the recomputed percentages are given instead
Unit of the serum calcium	9.9 mmol/L on admission	**the same unit on hospital days 14 and 24**	read as mg/dL throughout: 9.9 mmol/L is incompatible with life
Electrodiagnostic descriptor	a motor demyelinating axonal peripheral neurogenic lesion	**a conclusion of acute motor axonal neuropathy**	demyelinating and axonal are alternative categories of one taxonomy; the report is reported as unclassifiable rather than resolved
Sensory involvement	the nerve conduction report states no sensory involvement	**the admission examination records hyperaesthesia in all four extremities**	both are reported; normal sensory conduction with sensory symptoms is characteristic of the axonal form and of radicular pain
Notation of manual muscle testing	four digits per limb on admission and at both outpatient reviews	**three digits per limb on the four dates in between**	the grade is identical within each limb on every date, so a per-limb grade is well defined whatever the notation
Power in the left lower limb	graded 5 on hospital days 26, 28 and 30	**the same notes record a child unable to stand or walk**	a grade of 5 is not compatible with the concurrent functional state; the limb is excluded from the reconstructed series and the exclusion is stated
Day number of the immunoglobulin course	the third day of immunoglobulin, in the note of hospital day 4	**the course completed after 5 days, in the note of hospital day 5**	counted from the first dated entry the note of hospital day 4 is the fourth day; the total of 5 days is consistent with the admission plan
Dose unit of methylcobalamin	500 mg three times a day	**no other statement of the dose appears**	read as a unit error: methylcobalamin is dosed in µg
Strength of the ampicillin-sulbactam	500 mg four times a day	**no statement of whether this is the combination or the ampicillin component**	both readings are computed and both fall below the customary lower bound
Cerebrospinal fluid result	reported on hospital day 1	**reproduced verbatim, to the decimal, in the note of hospital day 4**	read as one sample reported twice; no second lumbar puncture is recorded
Date of the first plasma exchange	still planned in the note of hospital day 21	**completed once, in the note of hospital day 24**	carried as the interval hospital day 22 to 24, never as a date
Character of the blood gas sample	reported under an arterial blood gas heading	**annotated by the record itself as having a mixed venous impression**	the acid-base values are used and the oxygen tension and saturation are not
Date of the magnetic resonance study	requested in the plan of both outpatient reviews	**the images carry a clock time and no date**	the study is reported without a date
Date of admission	no admission date is printed anywhere	**the earliest dated entry, 31 December 2022, is headed as a follow-up**	hospital day 1 is defined as that entry and every day is counted from it

The first fifteen rows are the presenting data; the remaining 18 rows are the internal disagreements declared in the inconsistencies subsection of the Discussion, one row each. Red marks a statement that cannot stand beside the one to its left; grey marks a measurement the record does not contain. Hospital day 1 is defined throughout as 31 December 2022, the earliest dated entry in the record; no admission date is printed. Intervals are elapsed days, that is the difference between two calendar dates. Where the record fixes only a range for one endpoint the interval is carried as a range and is never collapsed to a point. The list is an inventory of what a subsequent reader can and cannot reconstruct from this record; it is not an assertion about what was done at the bedside.

Table 1 also carries the recomputations. Weight for height is 77.8% of the ideal weight the record itself derives, against the 77% the record prints; body mass index recomputes to 12.05 kg/m^2. Weight for age is given as 77% on the nutritional assessment form and as 70% in the admission physical examination four pages earlier, and no pair of weights in the record reproduces the second figure. The child's age is given as 9 years in the narrative, as 8 years 9 months on the nutritional assessment form, and as 8 years in the header of the radiographic study; the last two agree and the first does not, and because every paediatric reference used below is indexed by age we adopt 8 years 9 months throughout. Every row of Table 1 that is marked in red is a place at which the record contradicts itself, and the inconsistencies subsection

of the Discussion returns to them as a class rather than as a list.

The course, day by day

The clinical course from the antecedent diarrhoea to the second outpatient review is set out in Figure 1, which places each dated event on a single vertical axis together with the four quantities recomputed here. Hospital day 1 is defined as 31 December 2022, the earliest dated entry in the record and the day on which immunoglobulin was started and the lumbar puncture performed; the record prints no separate admission date, and every hospital day in this report is counted from that entry. Figure 1 is the only place in this report where the whole course appears at once. The whole admission ran to 34 days, and the last outpatient review in the record falls on hospital day 46. Four quantities in Figure 1 appear nowhere in the source record and are marked in the figure as recomputed: the reconstructed sum score, the respiratory insufficiency score, the neutrophil-to-lymphocyte ratio and the prognostic nutritional index.

Figure 1. *The clinical course from the antecedent diarrhoeal illness to the second outpatient review, on a single axis. Hospital day 1 is 31 December 2022, the earliest dated entry in the record; the record prints no admission date. Each entry carries what the record contains, together with the quantities recomputed here and marked as recomputed: the reconstructed Medical Research Council sum score, the Erasmus respiratory insufficiency score, the neutrophil-to-lymphocyte ratio and the prognostic nutritional index. Colour marks the character of each entry rather than its severity: teal for the antecedent illness and the presentation, amber for a new treatment or a new observation, red for a deterioration or for a point at which a computable score was not computed, violet for the escalation interval, green for an observation showing improvement, and grey for routine care. The escalation interval is drawn as a band rather than as a point because the record brackets the first exchange between the notes of hospital days 21 and 24 without dating it.*

Immunoglobulin was begun on hospital day 1 at 0.4 g/kg/day and completed on hospital day 5 after 5 days, a cumulative dose of 2.0 g/kg, which is 42.0 g at this child's weight and is exactly the course the record's own admission plan specifies. A lumbar puncture was performed on hospital day 1. Nerve conduction studies were performed on hospital day 5, the day after intubation. Respiratory failure occurred on hospital day 4 and mechanical ventilation continued for 21 days. Plasma exchange entered the written plan on hospital day 10 and the first session was completed between hospital

days 22 and 24. Three sessions were completed in all and a fourth was cancelled. The child was discharged on hospital day 34 and reviewed as an outpatient on hospital days 39 and 46. The interval before the first exchange is drawn in Figure 1 as a band rather than as a point, for the reason given in the decision-to-escalate subsection of the Results.

Laboratory and cerebrospinal fluid findings

The admission and serial laboratory results, each with its reference interval and its interpretation, are summarised in Table 2, in which values outside the reference interval are set in red. Haemoglobin was 12.6 g/dL, the leucocyte count 21,160/mm³ and the platelet count 512,000/mm³; the differential count read 0/0/1/81/12/6 for basophils, eosinophils, band forms, segmented neutrophils, lymphocytes and monocytes. As shown in Table 2, those percentages give an absolute neutrophil count of 17,351/mm³ counting band forms with the segmented cells and an absolute lymphocyte count of 2,539/mm³, so the neutrophil-to-lymphocyte ratio on the day of admission was 6.83 counting band forms and 6.75 counting segmented cells alone. Serum albumin was 4.6 g/dL, uric acid 3.3 mg/dL and blood glucose 98 mg/dL; sodium was 137 and potassium 4.4 mmol/L. Urinalysis was normal. A SARS-CoV-2 antibody result of 250 U/mL is recorded without an assay name, without a reference interval and without stating whether the target is the spike or the nucleocapsid protein.

Table 2. Laboratory examination results with reference intervals.

Investigation	Hospital day 1	Hospital day 14	Hospital day 24	Reference interval
Haemoglobin (g/dL)	12.6	**10.1**	**10.6**	11.5–15.5 g/dL
Leucocytes (/mm³)	**21,160**	**15,890**	5,850	4,500–13,500
Neutrophils, segmented (%)	**81**	*not recorded*	*not recorded*	35–70%
Band forms (%)	1	*not recorded*	*not recorded*	< 5%
Lymphocytes (%)	**12**	*not recorded*	*not recorded*	25–45%
Monocytes (%)	6	*not recorded*	*not recorded*	2–8%
Absolute neutrophil count (/mm³)*	**17,351**	*not computable*	*not computable*	1,500–8,000
Absolute lymphocyte count (/mm³)*	2,539	*not computable*	*not computable*	1,500–7,000
Neutrophil-to-lymphocyte ratio*	**6.83**	*not computable*	*not computable*	< 2 in health
Platelets (/mm³)	**512,000**	**647,000**	449,000	150,000–450,000
Reticulocytes (%)	**2.8**	*not recorded*	*not recorded*	0.5–1.5%
Mean cell volume (fL)	84	*not recorded*	*not recorded*	77–95 fL
Mean cell haemoglobin (pg)	29	*not recorded*	*not recorded*	25–33 pg
Mean cell haemoglobin concentration (%)	35	*not recorded*	*not recorded*	31–37%
Albumin (g/dL)	4.6	**3.1**	3.6	3.5–5.2 g/dL
Prognostic nutritional index*	58.70	*not computable*	*not computable*	> 49.395 and > 45.72[21]
Uric acid (mg/dL)	3.3	*not recorded*	*not recorded*	2–5.5 mg/dL
Glucose (mg/dL)	98	*not recorded*	*not recorded*	70–140 mg/dL
Sodium (mmol/L)	137	**134**	137	135–145 mmol/L
Potassium (mmol/L)	4.4	4.5	3.6	3.5–5.1 mmol/L
Chloride (mmol/L)	*not recorded*	101	101	98–107 mmol/L
Calcium (mg/dL)†	9.9	9.8	9.9	8.8–10.8 mg/dL
Ionised calcium around plasma exchange	*never measured*	*never measured*	*never measured*	**citrate anticoagulation binds ionised calcium**
Cerebrospinal fluid protein (mg/dL)‡	28.6	*not repeated*	*not repeated*	15–45 mg/dL
Cerebrospinal fluid cell count (/µL)‡	1.0	*not repeated*	*not repeated*	< 5 /µL
Cerebrospinal fluid glucose (mg/dL)‡	72	*not repeated*	*not repeated*	40–80 mg/dL
Blood gas pH§	*not measured*	*not measured*	**7.53**	7.35–7.45
Blood gas carbon dioxide tension (mmHg)§	*not measured*	*not measured*	41	35–45 mmHg
Blood gas bicarbonate (mmol/L)§	*not measured*	*not measured*	**34.4**	22–26 mmol/L
Blood gas base excess (mmol/L)§	*not measured*	*not measured*	**10.6**	-2–2 mmol/L
SARS-CoV-2 antibody (U/mL)	250	*not repeated*	*not repeated*	**no assay, no reference interval and no antigen target are stated**
Urinalysis	erythrocytes 0–1/HPF, leucocytes 0–1/HPF, bacteria negative, yeast negative, pH 6.5	*not repeated*	*not repeated*	normal
Electrocardiogram	*never performed*	*never performed*	*never performed*	**no rhythm strip or corrected QT interval on any day**

*Values outside the reference interval are set in red and bold; grey marks an investigation the record does not contain on that date. Rows marked with an asterisk are computed here from the values above them and appear nowhere in the source record. *Derived here. The absolute neutrophil count is the leucocyte count multiplied by the sum of the segmented and band percentages, the absolute lymphocyte count by the lymphocyte percentage, and the neutrophil-to-lymphocyte ratio is their quotient; counting segmented neutrophils alone the ratio is 6.75 rather than 6.83, and both readings are given in the laboratory and cerebrospinal-fluid subsection of the Results. The prognostic nutritional index is ten times the albumin in g/dL plus 0.005 times the absolute lymphocyte count; in an observational cohort of 252 patients it independently predicted disease severity (odds ratio 0.91, p = 0.036) and 26-week outcome (odds ratio 0.93, p = 0.033), with cut-offs of 49.395 and 45.72 respectively.[21] A differential count was performed only on admission, so none of the four derived quantities can be recomputed on the later dates. †The record prints all three calcium values in mmol/L; a serum calcium of 9.9 mmol/L is incompatible with life and the values are read here as mg/dL, in which unit all three lie within the reference interval. ‡One lumbar puncture was performed, on hospital day 1; the identical protein, cell count and glucose are reproduced verbatim in the note of hospital day 4 and no second sample was taken. The protein is 0.286 g/L against the 0.45 g/L used by the case definition, so there is no albuminocytological dissociation; in the international cohort that dissociation was present in 846 of 1,231 patients (70%), rising from 57% at four days or less from the onset of weakness to 84% afterwards.[19,22] §The record reports this sample under an arterial heading and annotates it as having a mixed venous character; the acid-base values are used here and the oxygen tension of 31 mmHg and saturation of 69% are not, because a venous sample cannot measure oxygenation. The reference intervals are those for a child of school age; where the source laboratory printed its own they are used unchanged.*

Two entries in Table 2 need comment before the discussion. The first is the calcium. The record prints 9.9 mmol/L on admission and repeats the same unit on hospital days 14 and 24. A serum calcium of 9.9 mmol/L is incompatible with life; every one of the three values is a milligram-per-decilitre result carrying a millimole label, and Table 2 reads them as mg/dL, in which unit all three lie within the reference interval. The second is the cerebrospinal fluid, and it decides the diagnostic certainty of the whole case. The fluid obtained on hospital day 1 was clear and non-turbid with a protein of 28.6 mg/dL, a cell count of 1.0 per microlitre and a glucose of 72 mg/dL. That protein is 0.286 g/L, which is 16.4 mg/dL below the upper reference value of this laboratory and 63.6% of the 0.45 g/L that the Brighton case definition uses. There is therefore no albuminocytological dissociation. The identical protein, cell count and glucose are reproduced verbatim in the ward note of hospital day 4; the lumbar puncture was never repeated.

Two further panels were taken later in the admission and are tabulated in the last two result columns of Table 2, where the values that fall outside the reference interval are marked in red. On hospital day 14 haemoglobin had fallen to 10.1 g/dL and albumin to 3.1 g/dL, falls of 2.5 g/dL (19.8%) and 1.5 g/dL (32.6%) from admission, with a platelet count of 647,000/mm^3 and a sodium of 134 mmol/L. No transfusion, no bleeding, no haemolysis screen and no explanation for the fall in haemoglobin appears in the record. By hospital day 24 haemoglobin was 10.6 g/dL, the leucocyte count had fallen to 5,850/mm^3 and albumin had recovered to 3.6 g/dL. An arterial blood gas taken the same day, labelled by the record as having a mixed venous character, read pH 7.53, carbon dioxide tension 41 mmHg, oxygen tension 31 mmHg, bicarbonate 34.4 mmol/L, base excess 10.6 mmol/L and saturation 69%. That is a metabolic alkalosis. The carbon dioxide tension expected for a bicarbonate of 34.4 mmol/L is 39.1 to 49.1 mmHg, and the measured 41 mmHg lies inside that interval, so the respiratory response is appropriate and no second acid-base disorder need be invoked. Because the record labels the sample venous, its oxygen tension and saturation cannot be read as measures of oxygenation and are not so used here.

Imaging

A four-view radiographic survey was obtained on hospital day 4 at 15:45, and the images are reproduced as Figure 2. The four views are a lateral cervical spine, an anteroposterior cervical spine and upper thorax, an anteroposterior chest and an anteroposterior abdomen and lumbosacral spine. They were requested to address the vertebral trauma that the history of a fall had raised, and they show no fracture, no vertebral collapse and no malalignment; the chest view, panel C of Figure 2, shows clear lung fields with no consolidation, no effusion and no pneumothorax. Two things about the timing of that survey matter and are returned to below. It was performed on hospital day 4, 3 days after the differential diagnosis it was meant to settle had been written down; and it was performed at 15:45, which is 1 hour 15 minutes before the respiratory arrest and the intubation that followed at 17:00. It is therefore the only chest radiograph in the record, and it predates the endotracheal tube. Every burned-in identifier in the source images lay outside the film itself and has been removed from Figure 2 by cropping, so no part of any radiograph is covered.

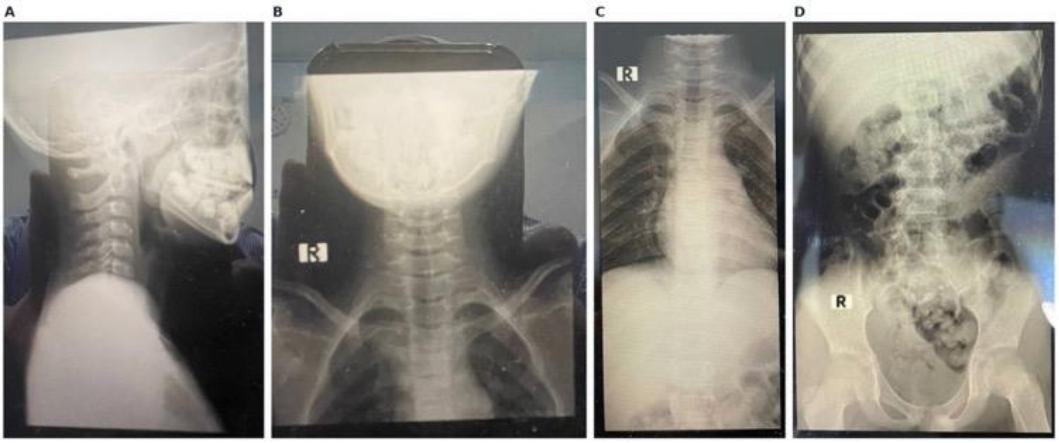

Figure 2. *The four-view radiographic survey obtained on hospital day 4 at 15:45, 1 hour 15 minutes before the respiratory arrest that led to intubation.* ***(A)*** *Lateral cervical spine.* ***(B)*** *Anteroposterior cervical spine and upper thorax, with the right marker visible.* ***(C)*** *Anteroposterior chest.* ***(D)*** *Anteroposterior abdomen and lumbosacral spine. The survey was requested to address the vertebral trauma raised by the history of a fall and shows no fracture, no vertebral collapse and no malalignment; the chest view shows clear lung fields with no consolidation, effusion or pneumothorax, and the abdominal view shows a normal bowel gas pattern with no obstruction. All four images reach us as photographs of a reporting monitor on which a physical film was held, so the greyscale is not the acquired one and no quantitative measurement is made from them here. Panel C is the only chest radiograph in the record and it predates the endotracheal tube; no film confirms the position of that tube. Every burned-in identifier – a patient name in a browser tab, a date of birth, a recorded age, an institution name and a study description – lay outside the film itself and has been removed by cropping rather than by masking, so no anatomical region is obscured and no black rectangle covers any part of the image. The panels are reproduced at their source resolution without upsampling: measured as source pixels per printed inch they run from 270 in the widest panel to 359 in the narrowest.*

Magnetic resonance imaging of the thoracolumbar spine was performed, and representative sagittal images are reproduced as Figure 3. The report states no intramedullary, intradural extramedullary or extradural space occupying lesion, which excludes cord compression, transverse myelitis with a mass effect and an extradural collection as explanations for the tetraparesis. The record does not print the date on which the study was performed; the request appears in the plan of both outpatient reviews, on hospital days 39 and 46, and the images carry a clock time but no date. As shown in Figure 3, the study as it reaches us consists largely of localiser and scout series photographed from a reporting monitor, and only the sagittal series carries diagnostic information at the resolution available; we reproduce those panels and say plainly what the others cannot support.

Two comments on the imaging strategy belong with the images. The four-view survey and the magnetic resonance study together answered the differential diagnosis that the history of a fall had raised, and answered it decisively; the

seventh item of the case definition scored in the diagnostic-certainty subsection of the Discussion rests on the magnetic resonance result. Set against practice elsewhere the strategy was restrained: of 148 patients with this syndrome in one South Asian series, brain computed tomography had been performed in 121 (82%) before the lumbar puncture, and brain and spine magnetic resonance imaging before admission in 48 (32%) and 59 (39%) respectively[23]. No brain imaging was performed here at all. What is open to criticism is the timing rather than the quantity: the survey that settled the differential was obtained on hospital day 4, and the magnetic resonance study of Figure 3, which settles it more completely, was requested only after discharge.

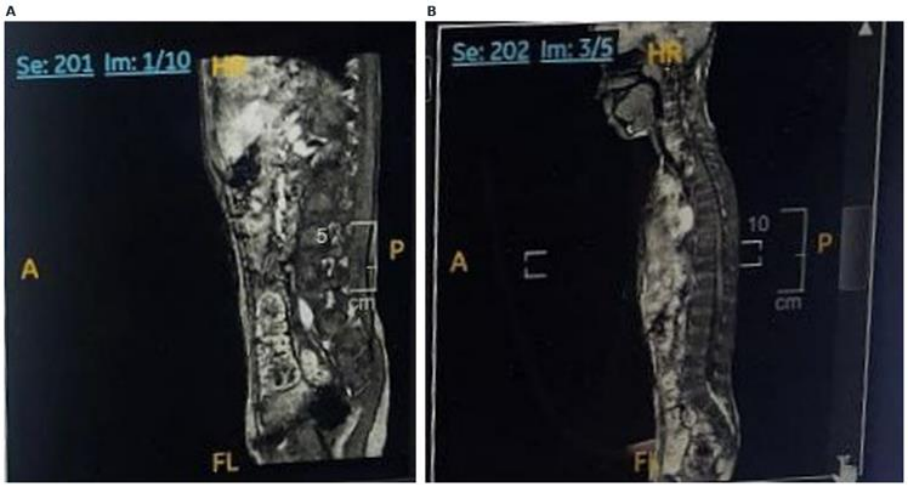

*Figure 3. Sagittal images from the thoracolumbar magnetic resonance study. **(A)** Sagittal series of the thoracolumbar spine. **(B)** Sagittal series extending to the cervical junction. The report states no intramedullary, intradural extramedullary or extradural space occupying lesion, which excludes cord compression, a mass lesion and an extradural collection as explanations for the tetraparesis and is the seventh Brighton item of Table 5. The record does not print the date of the study. The images reach us as photographs of a reporting monitor and the study as photographed consists largely of localiser and scout series; only the sagittal panels reproduced here carry diagnostic information at the available resolution, and the axial and coronal localisers are omitted for that reason rather than because they were not performed. No cord signal abnormality is asserted here: the resolution of these reproductions will not support such an assertion, and the negative finding reported above is the radiologist's, not ours. No patient identifier is present in either panel; the series and image numbers, the orientation letters and the centimetre scale bar are the scanner's own annotations and carry no identifying information. The panels are reproduced without upsampling at 138 to 147 source pixels per printed inch, well below the resolution at which parenchymal or cord signal could be adjudicated, which is why nothing of that kind is asserted here.*

Respiratory failure and mechanical ventilation

On hospital day 4 the child had received the third day of immunoglobulin. In the morning he had a productive cough that he could not clear, a heart rate of 136 beats per minute and a respiratory rate of 23 breaths per minute, and acetylcysteine was added. At 17:00 he became short of breath, cyanosed and restless, with copious secretions he could not expel; oxygen saturation fell to 75% and the respiratory rate to 12 breaths per minute, breath sounds were reduced bilaterally with coarse crackles, and the extremities were cold. He was intubated with a cuffed tube of internal diameter 5.5 mm secured at 18.0 cm and ventilated in a pressure-regulated synchronised intermittent mandatory mode with a positive end-expiratory pressure of 6 cmH_2O, a peak inspiratory pressure of 13 cmH_2O, a set rate of 20 per minute, an expired tidal volume of 10 mL/kg and an inspired oxygen fraction of 70%.

Two arithmetical observations follow from the intubation record and both are detailed in Table 3. The tube size is the one the age-based formula predicts: for a cuffed tube at this age the formula returns 5.69 mm, and 5.5 mm was used. The insertion depth is not. Three times the internal diameter gives 16.5 cm and the age-based estimate gives 16.4 cm, so the recorded 18.0 cm exceeds both by about 1.5 cm. We do not assert that the tube was malpositioned; we observe that no radiograph in this record can settle it, because the only chest film was obtained 1 hour 15 minutes before the tube was placed and none was repeated afterwards. The ward note of the following day records that air entry was present on both sides, which is the clinical substitute for the film that was not taken. The film that does exist is presented in Figure 2 as panel C, and it is 1 hour 15 minutes too early to answer the question.

Ventilation continued for 21 days. The settings were reduced in steps, from a peak inspiratory pressure of 16 cmH_2O with an inspired oxygen fraction of 50% on hospital day 9, to 11 cmH_2O on hospital day 13, to an inspiratory pressure of 8 cmH_2O with an oxygen fraction of 35% by hospital day 21, and to a set rate of 12 per minute by hospital day 24. Spontaneous breathing was recorded at 24 to 27 breaths per minute throughout the weaning period. Sedation with midazolam and morphine was stopped before hospital day 21, and the child was extubated to a simple face mask on hospital day 25. No tracheostomy was performed. No electrocardiogram, no rhythm strip and no corrected QT interval appears anywhere in the record, on any day, at any heart rate.

Drug and nutritional exposures, recomputed per kilogram

Every drug the record prescribes is set out in Table 3, in the words the record uses, recomputed per kilogram at this child's weight and placed beside the range customarily used in children of this age. Most of the regimen is unremarkable: the immunoglobulin course delivered exactly the 2.0 g/kg its own plan specified, ceftriaxone at 95.2 mg/kg/day sits at the upper end of the customary range, acetylcysteine and zinc are conventional, and the zinc dose is the one recommended for childhood diarrhoea. Four rows are not unremarkable, and they are marked in Table 3 in red.

Table 3. Every drug, fluid and nutritional prescription in the record, in the words the record uses, recomputed at the patient's weight, and placed beside the range customarily used in a child of this age.

Agent or item	As the record prescribes it	Recomputed at 21.0 kg	Customary paediatric range	What follows
Intravenous immunoglobulin	0.4 g/kg/day for 5 days	8.4 g/day, 42.0 g in total, 2.0 g/kg cumulative	2 g/kg over 2–5 days	exactly the course the record's own admission plan specifies; a second course has an adjusted common odds ratio of 1.4 (0.6–3.3) for improvement at 4 weeks[11]
Plasma exchange	3 sessions completed, 1 cancelled; the second recorded as 1,100 mL of 750 mL plasma protein fraction, 250 mL succinylated gelatin and 100 mL 0.9% sodium chloride	52.4 mL/kg, about 1.31 estimated plasma volumes	1–1.5 plasma volumes per session	the volume and composition of the first and third sessions are not recorded, and no ionised calcium was measured around any of them
Ceftriaxone	1 g intravenously twice daily	95.2 mg/kg/day	50–100 mg/kg/day	within range, at the upper end
Ampicillin-sulbactam	500 mg intravenously four times daily	**95.2 mg/kg/day, or 63.5 mg/kg/day of the ampicillin component**	100–200 mg/kg/day	**the record does not say whether 500 mg is the combination or the component; on either reading the exposure falls below the customary lower bound**
Midazolam infusion	4.0 µg/kg/min	**0.24 mg/kg/h, 5.04 mg/h**	0.06–0.12 mg/kg/h	**twice the customary upper bound**
Morphine infusion	5.0 µg/kg/h	**0.105 mg/h**	10–40 µg/kg/h	**between 2 and 8 times below the customary lower bound, in a child whose admission examination records hyperaesthesia in all four limbs; no pain score appears on any day**
Dobutamine and adrenaline infusions	5 and 0.05 µg/kg/min	as written	dose-titrated	listed in the same day's plan as the beta blocker below
Propranolol	4 mg orally three times daily	**0.57 mg/kg/day**	1–4 mg/kg/day	**below the customary lower bound, and prescribed on the same day as two beta agonists; no electrocardiogram exists for that day or any other**
Methylcobalamin	500 mg three times daily	**1,500 mg/day**	500–1,500 µg/day	**read here as a unit error: 1,000 times the conventional daily dose**
Acetylcysteine	200 mg twice daily	400 mg/day	200–600 mg/day	within range
Zinc	20 mg once daily	20 mg/day	20 mg/day for a child over 6 months	the dose recommended for childhood diarrhoea
Endotracheal tube	cuffed, internal diameter 5.5 mm, secured at 18.0 cm	**18.0 cm exceeds three times the internal diameter (16.5 cm) and the age estimate (16.4 cm)**	3 × internal diameter	**no radiograph in the record can settle the tube position: the only chest film was taken 1 h 15 min before intubation and none was repeated**
Maintenance fluid and enteral volume, day 1	43 mL/h intravenously plus 6 × 50 mL by nasogastric tube	1,332 mL/day in total	1,365 mL/day at 65 mL/kg/day	97.6% of the requirement the record itself computes; appropriate
Enteral volume at its maximum	6 × 250 mL	1,500 mL/day	-	**the energy delivered cannot be computed: the record never states the energy density of the formula**
Estimated basal metabolic rate	1115.826 kcal, from 19.59 W + 1.303 H + 414.9 at W = 27.0 kg	**998.286 kcal at the actual weight of 21.0 kg**	-	**the ideal rather than the actual weight was substituted, raising the estimate by 117.5 kcal, that is 11.8%**
Estimated energy expenditure	1338.9912 kcal, the above multiplied by a stress factor of 1.2	**1197.9432 kcal at the actual weight**	1,680 kcal at 80 kcal/kg/day	the record's own two targets, 1339.0 and 1,680 kcal, differ by 25.5%; neither can be compared with delivery

Red marks a row in which the recomputed exposure falls outside that range or in which the record cannot be checked at all. The customary ranges are those in general paediatric use and are given to locate each prescription, not as a standard against which any individual decision is judged; the ranges for continuous sedative and opioid infusions in particular are wide and are titrated to effect. Three rows carry a stronger claim than the others because they do not depend on any range: the methylcobalamin dose is printed in a unit 1,000 times larger than the one in which the drug is dosed; the ampicillin-sulbactam strength cannot be interpreted at all without knowing which component the figure refers to; and the energy delivered cannot be computed because the energy density of the formula is never stated. The Schofield weight-and-height equation for boys aged 3 to 10 years is the one the record uses and its arithmetic as printed is correct; the disagreement is over which weight to substitute, and both answers are given. Predicted equations are in any case approximate in hospitalised children: among 109 children undergoing indirect calorimetry the Schofield equation had the highest predictive accuracy at 47.7%.[24]

The first is the analgesia. The morphine infusion is prescribed at 5.0 µg/kg/hour in four separate ward notes, which at 21.0 kg delivers 0.105 mg/hour. Continuous morphine infusions in children of this age are customarily run at 10 to 40 µg/kg/hour, so the prescription is between 2 and 8 times below the lower bound of that range. Beside it the midazolam infusion runs at 4.0 µg/kg/minute, which is 0.24 mg/kg/hour or 5.04 mg/hour, twice the upper end of the range customarily quoted for continuous paediatric sedation. The combination is deep sedation with minimal analgesia in a child whose own admission examination records hyperaesthesia in all four limbs and cramping pain in both legs, and no pain score of any kind – no faces scale, no behavioural scale, no numerical rating – appears anywhere in the record on any day.

The second is the methylcobalamin, prescribed as 500 mg three times a day from hospital day 13, that is 1,500 mg a day. Methylcobalamin is dosed in µg; 500 µg three times a day is a conventional prescription and 500 mg is 1,000 times that amount. We read this as a unit error in the record rather than as an administered dose, and Table 3 says so. The third is the ampicillin-sulbactam started on hospital day 24 at 500 mg four times a day. The record does not state whether 500 mg is the combined amount or the ampicillin component. On the more generous reading, in which 500 mg is the ampicillin component, the exposure is 95.2 mg/kg/day; on the stricter reading it is 63.5 mg/kg/day. Both fall below the 100 mg/kg/day that is customarily the lower bound, and the ambiguity itself is the finding: a prescription that cannot be checked without knowing which number the preparation refers to.

The fourth is the propranolol. On hospital day 5, the day after intubation, the heart rate was 160 beats per minute and the blood pressure 107/68 mmHg. The plan for that day lists, together, a dobutamine infusion at 5 µg/kg/minute, an adrenaline infusion at 0.05 µg/kg/minute and oral propranolol 4 mg three times a day, which is 0.57 mg/kg/day and below the 1 to 4 mg/kg/day customarily used. Two beta agonists and a non-selective beta blocker appear in one day's plan. The record states neither the order in which they were given nor the indication for each, and no haemodynamic measurement other than the single blood pressure and heart rate is recorded for that day.

The nutritional prescription is tabulated in the same table. Maintenance fluid was started as a balanced maintenance solution at 43 mL/hour, that is 1,032 mL/day, alongside a liquid diet of 6 × 50 mL by nasogastric tube, giving 1,332 mL on the first day against the 1,365 mL that the record's own assessment computes at 65 mL/kg/day: 97.6% of requirement, which is appropriate. The enteral volume was then advanced from 50 mL to 250 mL per feed across the admission, reaching 1,500 mL a day. The energy that volume delivered cannot be computed, because the record never states the energy density of the formula. That single missing number is why the whole nutritional prescription is unauditable, and it is item 9 of the minimum data set below.

The record's own energy calculation contains a substitution that Table 3 makes explicit. The Schofield weight-and-height equation for boys aged 3 to 10 years is evaluated in the record as 19.59W + 1.303H + 414.9, and the arithmetic printed on the form is correct: 19.59 × 27.0 + 1.303 × 132.0 + 414.9 = 1115.826 kcal, and multiplying by the stress factor of 1.2 gives 1338.9912 kcal. But the weight substituted is the IDEAL weight of 27.0 kg, not the actual weight of 21.0 kg. Evaluated at the actual weight the same equation returns 998.286 kcal and the same stress factor returns 1197.9432 kcal, so the substitution raises the estimated basal rate by 117.5 kcal, that is 11.8%. Whether the ideal or the actual weight is the right input for a wasted child is a real question with two defensible answers; the point here is that the form does not say which it has used, and the two answers differ by 11.8%.

The decision to escalate, and the interval before it was delivered

Immunoglobulin was completed on hospital day 5 without a return of motor function. Plasma exchange first appears in the written plan on hospital day 10, 5 days later, as the single word "plasmapheresis" under the plan heading. It reappears, still as a plan and not as a procedure, in the notes of hospital days 12, 13, 14, 17 and 21. On hospital day 17 the record states its own reason in one clause: the anaesthesiology service had been contacted for plasmapheresis but the referral had not yet been connected. The note of hospital day 21 still records the child as planned to undergo plasmapheresis. The note of hospital day 24 records that he had completed plasmapheresis once.

The first exchange therefore falls between hospital day 22 and hospital day 24 inclusive; the record does not print the date on which it was performed, only the two notes that bracket it, and we treat that as an interval rather than a date. From the decision of hospital day 10 that gives an escalation interval of 12 to 14 days. From the completion of immunoglobulin it gives 17 to 19 days. From the onset of weakness, taking both readings of the onset date, it gives 24 to 27 days, so on every reading the first exchange fell inside four weeks of onset, by 1 to 4 days.

Three exchanges were completed in all. The second, on hospital day 26, is the only one for which the record gives a volume and a composition: 1,100 mL made up of 750 mL of a plasma protein fraction, 250 mL of succinylated gelatin and 100 mL of 0.9% sodium chloride. That is 52.4 mL/kg, or about 1.31 estimated plasma volumes at the conventional paediatric estimate of 40 mL/kg. The third was given on hospital day 28. A fourth was planned for hospital day 30 at 1,000 mL and was cancelled because the central dialysis line access was blocked. No further exchange was given, and the record contains no calcium measurement, no ionised calcium and no citrate level around any of the three sessions.

Motor strength, functional status and the recorded trajectory

Manual muscle testing as the record prints it, the per-limb grade it implies and the reconstructed Medical Research Council sum score are set out in Table 4, and the trajectories are illustrated in Figure 4. The record prints a group of identical digits for each limb: four digits on admission and at both outpatient reviews, three digits on every date in between. The number of digits therefore changes three times and the grade per limb does not, so a per-limb grade is well defined on every date whatever the notation, and it is the per-limb grade that Table 4 tabulates. The right limb is printed first: the note of hospital day 46 states that the RIGHT hand was still weak on a day when the upper-limb entry reads 2222/3333, which fixes the order. Panel A of Figure 4 plots the four per-limb grades and panel B the two reconstructions.

Table 4. Manual muscle testing exactly as the source record prints it, the per-limb grade it implies, the Medical Research Council sum score reconstructed from that grade, the same reconstruction over three limbs, and the disability score assigned here from the description in each note.

Hospital day	As the record prints it (upper / lower)	Per-limb grade (RU, LU, RL, LL)	Reconstructed MRC sum (of 60)	Three-limb reconstruction (of 45)	Disability score	What the note describes
1	1111 / 1111	1, 1, 1, 1	12	9	4	bedridden, unable to walk
4	*not graded*	*not graded*	*not graded*	*not graded*	5	respiratory failure at 17:00; intubated
13	*not graded*	*not graded*	*not graded*	*not graded*	5	**a motor response in the feet, under sedation; not a graded measurement**
17	*not graded*	*not graded*	*not graded*	*not graded*	5	**a motor response in feet and hands using the trapezius, under sedation**
21	000/222 / 000/111	0, 2, 0, 1	9	6	4	ventilated, sedation withdrawn
26	111/333 / 222/555	1, 3, 2, 5	**33**	18	4	extubated the previous day; after the second exchange
28	222/333 / 333/555	2, 3, 3, 5	**39**	24	4	after the third exchange
30	222/333 / 333/555	2, 3, 3, 5	**39**	24	4	fourth exchange cancelled
39	2222/3333 / 3333/3333	2, 3, 3, 3	33	24	4	outpatient; unable to walk
46	2222/3333 / 3333/3333	2, 3, 3, 3	33	24	4	outpatient; right hand weak, unable to walk

Red marks a grade that is not compatible with the functional state described in the same note, and the sum score that grade distorts; grey marks a date on which strength was described but not graded. RU, right upper limb; LU, left upper limb; RL, right lower limb; LL, left lower limb; MRC, Medical Research Council. The record prints a group of identical digits for each limb, four digits on admission and at both outpatient reviews and three digits on the four dates between; because the digits within a limb are always identical, a per-limb grade is well defined on every date whatever the notation. The right limb is printed first, which is fixed by the note of hospital day 46 recording that the right hand was still weak on a day when the upper-limb entry reads 2222/3333. The reconstruction multiplies each per-limb grade by the three muscle groups per limb of the twelve-muscle sum score; it is a reconstruction and not a recorded value, and it is reported to locate the patient on the instruments of the clinical-instruments subsection of the Discussion rather than as a measurement. The left lower limb is graded 5 on three consecutive dates on which the same notes record a child unable to stand, so the three-limb column excludes it; the disability score, which needs no reconstruction, is 4 on admission, 5 while ventilated and 4 at both outpatient reviews.[19] The two dates marked in red carry descriptions of movement made under midazolam and morphine sedation; they are the first documented return of motor activity in the admission and they are not commensurable with a graded measurement, so they are not joined to the graded series.

Read that way, the trajectory is this. On admission every limb was graded 1, a reconstructed sum score of 12 of 60. On hospital day 21, the first day on which strength was formally graded after sedation was withdrawn, the right upper and right lower limbs were graded 0, the left upper 2 and the left lower 1: a sum score of 9, lower than on admission. By hospital day 28 the sum score had risen to 39, a gain of 30 points from the nadir, and it did not change again on hospital day 30. At the two outpatient reviews the reconstructed sum score reads 33, which is lower than the 39 recorded 11 days earlier. That fall is produced entirely by the left lower limb, graded 5 on hospital days 26, 28 and 30 and graded 3 at both outpatient reviews. A limb graded 5 is a limb of normal power, and the same notes record a child who could not stand; the grade of 5 is not compatible with the functional state described alongside it. Excluding the left lower limb and reconstructing the sum over the other three, the series reads 9, 6, 18, 24, 24, 24 and 24 on the seven graded dates, which rises to a plateau on hospital day 28 and does not move for the following 18 days.

Alongside the graded measurements the record contains two qualitative observations that must not be read as part of the same series. On hospital day 13 the note records a motor response in the child's feet, and on hospital day 17 a motor response in the feet and hands, although the child was recruiting the trapezius. Both were made under midazolam and morphine sedation and neither is a graded strength measurement. They are the first documented return of motor activity in the whole admission, and they fall 8 days after the immunoglobulin course ended and 9 to 11 days before the earliest possible date of the first plasma exchange. The treatment-attribution subsection of the Discussion returns to what follows from that.

The functional level is unambiguous whatever the strength grades do. Assigning the disability score used throughout the literature to what each note describes, the child was bedridden and unable to walk on admission, which is grade 4; he required assisted ventilation from hospital day 4, which is grade 5; he returned to grade 4 on extubation on hospital day 25; and he was still grade 4, unable to walk, at both outpatient reviews, the second of which falls 45 days after admission. On the instrument the disease's own literature uses as its primary outcome, the child's state at last follow-up is the state he was admitted in.

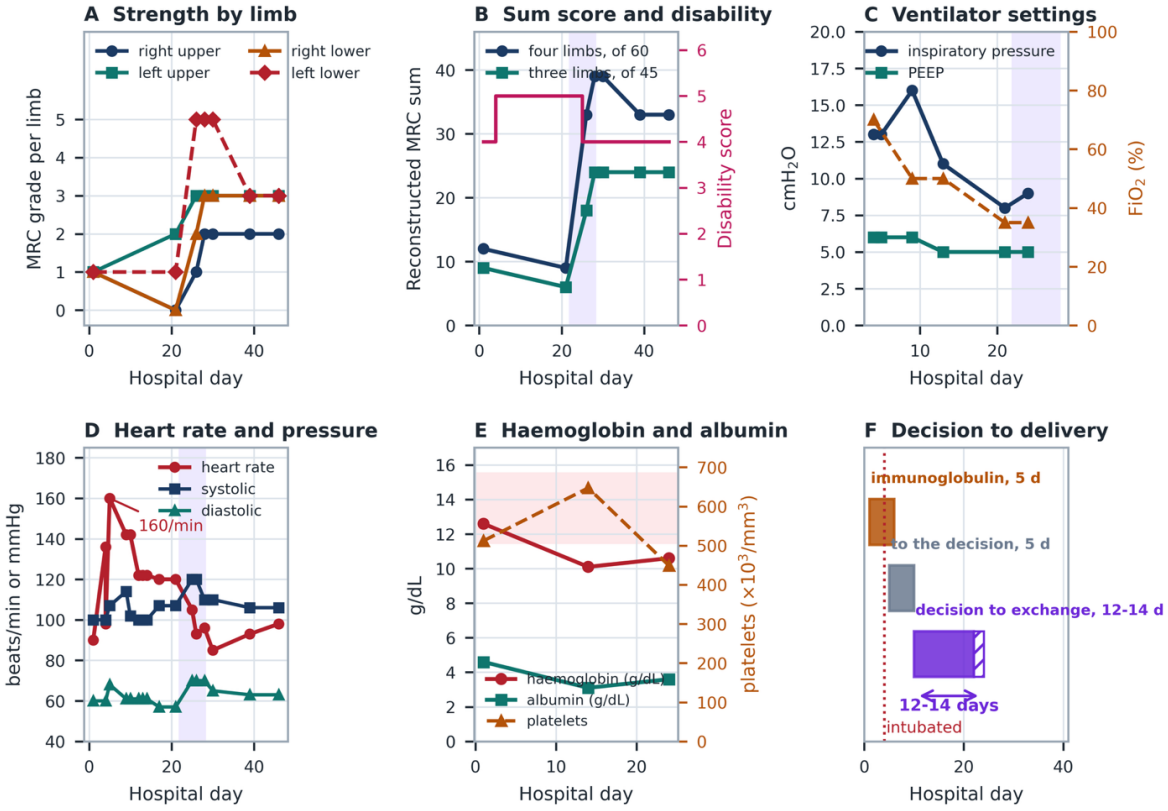

*Figure 4. Six panels drawn from the recorded data, with every plotted value traceable to Table 2, Table 3 or Table 4. **(A)** The per-limb Medical Research Council grade on each of the seven dates on which strength was graded; the left lower limb is drawn as a dashed line because a grade of 5 is not compatible with the functional state recorded on the same dates. **(B)** The reconstructed sum score over four limbs and over three, with the disability score on the right axis; the shaded band marks the interval within which the three exchanges were given. **(C)** Ventilator settings: inspiratory pressure, positive end-expiratory pressure and inspired oxygen fraction, with the interval of the exchanges shaded. The weaning of all three began before the first exchange. **(D)** Heart rate and blood pressure on every date on which they were recorded; the peak heart rate of 160 beats per minute falls on hospital day 5, the day on which two beta agonists and a beta blocker appear in the same plan. **(E)** Haemoglobin, albumin and the platelet count on the three dates on which a panel was taken. **(F)** The escalation interval: the immunoglobulin course, the interval from its completion to the written decision, and the interval from that decision to the first completed exchange, drawn as a band because the record does not date the exchange. The interval from decision to delivery is 12 to 14 days, longer than the 5-day course that preceded it.*

Course after extubation, and outcome

After extubation on hospital day 25 the child breathed spontaneously on a simple mask with no further desaturation. Blood pressure was 120/70 mmHg and the heart rate 105 beats per minute; over the following days the heart rate fell to 85 beats per minute. Feeding was changed to a low-lactose liquid diet at 6 × 250 mL and then to a high-protein diet. An oral intake trial on hospital day 34 was tolerated without nausea or vomiting, sensation was intact and bladder and bowel function were normal, and the child was discharged for outpatient follow-up and medical rehabilitation.

At the first outpatient review, hospital day 39, he was alert and cooperative with a Glasgow Coma Scale of 15, weighed 21.0 kg, and the limb weakness was described as improved although he still could not walk; physiological reflexes were reduced on both sides and pathological reflexes absent. At the second review, hospital day 46, the right hand remained weak, he still could not walk, and the recorded weight was 19.0 kg. That is a fall of 2.0 kg, 9.5% of the previous recorded weight, in the 7 days between the two visits. Against the ideal weight of 27.0 kg that the record itself derives, weight for height falls from 77.8% on admission to 70.4% at the second review, a fall of 7.4 percentage points. Either one of the two outpatient weights is wrong or the child lost almost a tenth of his body weight in a week; the record neither reconciles them nor responds to either possibility, and the plan written at the second review repeats the same four vitamins and the same zinc that were written at the first.

Patient and family perspective

The child's parents were present throughout the admission and were told at the outset that the weakness was expected to reach its worst before it improved and that breathing might have to be supported. What they found hardest, they said afterwards, was the period between being told that a further treatment was needed and that treatment being given: they understood that plasmapheresis had been decided upon and could not understand why, day after day, the answer to when it would happen was that the arrangements were still being made. They asked repeatedly whether the delay would cost their son the use of his hands. At the time of the last review recorded here the family's stated priorities were that he should be able to hold a pen and return to school, and rehabilitation was organised around those two goals. The parents gave written consent for this report and asked that the difficulty of arranging the exchange be described rather than omitted.

4. Discussion

What this record supports, and what it does not

It is worth stating at the outset what this case cannot be used to argue, because the obvious reading is the wrong one. The obvious reading is that a child failed immunoglobulin, received plasma exchange, and recovered; and that the case therefore supports plasma exchange as rescue therapy. The record forbids that reading, and it forbids it on its own evidence. The first documented return of motor activity falls on hospital day 13, which is 9 to 11 days BEFORE the earliest date on which the first exchange can have been performed. A

treatment cannot explain an improvement that began before it was given. The treatment-attribution subsection of the Discussion sets this out in full. What the record does support is different and, we think, more useful: an interval that can be measured, a set of instruments that could have been computed at the bedside and were not, and a list of measurements that were never made.

The instruments that were computable on the day of admission

Guillain-Barre syndrome is unusual among paediatric emergencies in the number of validated instruments it already possesses. They are set out in Table 5, scored item by item against what the record contains for each item. The striking result of that exercise is not that any single score is high or low; it is that every item of every instrument was available in the notes written on the day of admission, and that not one of the instruments appears anywhere in the record.

Table 5. The instruments this disease already possesses, scored item by item against what the source record contains.

Instrument and item	What the record contains	Points or verdict	Source and comparator
Brighton case definition			level 1 requires albuminocytological dissociation; level 2 does not[19]
Bilateral and flaccid weakness of the limbs	motor strength graded 1 in all four limbs on admission	met	
Decreased or absent deep tendon reflexes in weak limbs	physiological reflexes recorded as decreased on admission and the patellar and Achilles reflexes as absent from hospital day 14	met	
Monophasic course, interval from onset to nadir between 12 hours and 28 days	the graded nadir falls on hospital day 21, 23 to 24 days after the onset of weakness	met	
Cerebrospinal fluid white cell count below 50 per microlitre	1.0 per microlitre	met	
Cerebrospinal fluid protein above the laboratory normal value	28.6 mg/dL, that is 0.286 g/L, below the 0.45 g/L the case definition uses	**not met**	**this single item holds the case at level 2**
Electrophysiological findings consistent with the syndrome	absent F waves in four nerves and absent H reflexes bilaterally	met	
Absence of an alternative diagnosis for the weakness	thoracolumbar magnetic resonance imaging shows no compressive or intramedullary lesion	met	
Brighton level reached	6 of the 7 items met	**level 2**	among 248 patients, level 1 was fulfilled by 54.6%, level 2 by 45% and level 4 by 0.6%[25]
Erasmus respiratory insufficiency score			derived in 397 patients, validated in 191[26]
Days from onset of weakness to admission	3 days by the history of present illness, 4 by the chief complaint	2 or 1	the record supports both readings, so the total is carried as an interval
Facial or bulbar weakness at admission	absent: swallowing normal, no facial weakness recorded	0	bulbar dysfunction raised the odds of ventilation almost nineteenfold (18.67, 5.85–59.42) in one series[27]
MRC sum score at admission	every recorded muscle group graded 1; reconstructed sum 12	**4**	**the lowest band, odds ratio 30.9 (12.8–74.4) for ventilation against the highest band**[28]
Total	all three items recorded on hospital day 1	**5 or 6 of 7**	**computable at the bedside on the day of admission; not computed. The child was ventilated on hospital day 4**
Paediatric respiratory score (EGRIS-Kids)	age 8 years, no cranial nerve involvement, disability score 4 – all three items recorded	**computable**	a 9-point score developed in 421 children of whom 79 (19%) were ventilated; predicted risks 4 to 50%, area under the curve 0.71[6]
Modified Erasmus outcome score	*requires an MRC sum score at entry and at one week; the record contains no graded strength measurement between hospital days 1 and 21*	**not computable**	validated in 809 patients at entry and 671 at week 1, area under the curve above 0.7 in all regions[29]
Disability score, admission	bedridden, unable to walk	4	the score at nadir was the strongest predictor of walking at one month (odds ratio 0.43, 0.25–0.74)[6]
Disability score, last review	unable to walk, hospital day 46	**4**	**unchanged from admission on the instrument the literature uses as its primary outcome**
Neutrophil-to-lymphocyte ratio	computable from the admission differential count	6.83	in 61 patients the ratio did not correlate with change in MRC sum score but did correlate with the Erasmus outcome score (p = 0.006)[30]
Prognostic nutritional index	albumin 4.6 g/dL and absolute lymphocyte count 2,539/mm³	58.70	9.30 above the severity cut-off of 49.395 and 12.98 above the 26-week cut-off of 45.72 in a cohort of 252 patients; derived in adults and not validated in children[21]
Weight for height	21.0 kg against the record's own ideal weight of 27.0 kg	77.8%	**70.4% at the last review, a fall of 7.4 percentage points; the record neither re-weighs nor responds**
Electrodiagnostic subtype	**a motor demyelinating axonal lesion concluding acute motor axonal neuropathy; no latency, amplitude or conduction velocity is printed**	**unclassifiable**	**the two commonest criterion sets disagree in 360 of 1,137 studies (31.7%)[31], and subtype changes on serial testing in 37.8 to 44.7%[32]**

Red marks an item the record fails, a score that could not be computed, or a value that lies on the unfavourable side of a published threshold. Every item of every instrument in the upper half of the table was recorded in the notes written on the day of admission. MRC, Medical Research Council; EGRIS, Erasmus Guillain-Barre respiratory insufficiency score. The Erasmus respiratory score awards 0, 1 or 2 points for more than 7, 4 to 7 or 3 or fewer days from the onset of weakness to admission; 0 or 1 for the absence or presence of facial or bulbar weakness; and 0 to 4 across five bands of the MRC sum score, the lowest band being 20 or less.[26] The international validation of that score drew on 1,023 eligible patients aged six years or more, of whom 104 (10%) required mechanical ventilation within the first week from study entry, with an area under the curve of at least 0.80 in every validation subgroup.[28] A modified version predicts the same outcome with an area under the curve of 0.84 (0.80–0.88) in 1,133 of 1,500 patients.[33] The paediatric version is a nine-point score and its item weights are not reproduced here, so no total is asserted for it; what the table records is that all three of its items were available. The score reported for the MRC item does not depend on the reconstruction of Table 4: every muscle group recorded on admission was graded 1, so the sum score cannot exceed 12 under any convention and 12 falls in the lowest band. Two instruments in the lower half were derived in adults and are applied here for comparison and not for prediction.

The Erasmus Guillain-Barre respiratory insufficiency score is the one that mattered most here. It was derived in 397 patients and validated in a separate cohort of 191, and it uses exactly three items: the number of days from the onset of weakness to admission, the presence of facial or bulbar weakness at admission, and the Medical Research Council sum score at admission[26]. The first block of Table 5 records all three. This child's items are: 3 or 4 days, giving 2 or 1 points depending on which of the two onset dates the record's own text supports; no facial or bulbar weakness, giving 0; and a reconstructed sum score of 12, which falls in the lowest band and gives 4 points, the maximum the instrument awards for that item. The total is therefore 5 or 6 of a possible 7. In the international validation of that score, which drew on 1,023 eligible patients aged six years or more, 104 (10%) required mechanical ventilation within the first week from study entry and the area under the curve was at least 0.80 in every validation subgroup; the lowest band of the sum score item, the band this child occupied, carried an odds ratio for ventilation of 30.9 (95% confidence interval 12.8 to 74.4) against the highest band[28]. The child was ventilated on hospital day 4, that is within the first week from admission. A modified version of the same score, developed in 1,133 of 1,500 patients from the international cohort, predicts the same outcome with an area under the curve of 0.84 (0.80 to 0.88)[33], and independent validations have returned areas under the curve of 0.81 in 541 Bangladeshi patients[34] and 0.861 in 252 Chinese patients[35], although a Peruvian series of 177 patients found only moderate discrimination at 0.63[36].

Two qualifications belong here and we make them before drawing any conclusion. The first is that the reconstructed sum score is a reconstruction. The record does not print a twelve-muscle sum; it prints a grade per limb, and Table 4 explains the assumption under which those grades are expanded. If instead one simply notes that every muscle group recorded on admission was graded 1, the sum score cannot exceed 12 whatever the convention, and 12 falls in the same band, so the item score of 4 does not depend on the reconstruction. The second is that the score is not a paediatric instrument. A paediatric version exists: it was developed in a combined cohort of 421 children of whom 79 (19%) required mechanical ventilation, it is a nine-point score built on age, cranial nerve involvement and the disability score at admission, and it predicts risks of respiratory failure ranging from 4 to 50% with an area under the curve of 0.71[6]. All three of its items were recorded in this child's admission note – age 8 years, no cranial nerve involvement, disability score 4 – so that score too was computable at the bedside on hospital day 1. Neither score was computed. The point is not that a number would have changed the physiology; it is that a number would have changed the monitoring, and the respiratory arrest at 17:00 on hospital day 4 happened on a general ward round rather than in a planned setting.

The modified Erasmus outcome score, which predicts the inability to walk, could not be computed at all, and the reason is instructive. That score requires a sum score at entry and a second sum score one week later; its international validation used 809 patients at entry and 671 at week one, with areas under the curve above 0.7 in all regional subgroups[29], and a Bangladeshi validation of 506 patients returned 0.69 at entry and 0.78 at week one[37]. This record contains a graded strength measurement on admission and then no graded strength measurement at all until hospital day 21. The week-one item does not exist. An instrument that needs two measurements a week apart cannot be applied to a record that contains one measurement and then a gap of 20 days.

Two blood-based indices complete the instruments scored in Table 5, and both are computable from the admission full blood count and albumin. The neutrophil-to-lymphocyte ratio was 6.83 counting band forms with the segmented neutrophils and 6.75 counting segmented cells alone. In an Indonesian cross-sectional study of 61 patients with the syndrome, that ratio on the first day of hospitalisation showed no correlation with the change in Medical Research Council sum score at any interval up to discharge but did correlate significantly with the Erasmus outcome score ($p = 0.006$)[30], so the ratio is a marker of severity rather than of trajectory and we use it here only in that sense. The prognostic nutritional index, which combines albumin and the absolute lymphocyte count, recomputes to 58.70 at admission. In an observational cohort of 252 patients enrolled between 2019 and 2024, that index independently predicted disease severity (odds ratio 0.91, $p = 0.036$) and 26-week outcome (odds ratio 0.93, $p = 0.033$), with areas under the curve of 0.769 and 0.719 and cut-offs of 49.395 and 45.72 for severe disease and poor long-term outcome respectively[21]. This child's index sits 9.30 points above the first cut-off and 12.98 above the second, that is on the reassuring side of both, on the morning of a day on which every limb was graded 1. We report that as a negative result about the index in this patient, not as evidence against the index: it was derived in an adult cohort and has not been validated in children, and a single discordant patient is exactly what an area under the curve of 0.769 predicts will occur.

One prognostic marker that would have been informative here does not appear in this record and could not have: serum neurofilament light chain, a direct measure of axonal damage. Among 281 patients, a high concentration at four weeks was associated with the inability to walk unaided at 26 weeks (odds ratio 2.79, 95% confidence interval 1.72 to 4.90)[38]. It is named here not as a criticism, since the assay is not available in most units that treat this disease, but because it is the one item in this discussion that is not in the minimum data set of the minimum-data-set subsection of the Discussion; the distinction between what a unit cannot obtain and what it simply did not obtain is what makes the rest of that list worth making.

Diagnostic certainty, and the one item that keeps this case at level 2

The Brighton Collaboration case definition sets out levels of diagnostic certainty for the syndrome, and level 1 requires cerebrospinal fluid albuminocytological dissociation where level 2 does not[19]. Table 5 scores the seven items one by one. Six are satisfied without argument: bilateral flaccid limb weakness, reduced or absent tendon reflexes in the weak limbs, a monophasic course with a nadir 23 to 24 days after onset and therefore inside the 28-day window the definition allows, a cerebrospinal fluid cell count of 1.0 per microlitre, electrophysiological findings compatible with the syndrome, and no alternative diagnosis after magnetic resonance imaging of the thoracolumbar spine. The seventh is not. The protein was 28.6 mg/dL, that is 0.286 g/L, against a threshold of 0.45 g/L. The case therefore reaches level 2 and not level 1, and it does so on a single item.

That item is a function of when the sample was taken. In the international cohort, cerebrospinal fluid was examined in 1,231 of 1,500 patients (89%) and showed albuminocytological dissociation in 846 (70%), a proportion that rose with time from the onset of weakness: 57% at four days or less and 84% after four days[22]. This child's lumbar puncture was performed on hospital day 1, which is 3 to 4 days after the onset of weakness on the two readings the record supports, that is inside the four-day window in which

43% of patients in that cohort had no dissociation. A normal protein at that point is the expected finding in a substantial minority and does not argue against the diagnosis; the same study concludes explicitly that normal protein levels do not exclude it. What is harder to defend is that the puncture was never repeated. The identical protein, cell count and glucose are reproduced verbatim in the note of hospital day 4, which is the day the child arrested, and no second sample was taken at any point in the following 30 days of admission. A single early sample is a snapshot of a rising curve; the decision not to repeat it is what converts a level-2 case into a permanently level-2 case.

A second study bears directly on the interpretation. Among 94 patients from a cohort of 240 fulfilling the Asbury criteria, albuminocytological dissociation was more frequent in the demyelinating than in the axonal variants (60.6% against 26.2%, p = 0.002) and did not affect outcome[39]. If this child's electrodiagnostic label of acute motor axonal neuropathy is accepted, then a normal protein is the majority finding for his subtype, not the minority one. That is a reassuring reading, but it depends on the electrodiagnostic label, and the electrodiagnostic subsection of the Discussion shows that the label cannot be checked. Larger series put the frequency of dissociation between the two: in 156 patients in Saudi Arabia it was 69.1%[40]. Two independent diagnostic criteria sets applied to 43 patients returned sensitivities of 87.5% for the Dutch and Brighton criteria and 85.6% for the National Institute of Neurological Disorders and Stroke criteria, all lower than the sensitivity of the clinical criteria alone[41], and in 248 Mexican patients Brighton level 1 was fulfilled by 54.6%, level 2 by 45% and level 4 by 0.6%[25]. Roughly two patients in five reach level 2 rather than level 1 in an unselected series; this child is one of them.

One feature of this child's course deserves to be set against the case definition rather than passed over, because the definition names it as a feature that casts doubt on the diagnosis: marked and persistent asymmetry of weakness. On hospital day 21 the right upper and right lower limbs were graded 0 and the left upper and left lower 2 and 1, and the right hand was still the weaker limb at the last review 45 days after admission. That matters because the principal alternative diagnosis in a child with acute flaccid weakness is acute flaccid myelitis, and asymmetry is the feature that separates them: comparing 26 children with acute flaccid myelitis and 156 with this syndrome, asymmetric limb weakness occurred in 58% against 0% and sensory deficits in 0% against 40%, both p < 0.001[42]. This child had both sensory symptoms, which point to the syndrome, and asymmetry at nadir, which points away from it. The magnetic resonance study reproduced in Figure 3 excludes the myelitic alternative, which is why the asymmetry does not change the diagnosis; we report it because a reader given the strength grades of Table 4 and not this paragraph would be entitled to ask.

An electrodiagnostic report that contains no number

Nerve conduction studies were performed on hospital day 5, the day after intubation and 7 to 8 days after the onset of weakness. The report as it appears in the record describes a motor demyelinating axonal peripheral neurogenic lesion involving the bilateral median, ulnar, peroneal and tibial nerves, absent F waves in 4 nerves and absent H reflexes in 2, and concludes with a lesion consistent with acute motor axonal neuropathy, a variant of the syndrome. Three things follow.

First, the descriptive phrase is internally contradictory. A lesion cannot be both demyelinating and axonal under any published classification: the two are alternative categories of the same taxonomy. The original electrophysiological classification, applied to 369 patients studied within 15 days of onset, sorted them into five mutually exclusive groups – 69% demyelinating, 3% axonal, 3% inexcitable, 2% normal and 23% equivocal[43] – and the equivocal category exists precisely for studies that do not sort. Second, and more consequential, the report prints no numeric parameter at all: no distal motor latency, no compound muscle action potential amplitude, no conduction velocity, no F-wave latency. It therefore cannot be classified by any published criterion set, because every one of them is a set of numeric thresholds. This is not a pedantic objection. Of 1,137 nerve conduction studies from the international cohort, 777 (68.3%) were classified identically by the Hadden and the Rajabally criteria and 360 (31.7%) were classified differently[31]; and among 957 patients from 115 centres, reference values were derived locally in 49% of responding centres, taken from publications in 37% and from a combination in 15%[44]. A subtype label offered without the numbers that produced it, and without the criterion set used, cannot be independently re-adjudicated, and in this record it cannot be re-adjudicated at all.

Third, the study was performed early and was never repeated. Serial studies were available for 469 patients in the international cohort, and the electrodiagnostic subtype changed on serial testing in 37.8% of motor-sensory and motor patients under the Hadden criteria and 44.7% under the Rajabally criteria[32]; in a prospective study of 312 Bangladeshi patients, 44 to 59% had axonal and 9 to 42% demyelinating disease depending on the criteria used, and follow-up studies changed the classification in 11 to 26%[45]. A recent analysis states the conclusion in its title: an early electrodiagnostic study in this syndrome requires a second one for accurate electrodiagnosis[46]. The subtype matters because it is the strongest single predictor of the course. Among 57 classifiable patients, 74% of the demyelinating group but 38.46% of the axonal group were healthy or had only minor symptoms at three months[47]; among 224 critically ill children the axonal subtype accounted for 61.6% and mechanical ventilation was required in 54.9%[5]; and in 86 Mexican patients invasive ventilation was required in 39.5% overall and in 72.7% of those with the acute motor and sensory axonal variant[48]. In children, prolonged ulnar F-wave latencies in the axonal subtype were significantly associated with delayed walking in a prospective series of 57 (p = 0.02)[49]; among 772 cases in a large South American outbreak the case fatality rate was 4.3%, 32.2% of survivors reported sequelae, and axonal subtypes accounted for 75.6% of those in whom nerve conduction studies were performed[50]; among 595 patients in eastern China a pure motor phenotype occurred in 158 of 340 (46%) and 103 of 340 (30%) had a complication during the admission[51]; and among 65 children the short-term prognosis of demyelinating patients with A waves was worse than of those without (odds ratio 5.844, 95% confidence interval 1.118 to 30.553, p = 0.036)[52]. Every one of those findings is a numeric one; none of them can be applied to a report that contains no numbers.

An antecedent illness that was never investigated

The history is the textbook one and the investigation is absent. Diarrhoea for 4 days, beginning 5 to 6 days before the weakness, is the antecedent most strongly associated with the axonal form. In the original electrophysiological classification, 6 of 10 patients (60%) with axonal neurophysiology reported a preceding diarrhoeal illness against 71 of 359 (20%) in the other groups[43]. Serological evidence of recent infection in the international cohort, among 768 patients with biosamples, identified Campylobacter jejuni in 228 (30%), Mycoplasma

pneumoniae in 77 (10%), cytomegalovirus in 30 (4%) and hepatitis E in 23 (3%)[53], and a multinational case-control study in areas of arbovirus transmission found evidence of recent infection in 27 of 49 patients (55%), most often Campylobacter jejuni in 15 (31%)[54]. The mechanism is established experimentally: infection of interleukin-10-deficient mice with a syndrome-associated but not a colitis-associated Campylobacter isolate produced sciatic nerve inflammation and an abnormal gait, dependent on interleukin 4 and sialoadhesin[55], and more than a thousand autoreactive single T-cell clones targeting peripheral nerve myelin antigens have now been characterised in patients with the demyelinating variant[56].

No stool culture was sent. No anti-ganglioside antibody was requested. Both matter more than they used to. Among 1,413 patients in the international cohort, 1,309 (92.6%) were positive for at least one anti-glycolipid or anti-glycolipid-complex antibody against 1,061 controls[57], and in an earlier screen of 100 patients none reacted against the nodal or paranodal proteins tested while 61 (61%) were positive for at least one anti-ganglioside antibody[58]. An antibody result would have supported or undermined the axonal label that the numberless nerve conduction report cannot support on its own, and it would have done so on a single tube of serum drawn on the day of admission. The cost argument does not obviously survive contact with the arithmetic either: in one African setting the mean direct medical cost of an episode of this syndrome was 267.70 US dollars against 8.96 dollars for an episode of diarrhoea[59], so the investigations that distinguish them are small beside the illness they characterise.

The record's own coronavirus thread is left equally unresolved. A household contact and 2 vaccine doses are recorded, and a SARS-CoV-2 antibody titre of 250 U/mL is reported with no assay, no reference interval and no statement of whether the target is the spike or the nucleocapsid protein. In a vaccinated child only an anti-nucleocapsid result can separate past infection from vaccination, so the titre as printed is uninterpretable. The temporal argument is clearer: the last vaccine dose was given in March 2022, roughly nine months before the onset of weakness, and the risk windows used in the pharmacoepidemiological literature are measured in weeks. A multinational self-controlled case series across 20 global sites found the risk raised after ChAdOx1-S vaccination (relative incidence 3.10, 95% confidence interval 1.12 to 8.62) and after SARS-CoV-2 infection in 489 cases (relative incidence 3.35, 95% confidence interval 1.83 to 6.11)[20], and in the international cohort 49 patients were included of whom eight (16%) had a confirmed and three (6%) a probable SARS-CoV-2 infection[60]. In 19 children studied during the pandemic, 11 had positive coronavirus serology and post-coronavirus cases presented with variant rather than classical forms ($p = 0.03$)[61]. On the evidence in this record neither infection nor vaccination can be implicated and neither can be excluded, and the diarrhoeal antecedent that could have been characterised was not.

Second-line therapy after immunoglobulin: what the evidence actually supports

The present admission is set out in Table 6 beside the recent series that bear on it, and the comparison is deliberately made on three separate axes: what plasma exchange achieves in children, what it costs in adverse events, and how long ventilated children stay ventilated. The efficacy evidence in children is now respectable and comes largely from constrained settings. In 36 children with severe disease receiving 122 sessions, upper-extremity power rose by 2.0 ± 1.1 points (95% confidence interval 1.6 to 2.4, $p < 0.001$)[13]; among 23 children with acute transverse myelitis, this syndrome or acute flaccid myelitis, 17 (74%) improved significantly by the end of treatment[14]; and in 15 children with neurological disease treated by exchange, 12 improved and three did not, with 12 admitted to intensive care and 10 ventilated[62]. In an adult series of 43 patients receiving 246 exchanges, the disability score fell in 14 of 20 patients with this syndrome[63], and in 99 patients with autoimmune neurological disorders the median modified Rankin score improved from 5 (interquartile range 4 to 5) before exchange to 3 (2 to 4) afterwards[64].

Table 6. The present case set against the recent series that bear on it, grouped as efficacy of exchange in children, its adverse-event burden, the course of ventilated children, and the two published measurements of delay.

Series, setting and size	Design	What it reports	This patient
36 children with severe disease, Syria[13]	retrospective cohort	122 exchange sessions; upper-limb power rose from 1.7 ± 1.1 to 3.7 ± 0.9, an increase of 2.0 ± 1.1 (95% CI 1.6–2.4, $p < 0.001$)	3 sessions; upper-limb grade rose from 0 and 2 to 2 and 3 between hospital days 21 and 28
23 children with myelitis or this syndrome, France[14]	retrospective cohort	17 of 23 (74%) improved significantly by the end of treatment	improved on the strength grades, unchanged on the disability score
36 children with autoimmune encephalitis, Vietnam[65]	case series	54.5% needed antiepileptic drugs at discharge, 24.2% had seizures at one year	a demonstration that exchange is deliverable in a constrained system; the arranging interval is not reported
15 children with neurological disease[62]	retrospective cohort	12 of 15 improved; 12 admitted to intensive care and 10 ventilated	ventilated for 21 days
43 adults, 246 exchanges, Montenegro[63]	retrospective cohort	the disability score fell in 14 of 20 patients with this syndrome; 14 patients had an adverse event	the disability score did not fall
53 children, 378 procedures[66]	retrospective cohort	complications in 81% of patients and 23% of procedures; severe complications in 11% of patients; no deaths	*no adverse event recorded; no ionised calcium measured*
78 children, 731 procedures[67]	retrospective cohort	72 of 78 (92%) had at least one of 311 adverse events	*the record documents no adverse event and no monitoring for one*
25 children, 128 sessions[68]	retrospective cohort	128 sessions in 25 patients of mean age 59.6 ± 11.7 months	3 sessions in one child of 8 years
185 patients, 1,895 sessions[69]	retrospective cohort	at least one adverse effect in 805 sessions (42.5%), affecting 171 of 185 patients (92.4%)	*no adverse effect recorded for any of the three sessions*
99 children, 672 centrifugal procedures[70]	retrospective cohort	adverse events in 34 procedures (5%), most often allergic reaction to the replacement fluid (2.24%)	the replacement fluid is recorded for one session only

Series, setting and size	Design	What it reports	This patient
39 children, 172 sessions[71]	retrospective cohort	no adverse event during any procedure; one session (2.6%) ended early for insufficient vascular access	**a fourth session cancelled outright because the central dialysis line was blocked**
154 critically ill children, 486 sessions[72]	retrospective cohort	survival 72.7%; adverse events in 68 sessions (13.9%)	survived
84 critically ill children, 463 sessions[73]	retrospective cohort	survival 50%, lowest at 41.5% in the lowest indication category	survived
421 children, Europe[6]	retrospective cohort	79 of 421 (19%) required mechanical ventilation; one died	**ventilated on hospital day 4**
224 critically ill children[5]	retrospective cohort	axonal subtype 61.6%; mechanical ventilation 54.9%; tracheostomy 24.2%	axonal label, ventilated, no tracheostomy
102 children, Ethiopia[7]	retrospective cohort	13 of 102 (12.7%) died; respiratory failure the only determinant of mortality (adjusted odds ratio 11.40, 1.818–71.52, p = 0.009)	survived
886 adults in 47 intensive care units, France[74]	retrospective cohort	prolonged weaning in 64% of 513 patients with this syndrome against 35% of 373 with myasthenia gravis	21 days of ventilation, weaned without tracheostomy
51 children followed a median of 6 years 4 months[75]	prospective cohort	sequelae in 67% (95% CI 53–78)	**follow-up ends at hospital day 46; the three-month and six-month outcomes the literature uses do not exist in this record**
40 children[76]	prospective cohort	82.5% male; demyelinating phenotype 55%; 100% independently ambulant at 6 months	**unable to walk at hospital day 46**
142 children with classic disease[77]	retrospective cohort	18 of 142 (12.68%) ventilated; 134 (94.37%) walking independently at 6 months	**ventilated; not walking at last review**
100 adults, single centre[17]	retrospective cohort	50 treated within 24 h of arrival and 50 later; 41 of the 50 delayed had no clear-cut diagnosis at first	the diagnosis was made at once; the DELIVERY of second-line therapy took 12 to 14 days
21 children[18]	retrospective cohort	40 misdiagnoses among 17 patients; median diagnostic delay 9 days (interquartile range 6–17)	**no diagnostic delay; an escalation interval of 12 to 14 days that no published series reports**

Red marks a row in which this patient's course or record falls short of the comparator; grey marks a comparator quantity the record does not contain. CI, confidence interval. The comparators are cohorts and this is one patient, so the final column locates the case rather than testing it; no row should be read as a comparison of outcomes. The last two rows are the only published measurements of delay we could find, and both measure the interval before the FIRST treatment, which is a diagnostic interval. The interval this report describes begins after the diagnosis has been made and the escalation decided, and we found no series that reports it. The three sessions given here were preceded by a completed immunoglobulin course, so this patient belongs to the group covered by the systematic review of immunoglobulin-unresponsive disease – 422 patients in 37 articles, of which 33 were case reports or series, with no superiority of either repeat immunoglobulin or exchange on any clinical outcome.[12]

The safety evidence is more mixed than the efficacy evidence, and it is reported in Table 6 as it stands rather than selectively. Among 53 children undergoing 378 procedures, complications occurred in 81% of patients and 23% of procedures, with severe complications in 11% of patients and no deaths66; among 78 children undergoing 731 procedures, 72 (92%) had at least one of 311 adverse events67; among 81 children undergoing 360 procedures, adverse events occurred in 50% of procedures using fresh frozen plasma against 39.5% without it78. At the other end, adverse events were observed in only 34 (5%) of 672 centrifugal procedures in 99 children70, and in 39 children undergoing 172 sessions no patient had an adverse event during the procedure, one session (2.6%) being ended prematurely for insufficient vascular access71. In critically ill children the survival figures are what they are: 72.7% among 154 children undergoing 486 sessions72 and 50% among 84 children undergoing 463 sessions, the lowest survival of 41.5% falling in the lowest indication category73. This child had three sessions, no recorded adverse event, and no calcium measurement around any of them, which is a gap rather than a reassurance: citrate anticoagulation binds ionised calcium, and a metabolic alkalosis with a bicarbonate of 34.4 mmol/L and a base excess of 10.6 mmol/L appeared on hospital day 24, the last day on which the first exchange can have been performed. Citrate is metabolised to bicarbonate, and a post-exchange metabolic alkalosis is a recognised consequence. We cannot attribute it, because the record contains neither an ionised calcium nor the date of the exchange, but a single blood gas is a thin basis for either conclusion, and it is the second reason calcium belongs in the minimum data set. The alternative technique avoids the question: among 46 patients receiving 385 membrane exchanges without any anticoagulation the complication rate was 1.3%, with device coagulation in 2.1% of patients and no deaths79. Which technique was used here is not recorded either.

The question this child's course actually poses – what to do when the first immunoglobulin course does not work – has no good answer in the literature. The randomised trial of a second immunoglobulin dose in patients with a poor prognosis returned an adjusted common odds ratio of 1.4 (95% confidence interval 0.6 to 3.3) for improvement at four weeks and did not support the practice11; the systematic review of the alternative, covering 422 patients in 37 articles of which 33 were case reports or series, found no superiority of either option on any clinical outcome12. The head-to-head randomised comparison of the two first-line treatments, in 81 adults assigned 2:1, favoured immunoglobulin at one and three months and found no difference at one year8, and a retrospective comparison of 142 cases found similar 28-day mortality with a trend towards lower mortality after plasmapheresis (odds ratio 0.78, 95% confidence interval

0.62 to 0.97, p = 0.062)80. Nor is more immunotherapy always better: among 188 patients with mild disease, 148 (79%) received immunoglobulin and 40 (21%) supportive care, and immunoglobulin was not associated with a lower disability score at four weeks (adjusted odds ratio 1.62, 95% confidence interval 0.63 to 4.13)81. A randomised comparison of immunoadsorption with plasma exchange in 32 patients with neurological autoimmune disease found comparable response82, and treatment-related fluctuation – the phenomenon that most often prompts a second course – occurred in only 7 of 124 cases (5.6%)83. Against that background, and against the rows of Table 6 that report what exchange achieves in children, the decision taken here – to move to exchange after a completed course without recovery – is defensible and conventional. It is the delivery of that decision that this report is about, and the last two rows of Table 6 are the only published measurements of delay we could find.

The escalation interval as a measurable, modifiable quantity

The interval this record documents is 12 to 14 days between a decision written in the notes and the treatment that decision names. It is longer than the immunoglobulin course that preceded it (5 days), a comparison drawn as panel F of Figure 4, longer than the interval from the onset of weakness to admission (3 to 4 days), and longer than the interval from admission to respiratory failure (3 days). Set against the whole illness, the child spent 24 to 27 days between the onset of weakness and the first exchange, of which the escalation interval accounts for roughly half.

What makes this quantity worth naming separately is that it is not diagnostic delay and the literature has only measured diagnostic delay. Of 100 adults at a single centre, 50 were treated within 24 hours of arrival and 50 later, and 41 of the 50 delayed patients had not been given a clear-cut diagnosis, the alternatives considered being most often orthopaedic (11 of 41), vascular (7 of 41) or nutritional (6 of 41)[17]; among 21 children there were 40 misdiagnoses among 17 patients and a median diagnostic delay of 9 days (interquartile range 6 to 17)[18]. Both are intervals during which nobody had decided what to do. Here the decision was made, written down and repeated in six consecutive ward notes, and the treatment still did not arrive. The record's own explanation is administrative and is stated in one clause on hospital day 17: the anaesthesiology service had been contacted and the referral had not yet been connected. The fourth exchange was then cancelled for a technical reason of exactly the same class: the central dialysis line access was blocked. Neither reason is clinical.

The scattered evidence that exists suggests this is not a local peculiarity. A survey of 328 responses from 238 hospitals in 60 countries found that only 77 paediatric intensive care units (52.7%) had a paediatric intensivist or anaesthetist available around the clock and 71 (49.7%) had nurse-to-patient ratios below one to two[15]. Where the treatment depends on a machine and an operator belonging to another department, those two numbers describe the bottleneck directly. Among 45,515 hospitalisations for this syndrome, Black patients were less likely to receive plasmapheresis (odds ratio 0.75, 95% confidence interval 0.59 to 0.95) while each higher quartile of postcode income reduced inpatient mortality (odds ratio 0.80)[16] – an access gradient measured in a high-income system. In Kenya, among 207 patients, 25.7% had respiratory complications and 35.4% required intensive care, and immunoglobulin use reduced mortality by 75% (adjusted odds ratio 0.25, 95% confidence interval 0.08 to 0.85)[84]. In Vietnam, 36 children with severe autoimmune encephalitis were treated with plasma exchange in a single centre[65], and in Syria 36 children with severe disease received 122 sessions[13]: both appear in Table 6, both are demonstrations that the procedure can be delivered in constrained systems, and neither reports how long it took to arrange. In 102 Ethiopian children respiratory failure was the only determinant of mortality (adjusted odds ratio 11.40, 95% confidence interval 1.818 to 71.52)[7], which is the outcome the escalation interval most plausibly bears on. The measurement we propose is simple and free: record the date on which second-line therapy is decided and the date on which the first session is delivered, and report the difference. It is item 10 of the minimum data set below, and it is the violet band of Figure 1.

One further consequence of the delay is worth stating because it is favourable and we would not want it omitted. Whatever the administrative interval cost, the exchange was still delivered 24 to 27 days after the onset of weakness, which is inside four weeks by 1 to 4 days on the two readings of the onset date that the record supports. The margin is narrow and we state it as an interval rather than as a point precisely because a single further week of arranging would have removed it entirely.

Respiratory failure, predicted and unmonitored

Respiratory failure occurred on hospital day 4 at 17:00, with saturation falling to 75% and the respiratory rate to 12 breaths per minute. A falling respiratory rate in a child who is desaturating is not respiratory distress; it is respiratory muscle failure, and the record's own description of copious secretions the child could not clear is the bulbar and expiratory component of the same process. The event was predictable in the actuarial sense set out in the clinical-instruments subsection of the Discussion, and the rates in comparable series make the point: 19% of 421 children[6], 12.68% of 142 children with classic disease[77], 11.1% of 27 children[85], 19.7% of 46 North Indian children[86], 24% of 25 children studied with sympathetic skin responses[87], and 54.9% of 224 critically ill children[5]. In adults, 31 of 252 (12.3%)[35], 49 of 153 (32%)[88], 29% of 303[89] and 17% of one series in which dysautonomic events occurred in 36% and bulbar dysfunction raised the odds of ventilation almost nineteenfold (odds ratio 18.67, 95% confidence interval 5.85 to 59.42)[27]. Patients presenting very early are at higher risk still: among 100 patients, those with very early disease more often needed ventilation (50% against 22.4%, p < 0.01)[90].

What the record does not contain is any measurement of respiratory function before the arrest. There is no vital capacity, no negative inspiratory force, no single-breath count and no recorded assessment of neck flexion or of cough strength on any day. The only respiratory variables in the notes are the respiratory rate and, after the event, the oxygen saturation, and the chest radiograph of Figure 2 was already an hour and a quarter old when the arrest occurred. Deltoid strength of 2 or less carried an odds ratio of 7.1 (1.6 to 31.1) and autonomic dysfunction an odds ratio of 6.6 (2.0 to 22.0) for invasive ventilation in 153 patients[88]; this child had a deltoid graded 1 on admission and a heart rate that reached 160 beats per minute within a day of intubation. Both variables were in the notes. Neither was used as a trigger.

Ventilation then lasted 21 days, which is long but not exceptional. In a nationwide multicentre study of 886 patients in 47 French intensive care units – 513 with this syndrome and 373 with myasthenia gravis – prolonged weaning was commoner in the syndrome group (64% against 35%, p <

0.001)[74]. Among 303 patients, severe axonal degeneration of the median nerve was an independent risk factor for prolonged invasive ventilation (odds ratio 4.9, 95% confidence interval 1.1 to 21.5)[89], which is exactly the variable this child's numberless nerve conduction report cannot supply. No tracheostomy was performed here; in the largest paediatric critical-care series 24.2% of children required one[5].

Dysautonomia, treated in two directions on the same day

The autonomic record of this admission consists of a heart rate and a blood pressure. The heart rate rose from 90 beats per minute on admission to 136 on hospital day 4 and 160 on hospital day 5, then fell through 142, 122 and 120 to 85 by hospital day 30. Systolic pressure ranged from 100 to 120 mmHg and diastolic from 57 to 70 mmHg, and panel D of Figure 4 plots both against the heart rate. Dysautonomia in this syndrome is common and consequential: among 214 patients 51 (31%) had it, most often hypertension in 39 (84.8%), hypotension in 35 (76.1%) and tachycardia in 35 (76.1%)[91]; among 68 patients 79% had abnormalities on laboratory autonomic testing while only 25% had an abnormal symptom score[92]; among 17 patients tested after discharge, 6 (35%) had abnormal findings in at least one component of a standard autonomic battery and 3 (18%) met criteria for definite generalised dysautonomia[93]. In children the figures are similar: of 25 children, 18 (72%) had an abnormal sympathetic skin response and 17 (66.7%) had autonomic dysfunction[87], and autonomic profiles differ by electrodiagnostic subtype in critically ill children[5]. Cardiovascular autonomic function does recover: in six patients the average RR interval and baroreflex sensitivity were significantly worse than in controls at admission and had normalised by six weeks[94].

The treatment recorded on hospital day 5 is the reason this section exists. That day's plan lists a dobutamine infusion at 5 µg/kg/minute, an adrenaline infusion at 0.05 µg/kg/minute and oral propranolol 4 mg three times a day, on a day when the blood pressure was 107/68 mmHg and the heart rate 160 beats per minute. Two beta agonists and a non-selective beta blocker appear together in one plan. The record states neither the sequence nor the indication for any of the three, and the propranolol dose, at 0.57 mg/kg/day, is below the range customarily used. We do not assert that they were co-administered; we observe that the record cannot distinguish co-administration from sequential use, that a single blood pressure and a single heart rate are the only haemodynamic data for that day, and that no electrocardiogram, rhythm strip or corrected QT interval appears anywhere in the admission. Autonomic instability is the mechanism by which this disease kills patients who do not die of respiratory failure, and the record of it here consists of two numbers a day.

Sedation without analgesia, and a pain score that does not exist

The admission examination records hyperaesthesia in all four extremities and cramping pain in both legs, and pain is one of the two symptoms most reliably reported by survivors of this illness: among 24 adults followed for three years the predominant residual symptoms were fatigue (100%), neuropathic pain (70.8%), joint pain (54.2%) and autonomic dysfunction (50%)[95]. The prescription that ran for the greater part of this child's ventilated period was midazolam at 4.0 µg/kg/minute with morphine at 5.0 µg/kg/hour. Recomputed, that is 0.24 mg/kg/hour of midazolam – twice the upper end of the range customarily quoted for continuous sedation in children – beside 0.105 mg/hour of morphine, which is between 2 and 8 times below the lower bound of the range customarily used for a continuous paediatric infusion. The ratio is the finding: a child in whom the record itself documents pain was deeply sedated and barely analgesed, and no pain assessment of any kind appears on any day.

This is not a marginal point in a disease of this kind. A sedated, ventilated, paralysed child cannot report pain, and the instruments that exist for exactly that situation are behavioural. Anxiety and depression after the illness are common – among 111 patients with complete data, 45% had anxiety at week 13 falling to 36% at week 26 and 53% depression at week 13 falling to 41% at week 26[96] – and untreated pain during the acute phase is one of the modifiable contributors. The instrument literature has moved on to disability measures with good psychometric properties: in 1,226 patients the inflammatory Rasch-built overall disability scale showed adequate internal consistency (person separation index 0.95, alpha 0.98) but poor unidimensionality (12.1%, confidence interval 10.7 to 13.5%) with 10.1% floor and 9.4% ceiling effects[97]. A pain score is simpler than any of these and costs nothing.

Nutrition: an equation evaluated at the wrong weight, and two kilograms

This child was wasted on admission. Weight for height was 77.8% of the ideal weight the record itself derives, the body mass index recomputes to 12.05 kg/m^2, and the record labels the state malnutrition in the physical examination and underweight on the nutritional assessment form, without a z-score or a centile anywhere. Malnutrition on admission to paediatric intensive care is common: in 41 Latin American units, underweight was present in 42 of 88 children (47.7%) under 24 months and 14 of 103 (13.6%) aged 24 months or more[98]. It matters here for a specific reason. The prognostic nutritional index that the clinical-instruments subsection of the Discussion recomputes was developed precisely because, in low-resource settings, malnutrition and limited treatment worsen the prognosis of this syndrome[21].

The record's own energy calculation contains a substitution that changes the answer by 11.8%. The Schofield equation was evaluated at the ideal weight of 27.0 kg rather than the actual weight of 21.0 kg, returning 1115.826 kcal where the actual weight returns 998.286 kcal, a difference of 117.5 kcal that carries through the stress factor to a difference of 141.0 kcal in the estimated expenditure. Either choice is defensible for a wasted child; not saying which was made is not. Predicted equations are in any case approximate in hospitalised children: among 109 children undergoing indirect calorimetry the Schofield equation had the highest predictive accuracy at 47.7%, and in severe malnutrition the best-performing alternative reached only 34.4%[24]. The delivered energy cannot be compared with either estimate, because the record states the volume of the liquid diet and never states its energy density.

Delivery is where paediatric intensive care most often fails, and the comparators are unflattering to everybody. Of 533 unit patients, 402 (75.4%) were fed within 48 hours and 95 of 118 (80.5%) received two thirds of caloric needs after 7 days[99]; among 75 critically ill children there were 41 interruptions to enteral nutrition affecting 37.3% of patients[100]; and among 1,261 critically ill children 199 (15.8%) were undernourished and at risk, with probable refeeding syndrome in 93, an overall incidence of 7.4% and 46.7% among those at risk[101]. Getting it right is worth something: guideline-based enteral nutrition significantly preserved muscle mass after the first week in 63 critically ill children ($p < 0.01$)[102], and early enteral nutrition was associated with lower unit mortality in 151 intubated children with acute

respiratory distress syndrome (adjusted odds ratio 0.071, 95% confidence interval 0.009 to 0.542, p = 0.011)[103]. In a child with a neuromuscular disease whose recovery is a muscle-rebuilding problem, that last finding is the relevant one.

The outpatient weights are the part of this record we find hardest to explain. The weight was 21.0 kg on hospital day 39 and 19.0 kg on hospital day 46, 7 days later: a fall of 2.0 kg, 9.5% of body weight, in a week, in a child who was eating and being rehabilitated. Weight for height falls from 77.8% to 70.4%, a fall of 7.4 percentage points against the record's own ideal weight. Only two readings are available: either one of the two measurements is wrong, or the loss is real. The record does neither of the two things that would settle it. It does not re-weigh, and it does not respond: the plan written at the second review repeats the four vitamins and the zinc written at the first, with no dietary referral, no energy prescription and no comment. Recovery from this illness is slow – among 51 children followed for a median of 6 years 4 months the sequelae rate was 67% (95% confidence interval 53 to 78)[75] – and it is being attempted here on an unmeasured intake.

Why the recovery cannot be attributed to plasma exchange

The temptation to read this case as a demonstration of rescue therapy should be resisted, and the reason is a date. The first documented return of motor activity is the note of hospital day 13, which records a motor response in the feet; the note of hospital day 17 records a response in the feet and hands. The earliest date on which the first exchange can have been performed is hospital day 22. The first observation of recovery therefore precedes the first exchange by 9 to 11 days. Two further facts point the same way. The ventilator was already being weaned before the exchange: the inspiratory pressure fell from 16 to 11 cmH_2O on hospital day 13 and the oxygen fraction from 50% to 35% by hospital day 21, all before the first session, as is plotted in Figure 4 at panel C. And the natural history of the disease is recovery: among 40 children, 100% achieved independent ambulation at six months[76]; of 142 children, 134 (94.37%) could walk independently at six months[77]; and every child in a nationwide Japanese survey walked within one year[2]. A single-patient time series cannot separate a treatment effect from the natural history of a self-limiting disease, and here it cannot even establish the ordering.

There is a published framing that fits this course better than treatment failure, and we prefer it. Among 36 children with this syndrome, 18 (50%) were rapid responders to immunoglobulin and 18 (50%) slow responders, with a time to initial response of 5 days (interquartile range 3 to 6.25) against 10.5 days (8.75 to 15) respectively[104]. This child's first documented motor response falls 8 days after the immunoglobulin course ended and 12 days after it began, which places him among the slow responders rather than among the non-responders. Half of that cohort responded slowly, and did so without a second treatment. The distinction is not academic: a child labelled a non-responder is escalated and a child labelled a slow responder is watched. Nothing in this record allows the two to be told apart at the moment the decision was made, which is the strongest argument we can make for the weekly graded strength measurement that is item 1 of the minimum data set.

There is a second, subtler reason to be careful, and it concerns what a qualitative note can and cannot be compared with. The observations of hospital days 13 and 17 are descriptions of movement made under sedation. The measurement of hospital day 21 is a graded strength assessment made off sedation, and it reads 9 on the reconstructed sum score, which is LOWER than the 12 recorded on admission. Those two kinds of observation are not commensurable and cannot be assembled into a single trajectory; a note that a foot moved is not a Medical Research Council grade. We report both, keep them apart, and decline to draw a curve through them. The graded series alone, excluding the left lower limb for the reason given in the motor-strength subsection of the Results, reads 9, 6, 18, 24, 24, 24 and 24: a rise between hospital days 21 and 28, which is the period during which the exchanges were given, followed by 18 days with no change at all. The plateau is as much a part of the record as the rise, and any reading that credits the exchanges with the rise must also account for the plateau that followed while the child was still on the same rehabilitation.

The functional instrument that the disease's own literature uses as its primary endpoint makes the same point without any reconstruction. The disability score was 4 on admission, 5 while ventilated, and 4 at both outpatient reviews, the second of which falls 45 days after admission. On that scale the child ended the recorded period exactly where he began it. The literature would predict better: the disability score at nadir was the strongest predictor of recovery to independent walking at one month in the paediatric cohort (odds ratio 0.43, 95% confidence interval 0.25 to 0.74)[6], and in 44 children a high three-month disability score was the significant predictor of sequelae[105]. This child's three-month score is not in this record, and that is the honest end of the story: the outcome that would settle the question was never measured, because follow-up stopped at day 45.

Inconsistencies in the source record, declared

A case report is a reading of a record, and a reading is only as good as its account of where the record disagrees with itself. We found 18 such disagreements and Table 1 sets out every one of them, with the two places at which the record makes each statement and the resolution adopted here. The resolution adopted for each is given in the final column of Table 1, and the two places at which the record makes each statement are listed in Table 1 beside it. They fall into four classes. The first is arithmetical: an age given three ways, a weight for age given two ways, and an immunoglobulin day number one behind the calendar. The second is notational: a serum calcium printed in millimoles three times when the values are milligrams per decilitre, a methylcobalamin dose printed in milligrams when µg are meant, an antibiotic strength that does not say which component it refers to, and a manual muscle test whose digit count changes three times. The third is evidential: a nerve conduction report that is internally contradictory, a cerebrospinal fluid result reproduced verbatim on a later date, a blood gas labelled venous and reported as arterial, and a limb graded 5 in a child who could not stand. The fourth is temporal: no admission date, no date for the magnetic resonance study, and no date for the first plasma exchange. We have not smoothed any of these. Where a disagreement changes a number the paper depends on, that number is carried as an interval, and every such interval is stated as one.

Three of the eighteen are worth a sentence each because they show how a small notational problem becomes a substantive one. The calcium is the clearest: had the record's unit been taken at face value, every value would have been read as extreme hypercalcaemia and the citrate question raised in the second-line-therapy subsection of the Discussion would have been answered in exactly the wrong direction. The left lower limb graded 5 is the second: a single implausible grade shifts the reconstructed sum score by 6 points and turns a plateau into an apparent decline, which is

why the reconstruction excludes that limb and says so rather than averaging it away. The third is the missing date of the first exchange, which is the central quantity of this report: because the record brackets it between two notes rather than dating it, every interval built on it is a two-day interval and not a point, and we have written all of them that way.

A minimum data set for a child with Guillain-Barre syndrome

The purpose of an audit of this kind is not to find fault with a record written in conditions we did not share; it is to name what should be captured next time. The 10 items below are derived from this single admission. Each names a measurement, the moment at which it must be obtained, and the decision it would have changed. None requires equipment beyond what the unit already had: a stethoscope, a manometer or a peak-flow meter, a scale, a laboratory that already runs a full blood count and an albumin, an electrocardiograph, and a clock. Nine of the ten are free.

1. A Medical Research Council sum score across twelve muscle groups, on admission and then at least weekly. The record contains a per-limb grade on admission and then no graded measurement for 20 days. Without a week-one score the modified Erasmus outcome score cannot be computed at all[29,37], and without a series the effect of any treatment cannot be described. The measurement takes three minutes.

2. The Erasmus respiratory insufficiency score, computed on admission and written in the notes. All three items were recorded here, as the second block of Table 5 sets out, and the score was 5 or 6 of 7, the maximum being awarded for the strength item alone. In the international validation the lowest strength band carried an odds ratio of 30.9 (95% confidence interval 12.8 to 74.4) for ventilation within a week[28], and the paediatric version needs only age, cranial nerve involvement and the disability score[6]. What a written score changes is where the child sleeps.

3. A bedside measure of respiratory muscle strength, at least twice daily until the nadir has passed. A vital capacity, a negative inspiratory force or, in a child too young for either, a single-breath count and an assessment of neck flexion and cough. This record contains none of the four. The respiratory rate FELL to 12 breaths per minute as the child arrested, so the one respiratory variable that was recorded moved in the direction that looks reassuring.

4. An electrocardiogram on admission and after any heart rate above the age-specific threshold, with a corrected QT interval. The heart rate reached 160 beats per minute on hospital day 5 and two beta agonists and a beta blocker appear in that day's plan. No electrocardiogram, rhythm strip or corrected QT interval appears anywhere in the admission. Dysautonomia occurs in about a third of patients and is the mechanism of the deaths that are not respiratory[91].

5. A validated pain score at every nursing observation, and an analgesic prescription sized to it. The admission examination records hyperaesthesia in all four limbs; the morphine infusion ran at 5.0 µg/kg/hour, between 2 and 8 times below the customary lower bound, beneath a midazolam infusion at twice the customary upper bound. No pain score appears on any day. In a sedated ventilated child the score must be behavioural.

6. A stool culture and a single serum sample for anti-ganglioside antibodies, on the day of admission. Diarrhoea preceded the weakness by 5 to 6 days, and neither test was done. Anti-glycolipid antibodies were positive in 1,309 of 1,413 patients in the international cohort[57], and a positive result would have supported the axonal label that the electrodiagnostic report cannot support on its own.

7. A nerve conduction report that prints its numbers, and a second study at two to three weeks. The report here contains no latency, no amplitude and no conduction velocity, so no published criterion set can be applied to it. Subtype changes on serial testing in 37.8 to 44.7% of patients[32], and the two commonest criterion sets disagree in 31.7% of studies[31].

8. A repeat lumbar puncture between days 7 and 14 when the first is normal. The one sample here was taken 3 to 4 days after the onset of weakness, inside the window in which 43% of patients have no dissociation, and it was never repeated[22]. The single item that holds this case at Brighton level 2 is that protein[19].

9. The energy density of the enteral formula, recorded once, and a weight recorded at every review. The record states the volume of every feed and never states its energy content, so the delivered energy cannot be computed at all; and the two outpatient weights differ by 2.0 kg, 9.5% of body weight, in 7 days without comment. Early enteral nutrition was associated with an adjusted odds ratio for unit mortality of 0.071 (95% confidence interval 0.009 to 0.542) in intubated children[103].

10. Two dates: the date on which second-line therapy is decided and the date on which the first session is delivered. This is the item this report exists to propose. Here the two dates were hospital day 10 and hospital day 22 to 24, an interval of 12 to 14 days that nobody counted. Recording the pair costs nothing, makes the interval auditable, and turns an invisible property of the health system into a number a unit can act on.

Rehabilitation, and the plateau that follows

The graded strength series tabulated in Table 4 rises to a plateau on hospital day 28 and does not move for the following 18 days, and the child was on rehabilitation throughout that period. Two things should be said about it. The first is that a plateau at this point is not unexpected and is not evidence that rehabilitation was failing: recovery from an axonal neuropathy is limited by axonal regrowth and is measured in months, and among 51 children followed for a median of 6 years 4 months the sequelae rate was 67% (95% confidence interval 53 to 78)[75]. The second is that the rehabilitation itself is barely recorded. The plan of both outpatient reviews names medical rehabilitation and four vitamins; it names no exercise prescription, no frequency, no functional target and no measure of progress other than the strength grades themselves.

What the evidence supports is more specific than a referral. In 47 inpatients with this syndrome the Spinal Cord Independence Measure rose from 50 at rehabilitation admission to 80 at discharge and 95 at a mean 7.5-year follow-up (p = 0.001)[106], and in a randomised trial of 16 adults with chronic disease supervised individualised exercise produced a median between-group difference at six months of 5 points (95% confidence interval 0 to 20) on the 100-point Barthel Index against unsupervised home exercise[107] – a small and imprecise effect, and the honest reading of it is that supervision helps a little rather than transformatively. Timing is the part that is genuinely contested: a randomised trial of early active mobilisation in 750 ventilated intensive-care adults found median days alive and out of hospital at 180 days of 143 against 145 with usual care, an absolute difference of -2.0 days (95% confidence interval -10 to 6, p = 0.62)[108], so earlier is not automatically better. What does predict recovery of daily function after critical illness is cognitive state and delirium: among 65 patients with intensive-care-

acquired weakness, the cognitive component of the functional independence measure at unit discharge (beta 19.12, p = 0.001) and the duration of delirium (beta -9.83, p < 0.001) were independently associated with recovery of activities of daily living[109]. This child received a midazolam infusion at twice the customary rate for most of three weeks, and no delirium assessment appears anywhere in the record.

Limitations

This is one patient, described from a record written for clinical rather than research purposes, and every conclusion drawn here is bounded by that. Six limitations are specific enough to state. First, the escalation interval is an interval and not a point, because the date of the first exchange is not printed; every quantity built on it is reported as a range of 2 days' width and none is collapsed. Second, the Medical Research Council sum score is a reconstruction from per-limb grades under an assumption stated in Table 4, and although the assumption does not change the Erasmus score item it does change the sum scores themselves, which should be read as approximate. Third, the disability score is assigned here from the description in each note and was not recorded prospectively; it is the least ambiguous of the instruments used, but it is still an interpretation. Fourth, the prognostic nutritional index and the Erasmus score were both derived in adults and are applied here for comparison rather than for prediction, and the discordance of the nutritional index in this patient is a single observation about an instrument with an area under the curve of 0.769, not evidence against it[21]. Fifth, follow-up ends 45 days after admission, so the outcomes the literature uses – the ability to walk at four weeks, three months and six months – are not available, and the sequelae rate of 67% at a median of six years in a paediatric cohort[75] cannot be located against anything in this record. Sixth, the reproduction quality of the magnetic resonance images limits what Figure 3 can be used to assert, and the caption says so; we reproduce the sagittal panels because they carry the finding the report describes, and we do not annotate what the resolution will not support.

One further limitation is of a different kind and we state it plainly. This report criticises a record, and a record is not the care that produced it. Many of the measurements named above may have been made and not written down, and some of the reasoning we describe as absent may have taken place at a bedside and never reached the notes. What we can show is what a subsequent reader can reconstruct, which is precisely what an audit and a handover both depend on. The minimum data set is offered in that spirit: not as an accusation about what was done, but as a list of what has to be written down for the next reader to be able to check it.

5. Conclusion

An 8-year 9-month-old boy with axonal Guillain-Barre syndrome following a diarrhoeal illness became ventilator dependent on hospital day 4, completed a 5-day course of immunoglobulin without recovering, and was written up for plasma exchange on hospital day 10. The first exchange was completed between hospital days 22 and 24: an escalation interval of 12 to 14 days between a decision and its delivery, longer than the immunoglobulin course that preceded it, and attributed by the record itself to an inter-departmental referral that had not been connected and, later, to a blocked central line. That interval is not diagnostic delay, it is measurable to the day from any set of ward notes, and we have found no paediatric report that states it.

The case cannot show that plasma exchange rescued this child, and we have set out why: the first documented return of motor activity precedes the earliest possible date of the first exchange by 9 to 11 days, the ventilator was already being weaned, and on the disability score the disease's own literature uses as its endpoint the child ended the recorded period, 45 days after admission, in the state he was admitted in. What the record does show is that every instrument this disease possesses was computable from the notes written on the day of admission and that none was computed: an Erasmus respiratory insufficiency score of 5 or 6 of 7 on the morning of a day whose evening brought a respiratory arrest; a Brighton level of 2 held there by a single cerebrospinal fluid protein of 28.6 mg/dL that was sampled early and never repeated; a nerve conduction report carrying no number that any criterion set could use; a morphine infusion between 2 and 8 times below the customary lower bound beneath a midazolam infusion at twice the customary upper bound, with no pain score on any day; and a weight for height falling from 77.8% to 70.4% with no nutritional response in the plan.

We therefore propose two things. The first is a 10-item minimum data set, of which nine items are free and none requires equipment a unit treating such a child does not already own. The second is narrower and, we think, more portable: that every unit which escalates a child to second-line immunotherapy record two dates – the date the decision is made and the date the first session is delivered – and report the difference. In this admission that difference was 12 to 14 days and nobody counted it. A quantity nobody counts cannot be improved, and this one is counted with a calendar.

The indication for therapeutic plasma exchange was consistent with published practice guidance.[110]

Declarations

Ethics approval

Ethical approval for this case report was granted by the Research Ethics Committee of CMHC Research Center, Indonesia (reference no. CMHC/VI/2026/020).

Consent for publication

Written consent for publication of this de-identified case, including all tables and figures and both sets of images, was obtained from both parents, and the child gave age-appropriate assent. All directly identifying information has been removed: the child's name, medical record number and date of birth do not appear, and every interval is expressed as a hospital day counted from the earliest dated entry in the record. Calendar dates appear only where they define that anchor. The burned-in identifiers present in the source images – a patient name in a browser tab, a date of birth, a recorded age, an institution name and a study description – have been masked in Figures 2 and 3, and the masks obscure no anatomical region. Neither figure shows a face.

Data availability

All data supporting the findings are contained within the article. The underlying clinical record is held at Dr. M. Djamil Hospital, Padang, and cannot be shared publicly because it contains identifying information. Every derived quantity reported here – the absolute neutrophil and lymphocyte counts, the neutrophil-to-lymphocyte ratio, the prognostic nutritional index, the reconstructed Medical Research Council sum scores, the Erasmus respiratory insufficiency score, the Brighton level, the weight-for-height percentages, the body mass index, the per-kilogram drug exposures, the endotracheal tube depth estimates, the plasma-exchange volume per kilogram, the recomputed basal metabolic rate and every interval in days – is reproducible from the values printed in Tables 1 to 6 together with the sources cited in the table notes. Two exceptions are unavoidable and are stated as

such: the record prints no date for the first plasma exchange, so every interval that ends at it is reported as a two-day range; and the record prints no energy density for the enteral formula, so the delivered energy is reported as not computable rather than as a number.

Conflict of interest

The authors declare no financial or non-financial conflicts of interest related to this manuscript.

Clinical trial registration

Not applicable. This is a retrospective single-patient case report and not a clinical trial.

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Author contributions

Contributions are stated in the CRediT taxonomy. DT: conceptualization, investigation, data curation, formal analysis, visualization, writing – original draft, project administration. RL: conceptualization, methodology, validation, supervision, writing – review and editing. Both authors meet the four ICMJE criteria for authorship, have read and approved the final manuscript, and agree to be accountable for all aspects of the work.

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Use of artificial intelligence

No artificial intelligence (AI) tools were used in the preparation of this manuscript.

CARE guideline statement

This report was prepared in accordance with the CARE guideline for case reports. A completed CARE checklist with measured page references accompanies the submission, and the patient and family perspective required by item 12 appears in the patient and family perspective subsection of the Results.

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