CASE REPORT

Which Weight? An Obesity Label Justified Against the Criterion It Does Not Meet, and Per-Kilogram Annotations No Single Body Weight Reproduces, in a 7-Year-Old Girl with Dengue with Warning Signs and Upper Gastrointestinal Bleeding: A Case Report

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ABSTRACT

Background: In children with excess weight, fluid rates, drug doses, urine output, and energy prescriptions depend on a body weight that is often not identified in the clinical record.

Objective: To audit the nutritional classification and implied dosing weights documented for a child with dengue with warning signs and upper gastrointestinal bleeding.

Methods: This CARE-guided case report reviewed the de-identified record of a 7-year-old Indonesian girl and recomputed anthropometric indices, weight-indexed orders, and a recorded metabolic-rate calculation.

Results: At 30 kg and 125 cm, body mass index was 19.20 kg/m^2 (92.4th centile), below the recorded obesity criterion, whereas weight for height was 123.4% of the median and supported obesity. No single body weight reproduced all printed per-kilogram annotations, and height had been entered in metres in a metabolic-rate equation requiring centimetres. The child recovered without shock, fluid bolus, transfusion, or respiratory support and was discharged on illness day 8.

Conclusion: The nutritional label remained supportable by one criterion but not by the criterion cited in the record. Naming the dosing weight and its basis in every weight-indexed order would make paediatric dengue records more auditable.

1. Introduction

Dengue is the most consequential arboviral disease of childhood in South-East Asia, and Indonesia carries one of the heaviest burdens in the world. Hospital-based surveillance from Bandung, covering three years of notifications from sixteen hospitals, shows spatial clusters of relative risk that were essentially unchanged across the study period, with reported cases concentrated in males and in children.[1] The strongest intervention evidence yet generated for the disease also comes from Indonesia: in a cluster-randomised trial of *Wolbachia*-infected *Aedes aegypti* deployments in Yogyakarta, virologically confirmed dengue occurred in 67 of the 2905 participants analysed (2.3%) in intervention clusters against 318 of 3401 (9.4%) in control clusters, giving a protective efficacy of 77.1% (95% confidence interval 65.3 to 84.9) and, against hospitalisation for virologically confirmed dengue, of 86.2% (95% confidence interval 66.2 to 94.3); these are test-negative estimates among febrile clinic attenders aged 3 to 45 years rather than population incidence reductions.[2] The clinical problem that remains is not whether dengue can be prevented at population level but how the individual child who reaches hospital in the critical phase should be measured, classified and dosed.

The 2009 World Health Organization classification divides dengue into disease without warning signs, with warning signs, and severe dengue. In a ten-year retrospective series of 699 children with clinical dengue from Jakarta, of whom 614 (87.8%) were confirmed and 211 of 614 (34.4%) had severe disease, the warning sign with the highest negative predictive value in children and adolescents was a rising haematocrit together with a rapidly falling platelet count, at 76.6% and 91.9% respectively.[3] Case-control data from the Thai-Myanmar border show that within the first five days of illness, mucosal bleeding (adjusted odds ratio 5.39, 95% confidence interval 1.06 to 27.52), a haematocrit change of 10% or more (3.68, 1.15 to 11.74) and a serum albumin below 35 g/L (8.10, 2.55 to 25.72) are each associated with progression to severe dengue.[4] Gastrointestinal symptoms are prominent in children: in 556 hospitalised Thai children aged 0 to 14 years the commonest were anorexia, in 91.7%, and vomiting, in 57.6%, followed by abdominal pain in 38.3% and diarrhoea in 33.3%, while overt gastrointestinal bleeding occurred in only 3.2%.[5] A large population-based cohort using the Taiwanese national insurance database quantified how sharply that risk is concentrated in the acute phase, with an adjusted hazard ratio for non-variceal upper gastrointestinal bleeding of 55.40 (95% confidence interval 32.17 to 95.42) within 30 days of dengue onset, and no statistically significant excess thereafter, although the medium-term estimate is too imprecise to exclude one (adjusted hazard ratio 0.55, 95% confidence interval 0.05 to 6.18).[6]

Against this background, excess body weight has emerged as a candidate determinant of dengue severity in children. A nine-year prospective paediatric cohort in Managua, Nicaragua, following 5940 children across 26,273 person-years, found that obesity carried an adjusted odds ratio of 1.21 (95% confidence interval 1.03 to 1.42) for dengue virus infection and of 1.59 (1.15 to 2.19) for symptomatic dengue among those infected.[7] A Sri Lankan population survey of 4782 children aged 10 to 18 years found that 12 of 66 (18.2%) seropositive children above the 97th body mass index centile had been hospitalised for dengue against 103 of 1086 (9.48%) below it, and logistic regression in the same survey gave an odds ratio of 2.33 (95% confidence interval 1.47 to 3.67) for hospitalisation above that centile.[8] In 858 Vietnamese children with dengue shock syndrome, obesity was associated with severe respiratory failure requiring mechanical ventilation (odds ratio 2.3, 95% confidence interval 1.31 to 4.06) although not with mortality or acute liver failure.[9] The association is not uniform across cohorts: among 332 children hospitalised for dengue in Vietnam, of whom 50 (15.1%) had warning signs and none progressed to severe dengue, abnormal nutritional status was not associated with warning signs once thrombocytopenia at admission (odds ratio 13.36, 95% confidence interval 4.26 to 41.88) and age above five years had been accounted for.[10] Immunological work in a cohort of 124 dengue patients shows that the early T-cell and natural killer cell signatures that mark progression to severe disease are exacerbated in patients with overweight or obesity,[11] and a cohort of 577 Sri Lankan adults found higher aminotransferases, C-reactive protein and ferritin in those with central obesity.[12]

That literature has a feature which is rarely made explicit and which matters at the bedside. Every study defines excess weight differently. A United States quality initiative classified children by a body mass index at or above the 95th centile, while the Nicaraguan cohort does not state which reference it used;[7,13] the Thai and Vietnamese paediatric series classify on World Health Organization body mass index for age;[9,14] the Sri Lankan survey uses the 97th centile of the same reference;[8] the Taiwanese adult cohort uses an Asian body mass index cut-off of 27.5 kg/m^2;[15] and a Thai plasma-leak score uses 25.0 kg/m^2, an overweight rather than an obesity threshold.[16] When 4151 Brazilian schoolchildren aged six to ten years were classified simultaneously by five different criteria, a locally derived criterion agreed closely with the World Health Organization excess-weight criterion, but agreement across the five was not uniform and all of it was assessed against another index rather than against any measure of adiposity.[17] A comparison of three international references in 22,737 Brazilian schoolchildren found body-mass-index z scores highly correlated across the references (r of at least 0.99) while the obesity percentile itself performed differently between them.[18] Indonesian practice adds a further layer, because weight for height expressed as a percentage of the median is used alongside body mass index for age, and the two do not have to agree.

The problem compounds once treatment begins. In a child with excess weight, the fluid rate, the drug dose, the reported urine output and the energy prescription are all quotients whose denominator is a body weight, and the same order means different things depending on which weight is used. Physiologically based modelling in a virtual population of children with obesity shows why that choice is not cosmetic: weight-normalised clearance and volume of distribution are both lower in children with obesity than in children without it, so the same milligrams per kilogram do not deliver the same exposure; the model reproduced observed concentrations with average fold errors of 1.09 for clindamycin, 1.24 for trimethoprim and 1.53 for sulfamethoxazole.[19] Practice falls a long way short of naming any of this. In 21,488 children hospitalised with asthma in the United States, guideline-non-adherent corticosteroid orders rose from 19.4% of healthy-weight children to 36.0% of those with class III obesity;[20] a quality initiative found correct dosing-weight use at 1.2% before an electronic calculator and provider education were introduced, rising only to 24.2% afterwards;[13] and a second found baseline appropriate dosing of seven high-risk medications at 37%.[21] Two population studies show the same drift arising with no error made at all: in 1554 children prescribed melatonin the maximum dose was similar whatever the weight, so weight and age explained only a marginal part of the variance in the dose itself but a substantial part of the variance in the dose per kilogram, and the children with overweight or obesity were the ones who received the lower dose per kilogram;[22] and among 347 children prescribed fluoxetine and 733 prescribed sertraline the absolute doses were the same across weight categories while the weight-normalised doses were significantly lower in those with overweight or obesity ($p < 0.01$).[23] None of these reports concerns a child whose weight-indexed decisions were being made hourly during a plasma-leak syndrome.

Novelty and aim. Published case reports of dengue in children with excess weight describe the nutritional label and the clinical course, but we are not aware of one that audits the record's own arithmetic: that recomputes the anthropometry from the raw measurements, tests whether the stated nutritional criterion is actually met, and then asks, of every per-kilogram figure printed in the chart, which body weight could have produced it. The aim of this report is to describe a 7-year-old Indonesian girl with dengue with warning signs and upper gastrointestinal bleeding who was labelled obese, and to show three things about her record: that the nutritional label does not survive recomputation against the reference the record itself invokes; that interval arithmetic on the per-kilogram annotations in her chart excludes the existence of any single body weight that reproduces them all; and that a metabolic-rate equation was evaluated with height in the wrong unit, understating her estimated energy expenditure by 18.5% of the correct value. We propose a minimum data set that would make each of these errors impossible to repeat, and we state at the outset that this is a single case and that none of these observations is a claim about outcome.

2. Methods

Case design and reporting framework

This CARE-guided, single-patient retrospective case report was prepared from routine clinical records at a tertiary referral hospital in Padang, West Sumatera, Indonesia. The report describes the illness from symptom onset through discharge in January 2023.

Patient information and consent

Clinical history, examination findings, laboratory results, treatment records, anthropometric measurements, fluid records, and nutritional calculations were abstracted from the de-identified clinical record. Written informed consent for publication was obtained from the patient's father as her legal guardian, with age-appropriate assent from the child. No photograph, radiograph, or other patient image is reproduced; all figures were generated from the tabulated numerical data.

Clinical record audit and analysis

Anthropometric indices were recomputed from the recorded weight and height using the cited growth references. Serial laboratory values, fluid and medication orders, urine-output

entries, and nutritional calculations were audited against the source data. For each printed per-kilogram quantity, the possible dosing-weight interval was inferred from its recorded rounding precision.

Ethical considerations

The report describes routine clinical care and introduced no intervention, additional investigation, or change in management. Direct identifiers were removed before analysis and publication.

3. Results

Presentation and referral

A 7-year 5-month-old Indonesian girl was brought to the emergency department of a tertiary referral hospital in Padang, West Sumatera, at 02:54 in January 2023, on the fourth day of a febrile illness. The whole course from the first day of fever to discharge is set out in Figure 1, which places every measurement quoted below on the illness-day axis used throughout this report.

Figure 1. *Clinical timeline from the first day of fever to discharge. Blue marks the febrile phase before referral, red the critical phase spanning the bleeding episode and the haematocrit peak, amber the period of falling haematocrit and platelet nadir, and teal the recovery phase. Every laboratory value shown is taken from the record and is tabulated in Tables 2 and 3; the vital signs and balances are in Table 5. The history and the volumes described in the day-1 to day-3 blocks appear in the text of the presentation and referral subsection and in no table.*

Fever had begun on illness day 1, three calendar days before she reached the referral centre, was continuous, reached a maximum recorded temperature of 41°C at home, and was not accompanied by rigors, drenching sweats or convulsions. Intermittent epigastric abdominal pain began the same day. Watery stools without mucus or blood began on illness day 2, occurred six to eight times a day and amounted to roughly a quarter of a glass on each occasion. Appetite fell to about a quarter of her usual intake and she became lethargic. On the evening of illness day 3, 4 hours before she reached the referral centre, she vomited once, the vomitus containing dark blood and amounting to about half a glass, preceded by nausea and accompanied by abdominal pain and without projectile force; on the same evening she passed one stool mixed with dark red blood of about one tablespoon in volume. Urine colour, frequency and volume were unchanged. There had been no skin rash, epistaxis or gum bleeding, no headache, retro-orbital pain or arthralgia, no cough, coryza or breathlessness, and no history of jaundice. The events of this prodrome that carry a date or a measurement are placed on the illness-day axis in Figure 1.

She had been referred from a private hospital where she had been an inpatient for 2 days with a diagnosis of dengue haemorrhagic fever grade II with haematemesis and melaena together with acute diarrhoea without dehydration. There she had received ceftriaxone 600 mg intravenously twice daily for two days, ondansetron 2 mg intravenously three times daily, omeprazole 20 mg intravenously twice daily, tranexamic acid 200 mg intravenously three times daily, paracetamol 250 mg intravenously four times daily and oral rehydration solution. A blood count on the second day of that admission, illness day 3, showed haemoglobin 11.9 g/dL, leucocytes 2.60 × 10^3/mm^3, platelets 195 × 10^3/mm^3 and haematocrit 36%, with a positive dengue NS1 antigen. No blood culture was taken before the first dose of ceftriaxone.

Her medical history comprised three previous hospital admissions: typhoid fever at two years of age, acute diarrhoea four months before this illness and typhoid fever two months before it. There had been no previous haematemesis and no bleeding disorder in the family. She was born at term by caesarean section weighing 2640 g, cried immediately and required no neonatal care. Basic immunisation was complete, with a visible bacille Calmette-Guerin scar; she had not received a coronavirus disease 2019 vaccine. Development was normal: she rolled prone at 4 months, sat at 6 months, walked at 11 months and could read and write at 5 years. She was prepubertal, with Tanner stage A1M1P1 and no menarche. She was an only child living with both parents in a permanent house with adequate ventilation, a tiled floor, bottled drinking water, a piped water supply, an indoor toilet and municipal refuse collection. The single identified mosquito breeding site was an indoor aquarium containing no fish. A neighbour in the same street had been admitted locally with confirmed dengue one week earlier; no information was available about her school.

Examination and anthropometry

She was fully conscious, with a Glasgow Coma Scale score of E4V5M6, and was assessed as seriously ill. On the paediatric assessment triangle her appearance was eutonic, calm and interactive with intact eye contact and speech; breathing was vesicular without retraction or nasal flaring; and circulation showed neither pallor nor cyanosis. Blood pressure was 110/70 mmHg, which lies between the 50th centile of 97/58 mmHg and the 90th centile of 111/72 mmHg printed in her chart for her age, sex and height, and well below the 95th centile of 115/76 mmHg. Heart rate was 130 beats per minute, regular and of good volume, respiratory rate 20 breaths per minute, axillary temperature 37.0°C and peripheral oxygen saturation 99% in air.

The tourniquet test was positive. There was no lymphadenopathy. The conjunctivae were not pale, the sclerae were not icteric, the pupils were 3 mm and equally reactive, and there was no palpebral oedema. There was no ear discharge; the tonsils were T1-T1 and the pharynx was not injected. There was no circumoral cyanosis, the oral mucosa and lips were moist and there was no bleeding from the gums. The chest was symmetrical and resonant with vesicular breath sounds and no crackles or wheeze; the apex beat was impalpable to inspection but palpable one finger medial to the left mid-clavicular line in the fifth intercostal space, with normal cardiac borders, a regular rhythm and no murmur. The abdomen was not distended, abdominal striae were visible, the abdomen was soft, the liver and spleen were not palpable, there was epigastric tenderness without rebound, skin turgor returned normally, percussion was tympanitic and bowel sounds were normal. The genitalia were prepubertal. The extremities were warm with a capillary refill time under 2 seconds. **No oedema of the eyelids, abdominal wall or lower limbs was recorded at any point in the admission examination or in any subsequent daily entry.**

Weight was 30 kg and height 125 cm. The record states a body mass index of 19.8 kg/m^2, an ideal body weight of 24 kg, a height age of 7 years, weight for age 125%, height for age 101% and weight for height 125%, and classifies her as obese with normal stature on the basis that her body mass index lies above the 95th centile. Recomputation of these figures from the two raw measurements, which is set out in Table 1, does not reproduce the classification; that discrepancy is examined in the nutritional-label subsection of the Discussion. No waist circumference, skinfold thickness or any other measure of adiposity was recorded, and no further weight was measured at any point after admission.

Laboratory findings

The serial full blood counts obtained between admission and discharge are tabulated in Table 2 and plotted in Figure 2. At 02:54 on illness day 4 the haemoglobin was 13.0 g/dL, the haematocrit 37%, the leucocyte count 4.57 × 10^3/mm^3 and the platelet count 106 × 10^3/mm^3. The differential count showed 12% band forms and 44% segmented neutrophils, a left shift, with 40% lymphocytes and 4% monocytes and no pathological cells. Eleven hours later, at 14:00 on the same day, the haemoglobin had risen to 16.1 g/dL and the haematocrit to 48%, while the platelet count had fallen to 67 × 10^3/mm^3. That is a rise of 11 haematocrit points in 11.1 hours, a mean rate of 0.99 points per hour, and a relative rise of 29.7% against the admission value. Measured instead against the haematocrit of 36% recorded at the referring hospital, or against the convalescent value of 36% on illness day 8, the same peak represents a rise of 33.3%. The haemoglobin rose by 3.1 g/dL over the same interval, a relative rise of 23.8%, and the record annotated each of the three successive haemoglobin values above 15 g/dL as normal while simultaneously annotating the paired haematocrit as raised.

Table 1. Anthropometry and nutritional classification as recorded and as recomputed from the two raw measurements.

Measurement or index	As recorded	Recomputed	Reference position at 89.5 months
Weight	30 kg	30 kg (measured once)	median 24.0 kg
Height	125 cm	125 cm (measured once)	median 124.4 cm
Body mass index	19.8 kg/m²	19.20 kg/m²	85th 17.90, 95th 20.08 kg/m²
Body mass index z score	*not recorded*	+1.43	85th = +1.04, 95th = +1.65
Body mass index centile	**stated as > 95th; is 94.3th**	92.4th centile	obese at 95th centile or above
Body mass index z score, WHO 2007 reference	*not recorded*	+1.69 (95.4th centile)	overweight above +1, obese above +2
Weight for age	125%	124.9% of the median	88.2th centile
Height for age	101%	100.5% of the median	54.3th centile
Height age	7 years	7.56 years	age at which median height = 125 cm
Ideal body weight	24 kg	24.31 kg	median weight at height age
Weight for height	**125%**	**123.4% of the median**	obese above 120%
Classification on body mass index for age	**obese**	overweight	85th to below 95th centile
Classification on weight for height	**obese**	**obese**	above 120%

Red marks a statement in the record that the recomputation does not support, or a value that lies outside the criterion printed beside it; a grey cell marks an index the record does not state. Percentiles and z scores are computed from the Centers for Disease Control and Prevention 2000 lambda-mu-sigma parameters for girls, evaluated by linear interpolation at 89.5 months, the mid-month age corresponding to 7 years 5 months. Ideal body weight is the median weight at the height age, the age at which the median height equals the measured height. The recorded body mass index of 19.8 kg/m² would require a height of 123.1 cm at the recorded weight. The single grey cell marks the one index the record does not state at all: no body-mass-index z score was computed.

The platelet count fell from 106 × 10³/mm³ on admission to a nadir of 36 × 10³/mm³ at 12:00 on illness day 5, a fall of 66.0% over 33.1 hours, and had recovered to 176 × 10³/mm³ by illness day 8. The leucocyte count, which had been 2.60 × 10³/mm³ at the referring hospital, reached its lowest value at the referral centre of 3.88 × 10³/mm³ at 14:00 on illness day 4 and then rose to a peak of 13.58 × 10³/mm³ at 06:00 on illness day 6, a trajectory that is the expected convalescent leucocytosis of dengue rather than evidence of bacterial infection. Every value in this paragraph is listed in Table 2, and Figure 2C presents the leucocyte trajectory on its own axis.

Table 2. Serial full blood counts from the referring hospital admission to discharge, with the intravenous fluid rate in force at the time of each sample.

Illness day and time	Haemoglobin (g/dL)	Leucocytes (× 10³/mm³)	Haematocrit (%)	Platelets (× 10³/mm³)	Intravenous rate
Day 3 (referring hospital)	11.9	**2.60**	36	195	*not recorded*
Day 4, 02:54	13.0	4.57	37	**106**	110 mL/h
Day 4, 14:00	**16.1**	**3.88**	**48**	**67**	110 mL/h
Day 4, 21:00	**15.6**	7.14	**47**	**56**	110 mL/h
Day 5, 04:30	15.1	6.55	45	**50**	100 mL/h
Day 5, 12:00	14.5	6.81	44	**36**	70 mL/h
Day 5, 21:00	12.3	11.43	35	**45**	58 mL/h
Day 6, 06:00	12.1	**13.58**	36	**46**	16 mL/h
Day 6, 18:00	**11.2**	9.19	**32**	**51**	4 mL/h
Day 7	11.9	6.68	35	**90**	stopped
Day 8	11.8	8.57	36	176	stopped

Values outside the paediatric reference interval are shown in red; grey cells mark data the record does not contain. Reference intervals are conventional paediatric intervals for a school-aged child: haemoglobin 11.5–15.5 g/dL, leucocytes 4.5–13.5 × 10³/mm³, haematocrit 35–45%, platelets 150–450 × 10³/mm³. The referring hospital's sample was taken on the second day of that admission. The haematocrit rose by 11 points, or 29.7%, between the first two samples of illness day 4, an interval of 11.1 hours.

The biochemical, coagulation and hepatic results are set out in Table 3. At admission the sodium was 133 mmol/L, potassium 3.3 mmol/L, chloride 99 mmol/L, total calcium 7.6 mg/dL and albumin 3.2 g/dL. Correcting the calcium for the albumin using the conventional adjustment[24] of 0.8 mg/dL per 1 g/dL of albumin below 4.0 g/dL gives 8.24 mg/dL, which reproduces the value printed in the record and remains below the lower reference limit. The second sample, drawn at 14:00, showed urea 19 mg/dL, creatinine 0.5 mg/dL, prothrombin time 11.7 seconds, activated partial thromboplastin time 41.6 seconds, aspartate aminotransferase 125 U/L and alanine aminotransferase 36 U/L, an aspartate to alanine ratio of 3.47 and multiples of the laboratory upper limit of 3.4 and 1.4 respectively. Serum albumin was measured once only, at 02:54, before the haematocrit peak; it was never repeated.

Table 3. Biochemistry, coagulation, hepatic profile, stool examination, urinalysis and dengue serology, with the investigations that were not performed.

Investigation	Result	Reference interval
Admission sample, illness day 4, 02:54		
Erythrocytes	4.85 × 10^6/mm^3	4–5.2 × 10^6/mm^3
Reticulocytes	0.54%	0.5–2.5%
Mean corpuscular volume	**76 fL**	77–95 fL
Mean corpuscular haemoglobin	27 pg	25–33 pg
Mean corpuscular haemoglobin concentration	35%	32–36%
Band neutrophils	**12%**	< 5%
Segmented neutrophils	44%	40–70%
Lymphocytes	40%	25–45%
Monocytes	4%	2–10%
Eosinophils	**0%**	1–5%
Basophils	0%	< 1%
Sodium	**133 mmol/L**	135–145 mmol/L
Potassium	**3.3 mmol/L**	3.5–5.1 mmol/L
Chloride	99 mmol/L	98–107 mmol/L
Total calcium	**7.6 mg/dL**	8.8–10.8 mg/dL
Albumin-corrected calcium	**8.24 mg/dL**	8.8–10.8 mg/dL
Albumin	**3.2 g/dL**	3.8–5.4 g/dL
Second sample, illness day 4, 14:00		
Urea	19 mg/dL	10–50 mg/dL
Creatinine	0.5 mg/dL	0.3–0.7 mg/dL
Prothrombin time	11.7 s	10.8–14.4 s
Activated partial thromboplastin time	**41.6 s**	26.4–37.5 s
Aspartate aminotransferase	**125 U/L**	< 37 U/L
Alanine aminotransferase	**36 U/L**	< 26 U/L
Stool examination, illness day 5		
Occult blood	Negative	-
Colour	Yellow	-
Consistency	Soft	-
Blood	Negative	-
Mucus	Negative	-
Leucocytes	0–1/hpf	-
Erythrocytes	0–1/hpf	-
Amoebae	Negative	-
Helminth ova	Negative	-
Larvae	Negative	-
Undigested food residue	Positive	-
Stool culture	*planned, no result recorded*	-
Urinalysis, illness day 5		
Colour	Yellow	-
Turbidity	Clear	-
Specific gravity	1.024	-
pH	6.5	-
Erythrocytes	0–1/hpf	-
Leucocytes	1–2/hpf	-
Casts	Negative	-
Crystals	Negative	-
Epithelial cells	Positive, squamous	-
Protein	Negative	-
Glucose	Negative	-

Investigation	Result	Reference interval
Bilirubin	Negative	-
Urobilinogen	Positive	-
Dengue serology		
NS1 antigen, illness day 3 (referring hospital)	**Positive**	negative
IgM antibody, illness day 8	**Positive**	negative
IgG antibody, illness day 8	**Positive**	negative
Serotyping or polymerase chain reaction	*not performed*	-
Blood culture	*not performed*	-
Abdominal or chest ultrasonography	*not performed*	-
Chest radiograph	*not performed*	-

Values outside the reference interval are shown in red; grey cells mark data the record does not contain. Albumin-corrected calcium is total calcium + 0.8 x (4.0 – albumin in g/dL) and is shown in red because it remains below the reference interval after correction. The aminotransferase upper limits are those that reproduce the multipliers printed in the record, 3.4 and 1.4 times the limit respectively. The record annotates the activated partial thromboplastin time as 1.5 times raised, which implies a comparator of 27.7 seconds that it does not print. Grey cells mark investigations that were planned but not reported, or never performed.

A stool examination on illness day 5 was negative for occult blood and showed no blood, mucus, amoebae, helminth ova or larvae, with 0 to 1 leucocytes and 0 to 1 erythrocytes per high-power field and positive undigested food residue. A stool culture was planned but no result appears in the record. Urinalysis on the same day showed clear yellow urine, specific gravity 1.024, pH 6.5, no protein, glucose or bilirubin, positive urobilinogen, 0 to 1 erythrocytes and 1 to 2 leucocytes per high-power field, and squamous epithelial cells. On illness day 8 dengue IgM and IgG were both positive. No serotyping or reverse-transcription polymerase chain reaction was performed. No abdominal or chest ultrasonography and no chest radiograph were obtained at any point; Table 3 records these omissions alongside the investigations that were performed.

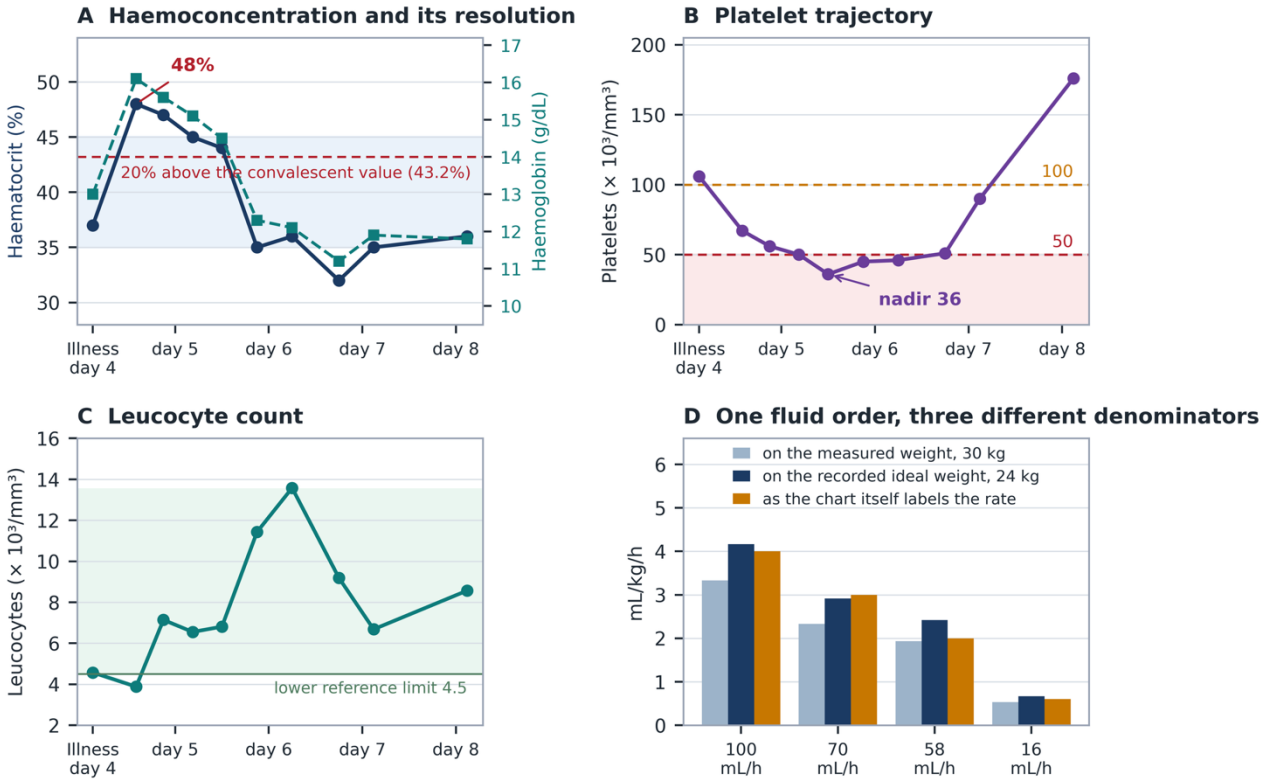

Figure 2. *Laboratory trajectories and the fluid arithmetic.* ***(A)*** *Haematocrit and haemoglobin, with the paediatric haematocrit reference band shaded and a dashed line at 20% above the convalescent value; the peak of 48% lies above that line however the baseline is anchored.* ***(B)*** *Platelet count, with the 100 and 50 × 10³/mm³ thresholds marked and the nadir of 36 × 10³/mm³ on illness day 5 annotated.* ***(C)*** *Leucocyte count, showing the early leukopenia and the convalescent leucocytosis.* ***(D)*** *Each labelled step of the fluid ladder read on the two candidate denominators, the measured weight and the recorded ideal weight, alongside the figure the chart itself prints.*

Diagnosis and initial management

Four diagnoses were recorded: dengue with warning signs (A91); haematemesis and melaena attributed to thrombocytopenia due to dengue (K92); acute diarrhoea without dehydration (K52.9); and obesity (E66.9). The warning signs documented were persistent abdominal pain with epigastric tenderness, mucosal bleeding and a rising haematocrit with a rapidly falling platelet count. Clinical fluid accumulation, hepatomegaly, lethargy with restlessness and persistent vomiting were not present, and no imaging was performed to look for the first of these.

Initial treatment was intravenous Ringer's acetate at 110 mL/h, annotated in the same order as 2700 mL over 24 hours

and described as maintenance plus a 5% deficit; paracetamol 250 mg intravenously as required for fever, annotated as 10 mg/kg/dose; insertion of a nasogastric tube with temporary fasting and measurement of gastric aspirate; omeprazole 30 mg intravenously once daily, annotated as 1 mg/kg/dose; tranexamic acid 200 mg intravenously three times daily, annotated as 10 mg/kg/dose; zinc 20 mg orally once daily; and oral rehydration solution 200 mL after each loose stool. Ceftriaxone 1200 mg intravenously twice daily was continued from the referring hospital at double the previous dose. The plan specified a full blood count every 6 hours, measurement of fluid balance and urine output, urinary catheterisation and surveillance for fluid overload. The complete set of weight-indexed orders, with the quotient each one gives on the measured weight and on the recorded ideal weight, is set out in Table 4.

Hospital course

She remained afebrile from illness day 5 apart from one transient recorded temperature of 37.4°C on illness day 6, and never developed shock: the systolic pressure ranged from 95 to 110 mmHg, the heart rate fell from 130 to 90 beats per minute, capillary refill remained under 2 seconds and peripheral oxygen saturation stayed at 99% throughout, as Table 5 records. There was no further haematemesis, no melaena and no blood in the nasogastric aspirate after admission. Diarrhoea settled by illness day 7. A generalised erythematous rash appeared on the hands and feet on illness day 6 and evolved into a convalescent rash by illness day 7, as shown in Figure 1.

Table 4. Every weight-indexed decision in the record, with the quotient it gives on the measured weight and on the recorded ideal weight, set against the per-kilogram figure the chart itself prints.

Weight-indexed decision	As ordered	On the measured weight, 30 kg	On the recorded ideal weight, 24 kg	Per-kilogram figure printed in the chart
Initial intravenous fluid	110 mL/h	3.67 mL/kg/h	4.58 mL/kg/h	*none*
Fluid ladder step	100 mL/h	**3.33 mL/kg/h**	4.17 mL/kg/h	4 mL/kg/h
Fluid ladder step	70 mL/h	**2.33 mL/kg/h**	2.92 mL/kg/h	3 mL/kg/h
Fluid ladder step	58 mL/h	1.93 mL/kg/h	2.42 mL/kg/h	2 mL/kg/h
Fluid ladder step	16 mL/h	**0.53 mL/kg/h**	**0.67 mL/kg/h**	0.6 mL/kg/h
Fluid ladder step	4 mL/h	0.13 mL/kg/h	0.17 mL/kg/h	*none*
Paracetamol	250 mg IV as required	**8.33 mg/kg/dose**	10.42 mg/kg/dose	10 mg/kg/dose
Omeprazole	30 mg IV once daily	1.00 mg/kg/dose	1.25 mg/kg/dose	1 mg/kg/dose
Tranexamic acid	200 mg IV three times daily	**6.67 mg/kg/dose**	**8.33 mg/kg/dose**	10 mg/kg/dose
Ceftriaxone	1200 mg IV twice daily	80.00 mg/kg/day	100.00 mg/kg/day	*none*
Zinc	20 mg orally once daily	*not weight-indexed*	*not weight-indexed*	*none*
Urine output, hospital day 2	0.7 mL/kg/h	= 504 mL/24 h	= 403 mL/24 h	0.7 mL/kg/h
Maintenance fluid (Holliday-Segar)	1580 mL/day prescribed on hospital day 2	1700 mL/day	1580 mL/day	*none*
Maintenance plus a 5% deficit (the initial order)	2700 mL/24 h as annotated	3200 mL/day	2780 mL/day	*none*
Energy prescription	1440 kcal/day	would be 1800 kcal/day	1440 kcal/day	60 kcal/kg/day
Dietary recall in the month before admission	1700 kcal/day	*not weight-indexed*	= 118.1% of the allowance	*none*
Basal metabolic rate (weight-and-height equation)	**882 kcal/day recorded, with the height entered in metres**	1082.5 kcal/day with the height in centimetres	*not applicable*	*none*
Fluid requirement on the nutritional care form	2400 mL/day	2400 mL/day	would be 1920 mL/day	80 mL/kg/day

Red marks a quotient that does not reproduce the printed figure at the precision at which that figure is written, and, in the last block, the one entry that is arithmetically wrong; the last four rows carry no printed per-kilogram figure, so no quotient in them can be marked on that rule. A per-kilogram figure printed to a stated number of decimal places constrains the weight that produced it: a rate written as 4 mL/kg/h implies an underlying value between 3.5 and 4.5, so an order of 100 mL/h implies a weight between 22.2 and 28.6 kg. Intersecting the intervals implied by the 4 labelled steps of the fluid ladder gives 24.6 to 28.0 kg, which excludes both weights in the record. The energy prescription is the one decision that uses the ideal weight explicitly and correctly; the fluid requirement on the same form uses the measured weight.

The intravenous rate was reduced in four documented steps as the haematocrit fell, from 100 mL/h through 70, 58 and 16 mL/h to 4 mL/h; the first 4 of those five rates are labelled in the chart with a figure in millilitres per kilogram per hour and the last carries no label at all. Total prescribed fluid was 1580 mL on hospital day 2 and 1000 mL on hospital day 3. Enteral feeding was reintroduced as a low-lactose milk formula, initially 6 × 50 mL and then 6 × 100 mL by nasogastric tube, and by hospital day 4 as a soft diet of 500 kcal with 4 × 150 mL of formula. The daily net fluid balance is set out in Table 5: +800 mL on hospital day 2 with a urine output recorded as 0.7 mL/kg/h, +53 mL on hospital day 3 with 1.1 mL/kg/h, and +5 mL on hospital day 4 with 1.2 mL/kg/h. The body weight against which those urine outputs were indexed is not stated anywhere in the record, and no repeat weight was measured, so the actual urine volume cannot be recovered. The de-escalation is illustrated in Figure 1 and the daily balances are tabulated in Table 5.

Table 5. Vital signs, cumulative fluid balance and urine output through the admission.

Hospital day (illness day)	Blood pressure (mmHg)	Heart rate (/min)	Respiratory rate (/min)	Temperature (°C)	Fluid balance (mL)	Urine output (mL/kg/h)
Admission, 02:54 (day 4)	110/70	130	20	37.0	*not recorded*	*not recorded*
1, 21:30 (day 4)	110/70	130	20	37.0	*not recorded*	*not recorded*
2 (day 5)	100/70	110	21	36.9	+800	0.7
3 (day 6)	110/60	100	20	37.4	+53	1.1
4 (day 7)	95/60	97	20	37.0	+5	1.2
5 (day 8)	105/60	90	20	36.8	*not recorded*	*not recorded*

Peripheral oxygen saturation was 99% at every recorded observation and capillary refill remained under 2 seconds throughout; neither is tabulated. Grey cells mark data the record does not contain. Heart rates above the conventional upper limit of 110 beats per minute for a school-aged child are shown in red; two of the six recorded values exceed it. Blood-pressure centiles printed in the record for this age, sex and height were 5th 80/40, 50th 97/58, 90th 111/72, 95th 115/76 and 99th 122/83 mmHg. Fluid balance and urine output were not recorded on the day of admission or on the day of discharge. The body weight against which the urine output was indexed is not stated anywhere in the record.

No fluid bolus was given at any point, no colloid was used, no blood product was transfused, no vasoactive agent was required and no respiratory support was needed. She was transferred out of high-dependency care on illness day 7 and discharged home on illness day 8, after 5 days at the referral centre and 7 inpatient days across both institutions. The final platelet count was 176 × 10^3/mm^3 and the final haematocrit 36%. Table 5 shows that the daily net balance fell towards zero over the same three days.

Nutritional assessment during the admission

A structured paediatric nutritional care assessment was completed on hospital day 3. It recorded the same weight and height as on admission, gave the ideal body weight as 24 kg and the height age as 7 years, and prescribed energy as the ideal body weight multiplied by the recommended daily allowance for height age, 24 kg × 60 kcal/kg/day = 1440 kcal/day. A dietary recall taken for the month before admission totalled 1700 kcal/day across five eating occasions, of which a single mid-afternoon portion of fried snacks contributed 300 kcal and the 3 rice portions 525 kcal in total; that is 118.1% of the prescribed allowance. Basal metabolic rate was calculated from the weight-and-height Schofield equation for girls aged 3 to 10 years, and the total energy expenditure was set equal to it because the stress and activity factors were both taken as 1.0. The form recorded a basal metabolic rate of 882 kcal/day. Fluid requirement was calculated on the same form as the measured body weight multiplied by 80 mL/kg/day, giving 2400 mL/day. At the time of the assessment the inpatient intake was 600 kcal/day by nasogastric tube, recorded as 41% of the ideal allowance, which recomputes to 41.7%, and 68% of the total energy expenditure.

Differential diagnosis, severity classification and discharge

The bleeding was attributed to thrombocytopenia due to dengue, and the alternatives were addressed on the clinical record as follows. **Mallory-Weiss tear** is the closest competitor and is not excludable: she had vomited repeatedly before the single episode of haematemesis, no endoscopy was performed, and a mucosal tear would have produced exactly the picture seen, a single self-limiting bleed followed by none. **Peptic or stress ulceration** was clearly entertained clinically, since a proton pump inhibitor was started at the referring hospital and continued; there was no history of chronic dyspepsia, no previous ulcer and no antisecretory drug or non-steroidal anti-inflammatory exposure before the illness, but again no endoscopy was performed. **Enteric fever with intestinal bleeding** deserved more attention than it received: she had been treated for typhoid twice, most recently two months before this illness, in an endemic city, and she had six to eight watery stools a day. No blood culture, no Widal test and no typhoid-specific serology was obtained, and the stool culture that was planned was never reported, so enteric fever was never formally excluded. **Variceal bleeding** is excludable on the record: there was no hepatosplenomegaly, no ascites, no jaundice, no history of neonatal umbilical catheterisation and no stigmata of chronic liver disease, and the aminotransferase pattern of 125 and 36 U/L with a normal prothrombin time is that of dengue hepatitis rather than of cirrhosis. **Swallowed blood** from epistaxis or the gums is excluded by the examination, which records neither. **An inherited bleeding disorder** is not supported: the prothrombin time was normal, the family history was negative and there had been no previous bleeding, although the isolated prolongation of the activated partial thromboplastin time was never repeated or investigated.

The record labels the second episode melaena. What is described is one stool mixed with dark red blood of about one tablespoon, which is haematochezia rather than melaena; the term melaena is reserved for a black tarry stool. The distinction matters here only for the accuracy of the label, since either finding places the bleeding in the gastrointestinal tract and neither changes the management, but a report whose subject is the auditability of clinical labels should audit this one. The stool examination on illness day 5 was negative for occult blood, which is consistent with a single episode that had stopped.

Severe dengue was excluded against the four World Health Organization 2009 criteria item by item. **Severe plasma leakage leading to shock or to fluid accumulation with respiratory distress:** her systolic pressure ranged from 95 to 110 mmHg, her pulse pressure from 30 to 50 mmHg and was never narrowed, her capillary refill time never exceeded 2 seconds, her respiratory rate never exceeded 21 breaths per minute and her oxygen saturation was 99% at every observation. No imaging was performed to look for fluid accumulation, so that half of the criterion rests on clinical examination alone. **Severe bleeding as evaluated by the treating clinician:** this is the criterion on which reasonable clinicians could differ, since haematemesis and melaena in a 7-year-old may be judged severe on their face. The record supports the judgement that it was not: there was a single episode of each before transfer and none afterwards, the haemoglobin rose from 11.9 g/dL at the referring hospital through 13.0 to 16.1 g/dL rather than falling, no blood product was transfused, there was no haemodynamic change attributable to blood loss and faecal occult blood was negative by illness day 5. **Severe organ involvement:** the aspartate aminotransferase of 125 U/L is far below the 1000 U/L threshold, the Glasgow Coma Scale score was 15

throughout, and there was no cardiac or other organ failure. She therefore satisfies dengue with warning signs and not severe dengue on every limb of the definition that the record can address.

Discharge on illness day 8 met the conventional criteria, which the record does not itself enumerate. She had been afebrile for more than 48 hours without an antipyretic, her clinical condition was improving, her platelet count was rising and had reached 176 × 10^3/mm^3, her haematocrit was stable at 36% off intravenous fluid, she had no respiratory distress, her urine output was 1.2 mL/kg/h, her appetite had returned and she was tolerating a soft diet. What the record does not contain is any follow-up: no post-discharge review, no repeat platelet count, no convalescent serology, no repeat weight or height, no dietetic referral and no weight-management plan. Nor does it record any counselling: no documented advice on the warning signs that should prompt return, on the convalescent rash and post-dengue fatigue, on the avoidance of aspirin and non-steroidal anti-inflammatory drugs, or on eligibility for dengue vaccination in a child who is now demonstrably seropositive. The single identified household breeding site, an indoor aquarium containing no fish, and the confirmed case in a neighbouring household are recorded in the social history and are never revisited; no vector-control advice and no notification to the primary care service appear anywhere in the record.

Patient and family perspective. The child's father, who gave the history and the consent for this report, said that what frightened the family most was the vomiting of blood, and that they had not known that a child could bleed from dengue; he said that the explanation he most wanted, and did not receive in writing, was what to watch for at home after discharge. The child herself, asked at the time of discharge, said that the nasogastric tube and the repeated blood tests were the worst part of the admission and that she had not minded being in hospital otherwise. Both statements are reproduced here with their permission.

4. Discussion

Before anything else is said about this record, what it documents should be stated plainly. The diagnosis of dengue was made on illness day 3 on a positive NS1 antigen and the child was correctly classified as dengue with warning signs on arrival. All seven of the World Health Organization 2009 warning signs were addressed on the admission examination. Full blood counts were obtained every 6 to 12 hours through the critical phase. No fluid bolus, no colloid, no blood product and no vasoactive agent was given at any point, which in dengue is the correct restraint rather than an omission. The intravenous rate was de-escalated in four documented steps as the haematocrit fell and was below ideal-weight maintenance within 42.1 hours of arrival. The daily net balance was brought to +5 mL by illness day 7 without a single recorded sign of fluid overload, and the child went home well on illness day 8. The care was appropriate and the outcome was excellent. What follows concerns the record, not the clinical judgement, and a retrospective audit of a record establishes what was written, not what was thought.

The nutritional label does not survive recomputation against the reference the record invokes

The record classifies this child as obese, and the stated basis for that classification is that her body mass index lies above the 95th centile. Both halves of that statement can be checked. Her measured weight of 30 kg and height of 125 cm give a body mass index of 19.20 kg/m^2. The record prints 19.8 kg/m^2, a value that would require a height of 123.1 cm at the same weight. Evaluating the Centers for Disease Control and Prevention 2000 lambda-mu-sigma parameters for girls[25] at 89.5 months, the mid-month age corresponding to 7 years 5 months, places the 85th centile at 17.90 kg/m^2 and the 95th centile at 20.08 kg/m^2. The recomputed value of 19.20 kg/m^2 corresponds to a z score of 1.43 and the 92.4th centile. Neither the recomputed value nor the value printed in the record reaches the 95th centile: 19.8 kg/m^2 corresponds to the 94.3th centile, which is still below the threshold the record invokes. On body mass index for age she is overweight, in the band between the 85th and the 95th centile, and she is a long way from the 120% of the 95th centile, 24.10 kg/m^2, at which severe obesity is conventionally set because z scores behave poorly above the 97th centile of this reference.[26] Figure 3A plots both values against the reference curves, and Table 1 records the recorded and the recomputed figure side by side.

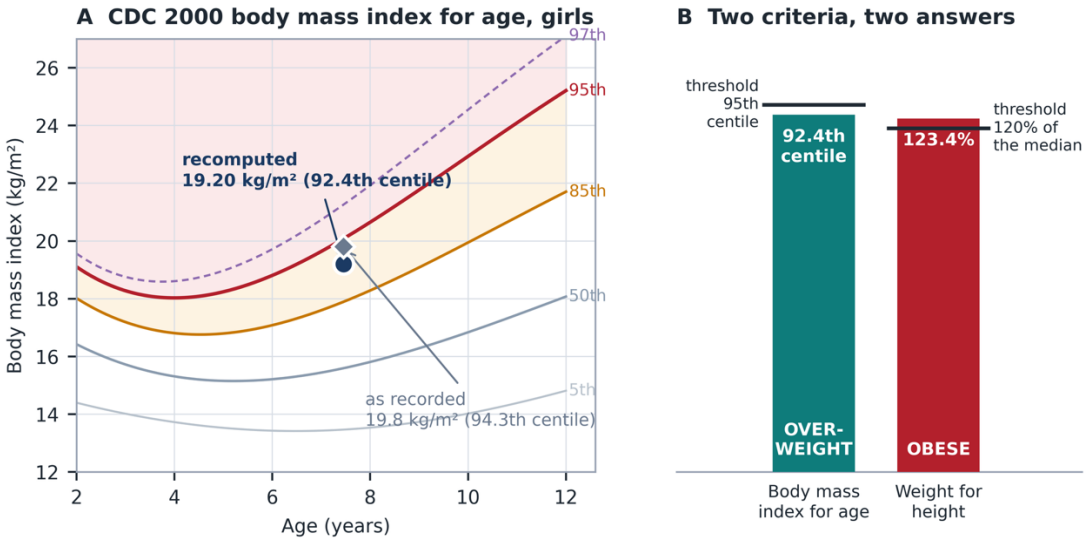

Figure 3. Nutritional classification. ***(A)*** *Centers for Disease Control and Prevention 2000 body-mass-index-for-age curves for girls, with the recomputed value of 19.20 kg/m² (circle) and the value printed in the record, 19.8 kg/m² (diamond). Both lie inside the amber 85th-to-95th centile band and below the red 95th centile curve.* ***(B)*** *The same child by the two criteria her record uses: the 92.4th centile of body mass index for age, which is below the 95th-centile threshold, and 123.4% of the median weight for height, which is above the 120% threshold. Bar heights are scaled to each panel's own axis and are not comparable between the two bars.*

The other anthropometric statements in the record reproduce, with one exception. Weight for age is 124.9% of the median against 125% recorded; height for age is 100.5% against 101% recorded, and 100.5 rounds to 100 rather than to 101, so that one figure does not reproduce; and the height age at which the median height equals 125 cm is 7.56 years, close to the 7 years recorded. The median weight at that height age, which is the ideal body weight, is 24.31 kg against the 24 kg recorded. Weight for height is therefore 123.4% of the median on the recomputed ideal weight and 125% on the recorded one. Both exceed the 120% threshold that Indonesian paediatric practice uses to define obesity by the Waterlow convention. **The two criteria therefore give two different answers in the same child on the same day: overweight by body mass index for age, obese by weight for height.** Figure 3B places the two side by side, and the last two rows of Table 1 state the classification each criterion yields. Three things must be said about the strength of this finding, none of which the record allows us to avoid. **First, the margin.** The recomputed index falls 0.88 kg/m², or 0.21 of a z score, short of the 95th centile. That gap would be closed by 1.38 kg more weight at the same height, or by a height 2.78 cm lower at the same weight – neither of which a single unduplicated measurement excludes. **Second, and against that, the finding survives the printed precision of its own inputs.** Taking the weight as 30 kg to the nearest kilogram and the height as 125 cm to the nearest centimetre, the index lies between 18.73 and 19.68 kg/m² and the centile between the 90.3th and the 93.9th. The whole of that range lies below the 95th centile, so the classification does not turn on rounding, even though the point estimate of 92.4th is more precise than the measurements justify. **Third, the other body-mass-index reference agrees.** On the World Health Organization 2007 reference for 5 to 19 years,[27] which sets overweight at a z score above +1 and obesity above +2, her index gives a z score of +1.69, the 95.4th centile, against an obesity threshold of 20.12 kg/m²: overweight again. **The obesity label is not wrong under Indonesian convention, which uses weight for height as well as body mass index;[28] what fails is the sentence that justifies it, which cites the criterion the child does not meet.**

Why the two criteria disagree, and why the disagreement is not academic

The disagreement is not an artefact of this record. Body mass index for age and weight for height are different constructions: the first compares a child with children of her own age, the second with children of her own height. In this child, however, the two constructions do not in fact place her very differently. Her stature sits close to the median for her age, at the 54.3th centile, so the two references compare her with almost the same children: she is at the 92.4th centile of body mass index for age and at the 86.9th centile of weight for height. **What differs is not where the two scales put her but where the two cut-points are set.** The 95th centile of body mass index for age is, by construction, the 95th centile; 120% of the median weight for height, which is 29.2 kg at her height age, falls at the 83.8th centile of the same reference. She lies between the two thresholds. The disagreement in this child is therefore not a deep difference between two ways of describing a body; it is the arithmetic consequence of two conventions that draw their line at different strictness, and it would arise in every child whose position falls between them. When 4151 Brazilian schoolchildren aged six to ten years were classified simultaneously by the World Health Organization, International Obesity Task Force, Centers for Disease Control and Prevention and two national criteria, a locally derived criterion agreed closely with the World Health Organization excess-weight criterion, at a kappa of 0.895 with a sensitivity of 0.8680 and a specificity of 0.9956, but the pairwise agreement was not uniform across the five, and all of it is agreement between indices, measured against another index rather than against any measure of body fat.[17] Comparing three international references in 22,737 Brazilian schoolchildren, the World Health Organization obesity percentile performed less well than a multicentric alternative in boys, with a positive likelihood ratio of 6.99 against 10.99, 8.99 and 8.09 at the comparator's three anchor ages, for the same negative likelihood ratio of 0.04, an advantage the study reports for boys only.[18] Even within a single system the choice of reference changes the answer: transitioning between the World Health Organization and the Centers for Disease Control and Prevention charts at two years of age produced a mean fall in body-mass-index-for-age z score of 0.59, with 28.3% of 7429 children recording a fall of more than a whole z score, a proportion that falls to 11.6% when the transition is smoothed.[29] A national reference constructed from a Swedish birth cohort deliberately excluded 145 children with extreme body mass index, and its normal range is narrower than the World Health Organization standard on both the weight and the weight-for-height scales.[30] The recent reframing of obesity into preclinical and clinical categories illustrates the same point from the other direction: in 5513 United States youth aged 5 to 18 years, the combined prevalence of 20.8% under the new definition was similar to the roughly 22% obtained from the body mass index percentile alone, but the individuals so classified are not the same set, which is the same phenomenon that the two classification rows of Table 1 show in a single child, and that Figure 3B shows graphically.[31]

Three studies measure that disagreement directly, and each measures a different face of it. In 965 Korean adolescents screened simultaneously by the three criteria that matter here, body mass index above the 85th centile classified 188 children as overweight, weight for height above the 85th centile classified 139 and weight above 120% of the ideal for height classified 115; agreement was substantial but incomplete, with a kappa of 0.798 between the first two and 0.710 between the first and the third, so the three criteria identify three overlapping but different sets of children.[32] In 7,863,520 Japanese schoolchildren the bias has a direction predictable from stature alone: under a body-mass-index criterion, children in the lowest quartile of height for their age were classified as overweight four to five times more often than children of the same age in the highest quartile, whereas a percentage-of-median criterion was less height-dependent, the difference between height classes staying below three-fold.[33] And in 11,134 Irish children the prevalence of overweight or obesity ran from 11% to 40% at 9 months, 14% to 46% at 3 years and 8% to 32% at 5 years according only to which growth criterion was applied.[34] **A three-to-fourfold range in a population prevalence produced by nothing but the choice of criterion is the population-scale shadow of what happened to this one child.**

Why does this matter for a child with dengue? Because the literature that would be invoked to justify closer monitoring uses whichever definition the study chose. The Nicaraguan cohort's adjusted odds ratio of 1.59 for symptomatic dengue among the infected applies to children whose obesity classification that report does not specify.[7] The Sri Lankan hospitalisation odds ratio of 2.33 applies to children above the 97th World Health Organization centile.[8] The Vietnamese odds ratio of 2.3 for mechanical ventilation applies to children classified on World Health Organization growth charts.[9] The Thai paediatric series, which found higher rates of overweight among children with severe plasma leakage

than with mild leakage (45.5% against 18.8%) but no overall difference in nutritional status across severity categories, used World Health Organization body mass index for age as well.[14] A prospective Malaysian adult cohort reported an adjusted odds ratio of 1.17 per unit of body mass index (95% confidence interval 1.04 to 1.34) for a composite of warning signs or severe disease.[35] This child would be inside the exposed group under the Indonesian weight-for-height convention and outside it under every body-mass-index-based definition in that list. She cannot be both, and the record does not say which one it means. **The practical consequence is that the sentence 'this child is obese' carries no information unless the criterion is named.** The two answers are illustrated in Figure 3.

Two further points follow. First, an adiposity measure other than weight and height would have narrowed the ambiguity, and none was taken. Body mass index is an index of weight for height rather than a measurement of body fat, and the comparison in 22,737 schoolchildren evaluated the obesity percentiles against the 95th percentile of the sum of triceps and subscapular skinfolds rather than against any criterion measure of body fat.[18] Second, the label was not inert: it entered the problem list as a diagnosis with an International Classification of Diseases code, it generated a nutritional care plan and an energy prescription, and it was the stated reason in the case analysis for describing her general condition as severe. A label that does more work than its evidence supports is worth correcting even when, as here, it made no difference to the outcome. Table 1 is therefore set out so that each recorded index can be read against its recomputed counterpart.

Every weight-indexed decision, and the weight behind it

Once nutritional status is set aside, a second and more consequential question remains. In this child, 9 distinct classes of decision were indexed to a body weight: the intravenous fluid rate, the paracetamol dose, the omeprazole dose, the tranexamic acid dose, the ceftriaxone dose, the reported urine output, the energy prescription, the fluid requirement written on the nutritional care form and the basal metabolic rate from which that form derived her energy expenditure. Table 4 sets each of them out with the quotient it gives on the measured weight of 30 kg and on the recorded ideal weight of 24 kg; the recomputed ideal weight of 24.31 kg differs from the recorded one by too little to change any of them. **The body weight actually used is not stated anywhere in the record, for any of them.**

It can be recovered to within an interval, but only on a premise that must be stated before the result rather than after it. **The premise is that each per-kilogram figure printed in the chart is a rounded record of a quotient that was actually computed, and not a target read off a protocol card.** The premise is not certain, and the evidence cuts both ways: 10 mg/kg, 1 mg/kg and 10 mg/kg are the textbook doses of paracetamol, omeprazole and tranexamic acid, and a ladder of 4, 3, 2 and 0.6 mL/kg/h is close to the shape of a printed de-escalation ladder, both of which favour the protocol reading; while a chart that prints three of the four ladder figures as integers and the fourth to one decimal place is behaving like something calculated rather than something copied. **If the annotations are protocol targets, the weight used is simply unrecoverable and everything that follows in this section falls away, leaving the same practical conclusion by a shorter route.** On the premise that they are rounded quotients, a per-kilogram figure printed to a stated number of decimal places constrains the weight that produced it. If a chart prints a rate as 4 mL/kg/h, the underlying value lies between 3.5 and 4.5, so an order of 100 mL/h implies a weight between 22.2 and 28.6 kg. Applying that reasoning to each of the 4 labelled steps of the fluid ladder and intersecting the results gives a single window of 24.6 to 28.0 kg. **That window contains neither the measured weight of 30 kg nor the recorded ideal weight of 24 kg.** Taken step by step rather than jointly, 3 of the 4 labels are reproduced by the recorded ideal weight and only 1 by the measured weight, so the ladder was evidently built on something close to the ideal weight, but not on the ideal weight as the record states it. Table 4 shows the two quotients for every step.

The same arithmetic applied to the three drug orders that carry a per-kilogram annotation produces the intervals drawn in Figure 4. Paracetamol 250 mg annotated as 10 mg/kg/dose implies a weight between 23.8 and 26.3 kg. Omeprazole 30 mg annotated as 1 mg/kg/dose implies a weight between 20.0 and 60.0 kg, an interval so wide that it constrains almost nothing. Tranexamic acid 200 mg annotated as 10 mg/kg/dose implies a weight between 19.0 and 21.1 kg. **The tranexamic acid interval and the paracetamol interval do not overlap, and the tranexamic acid interval does not overlap the fluid ladder window either. It follows that no single body weight, whatever it might be, reproduces every per-kilogram figure this chart prints.** The measured weight of 30 kg satisfies 1 of the 4 annotated groups and the recorded ideal weight of 24 kg satisfies 2 of 4. One caution about what these intervals are: they bound the weights consistent with a correctly computed and correctly rounded annotation, not the child's weight, which was measured once and is 30 kg. **There is a shorter and stronger form of the same result which does not require any weight to be named at all.** Paracetamol 250 mg and tranexamic acid 200 mg carry the same annotation, 10 mg/kg/dose. Two doses annotated to the same integer per-kilogram figure P can be quotients of one weight only if they differ by no more than (P + 0.5) divided by (P – 0.5), which at P = 10 is 1.105. These two doses differ by 250 divided by 200, which is 1.25. **The contradiction therefore follows from the two doses and the one annotation alone, without reference to any body weight, and it is what makes this half of the argument robust.** It is not, however, robust to very much: the ratio exceeds its limit by 13.1%, so a tranexamic acid dose of 226 mg or a paracetamol dose of 220 mg would remove it. The fluid-ladder half is more fragile still. Its lower bound of 24.6 kg is set by one label alone, the 16 mL/h step annotated 0.6 mL/kg/h; on the recorded ideal weight of 24 kg that step is 0.667 mL/kg/h, which rounds to 0.7 and truncates to 0.6. **If that one figure was truncated rather than rounded, the ladder window becomes 23.2 to 24.6 kg, which does contain the recorded ideal weight, although it still excludes the measured weight.** The two halves of this section are therefore of very different strength, and they should be read that way.

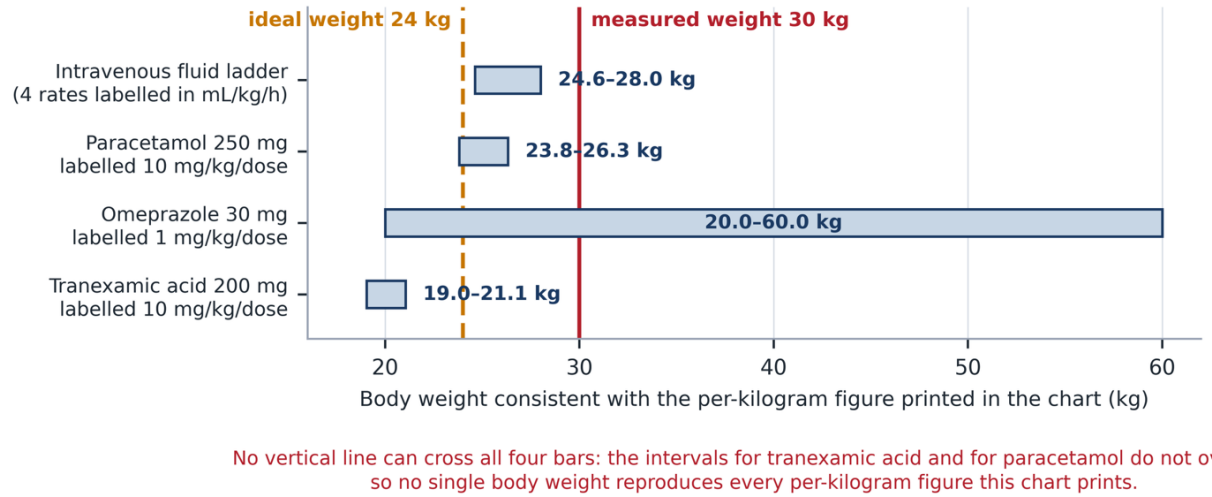

Figure 4. *The body weights consistent with each per-kilogram figure printed in the chart. Each bar is the interval of weights that could have produced the printed figure at the precision at which it is written; the fluid-ladder bar is the intersection of the intervals implied by its 4 labelled steps. The solid red line marks the measured weight of 30 kg and the dashed amber line the ideal weight of 24 kg recorded on the same chart. The tranexamic acid and paracetamol intervals do not overlap, so no vertical line can cross all four bars.*

Which weight should have been used is a separate and more difficult question, and the literature does not give one answer for every agent, and it does not allow the answer to be borrowed from adults. In a population analysis of 125 overweight and obese adolescents weighing 28.3 to 188 kg together with 81 overweight and obese adults, vancomycin clearance rose with standard weight along a different function in each group, adolescents clearing more per unit of standard weight than adults and adolescent males 21% more than adolescent females of the same standard weight, so the adult regimen cannot simply be carried across.[36] For dexamethasone, a population model built on 82 plasma samples from 59 children with obesity retained total body weight as its only covariate and placed 68% or more of a modelled population of 1000 children within the exposure range reported in adults at 0.5 and 1 mg/kg.[37] For enoxaparin, a population model built on 552 anti-Xa samples from 196 encounters in 160 children found that allometrically scaled fat-free mass with an exponent of 0.712 was the most influential covariate on clearance, and modelled fat-free-mass dosing reached the target in 77.5% of children with obesity against 65.2% for total-body-weight dosing.[38] For proton pump inhibitors the direction is the same: obesity was associated with an estimated 18% reduction in pantoprazole clearance after accounting for total body weight and CYP2C19 phenotype in a study of 39 children and young adults,[39] and a physiologically based model comparing 1 mg/kg total body weight, 1.2 mg/kg lean body weight and the regulatory weight-tiered regimen found that the tiered regimen placed more than 90% of children in the reference exposure range irrespective of obesity status or phenotype.[40] For vancomycin, a multicentre cohort of 1099 children found serum concentrations higher in children with overweight or obesity than in normal-weight children, although 75% of the whole cohort were still subtherapeutic on trough and 63% on the area-under-the-curve target.[41] For ceftriaxone, a population model of total and free drug in critically ill children gave a free clearance of 6.54 L/h/70 kg and modelling that supports 50 mg/kg every 12 hours to maintain free concentrations above 1 µg/mL throughout the interval, with allometric weight scaling as a significant covariate.[42] None of these is a licence to pick a weight silently. They are arguments for naming the weight and the rationale in the order itself, which is precisely the field that is missing from every row of Table 4. The intervals these orders imply are set out in Figure 4.

Against those recommendations, three of the orders in this chart are worth stating explicitly. The intravenous rate of 110 mL/h corresponds to 3.67 mL/kg/h on the measured weight and 4.58 mL/kg/h on the recorded ideal weight, against maintenance, which is 2.74 mL/kg/h on the recorded ideal weight and 2.36 mL/kg/h on the measured weight, so the opening rate was 1.67 times maintenance on the ideal weight and 1.55 times maintenance on the measured weight. It was never increased. Tranexamic acid 200 mg is 6.67 mg/kg on the measured weight and 8.33 mg/kg on the recorded ideal weight, so the annotation of 10 mg/kg/dose overstates the delivered dose on every candidate weight. Ceftriaxone 1200 mg twice daily is 80 mg/kg/day on the measured weight and 100 mg/kg/day on the recorded ideal weight. Neither figure is unusual for a child being treated for a bacterial infection: population modelling in 45 critically ill children found 100 mg/kg once daily adequate for most of them,[43] 80 mg/kg is a dose prescribed routinely enough in a United Kingdom paediatric ambulatory service for its rate of infusion to have been the subject of a service evaluation,[44] and the modelling of free ceftriaxone quoted above supports 50 mg/kg every 12 hours in critically ill children.[42] What cannot be defended here is the indication, not the size of the dose. What differs between agents is the direction in which the delivered quotient departs from the per-kilogram figure the chart prints – overstated for tranexamic acid, reproduced for omeprazole, understated for paracetamol – and that is precisely what an undeclared denominator produces. Table 4 records the two quotients against the printed figure for each of them. Figure 4 presents the same result as a set of intervals rather than as a list of quotients.

The silence about the denominator is the general case rather than this hospital's lapse, and the two studies that quantify it also mark its limit. Documenting a weight helps, but only a little: among 3618 weight-based medication administrations by one statewide service, the dosing-error rate was 22% where a weight had been documented against 26% where it had not, a difference that reached significance for epinephrine (34% against 56%) and fentanyl (10% against 31%) and, among the age groups, only for infants.[45] Issuing a dosing reference helps no more reliably: in a statewide review of 1078 paediatric medication administrations after such a reference was introduced, 380 (35.2%, 95% confidence interval 25.3 to 48.4) still deviated by more than 20% from the weight-appropriate dose, with rates from 3.6% for albuterol to 68.5% for intravenous

fentanyl.[46] Both studies measure the accuracy of a dose given a weight that was at least chosen. Neither addresses the situation in this chart, where no weight was written against any order and the denominator can be recovered only by arithmetic from the annotations it produced.

The urine output cannot be converted back into a volume

Urine output was recorded on three consecutive days as 0.7, 1.1 and 1.2 mL/kg/h, and the weight used to compute those figures is not stated. On the measured weight of 30 kg the corresponding 24-hour volumes are 504, 792 and 864 mL; on the recorded ideal weight of 24 kg they are 403, 634 and 691 mL. The two readings of the same record differ by 20%. Since urine output is one of the few bedside measurements that speaks directly to perfusion in a child who is not shocked, and since the conventional adequacy threshold in a school-aged child is expressed per kilogram, the ambiguity is not trivial: a figure of 0.7 mL/kg/h read on the larger denominator looks like relative oliguria and could prompt an increase in fluid, whereas the same measured volume read on the ideal weight is closer to the conventional target. In dengue, unnecessary escalation is the principal iatrogenic hazard, and the observational evidence points in one direction, although the first of the three estimates below is subgroup-specific and does not survive adjustment for multiplicity: in 4781 hospitalised adults, an intravenous volume above 2000 mL/day was associated with a higher risk of severe dengue among those with warning signs (hazard ratio 1.77, 95% confidence interval 1.05 to 2.97) while a volume below 1000 mL/day was protective in several subgroups.[47] In 1278 children with dengue shock syndrome the total intravenous volume predicted mechanical ventilation with an area under the curve of 0.871 (95% confidence interval 0.836 to 0.905) and an optimal cut-off of 181 mL/kg.[48] In a multicentre review of 691 children with dengue shock syndrome, a mixed crystalloid-colloid initial regimen was associated with more respiratory compromise (hazard ratio 2.08) and more need for ventilatory support (odds ratio 2.34) than crystalloid alone.[49] None of these studies states the weight it used either, and the cut-off of 181 mL/kg inherits exactly the same ambiguity when it is applied to a child with excess weight. The three recorded outputs are tabulated in Table 5. The point generalises beyond volumes: in 60 children with dengue shock syndrome admitted to a Bangkok intensive care unit, 26 (43.3%) developed acute respiratory failure, fluid accumulation over the first 72 hours was greater in that group (12.2% against 8.3%, p = 0.009) and accumulation above 15% was independently associated with respiratory failure (adjusted odds ratio 5.67, 95% confidence interval 1.24 to 25.89, p = 0.025).[50] Percentage fluid accumulation is itself a quotient whose denominator is a body weight, so the threshold that identifies the child at risk of respiratory failure inherits the same ambiguity as everything else on this chart.

What this child's record does show is that the fluid strategy worked. She received no bolus, no colloid and no blood product; the daily net balance fell from +800 mL to +53 mL and then to +5 mL over three days while the haematocrit returned from 48% to 36%; and no clinical sign of fluid overload was recorded. The quota chosen at admission was maintenance plus a 5% deficit, the same quota whose adequacy has been studied prospectively in dengue haemorrhagic fever: of 115 adults followed through the critical phase, 80 (69.6%) received fluid in excess of that quota, and the group that exceeded it had a higher body mass index than the group that did not (22.75 against 20.76, p = 0.03).[51] This child was not among those who exceeded the quota. Maintenance by the Holliday-Segar rule, which was derived from measured energy expenditure and takes total body weight as its argument,[52] is 1580 mL/day, or 65.8 mL/h, on the recorded ideal weight and 1700 mL/day on the measured weight; the opening rate of 110 mL/h is therefore about 1.7 times ideal-weight maintenance, which is what a 5% deficit is meant to be, and the rate first fell below ideal-weight maintenance at the 58 mL/h step, 42.1 hours after arrival. The written daily totals of 1580 mL on hospital day 2 and 1000 mL on hospital day 3 are lower still. Using the ideal rather than the measured weight avoided 120 mL of maintenance fluid a day in a child whose principal risk after the leak reverses is reabsorption and overload. The falling balances are detailed in Table 5, and Figure 2D reads the same order on the two candidate denominators alongside the figure the chart itself prints. One caution belongs here: integrating the hourly rates documented for hospital day 2 gives about 2004 mL, against the 1580 mL written as that day's total on the same chart, a gap of roughly 424 mL. The two entries cannot both be right, and the coincidence between the written total and ideal-weight maintenance should therefore not be read as evidence that the total was calculated that way.

An arithmetic error in the energy prescription

The nutritional care form calculates basal metabolic rate from the weight-and-height equation for girls aged 3 to 10 years, published in megajoules with height in metres and in general use in kilocalories with height in centimetres,[53] which in kilocalories per day is 16.969 × weight in kilograms + 1.618 × height in centimetres + 371.2. The form records 882 kcal/day. Substituting her measurements with the height in centimetres gives 16.969 × 30 + 1.618 × 125 + 371.2 = 1082.5 kcal/day. Substituting the height in metres instead gives 882.3 kcal/day, which is the figure the form records. The height was entered in metres into a coefficient expressed per centimetre, and the resulting basal metabolic rate is 200 kcal/day, or 18.5% of the correct value, below the value the equation gives. The reconstruction is as certain as a reconstruction can be: substituting the height in metres reproduces the figure printed on the form to within 0.29 kcal, or 0.033%, and no other plausible combination of equation and inputs gives 882 kcal/day. It should be said at once how far the error travelled: the energy actually prescribed was 1440 kcal/day, computed as ideal body weight times the allowance for height age and not from the metabolic rate at all, so the error changed one descriptive percentage on one form and nothing that the child received. Because the stress and activity factors were both set to 1.0, the same error propagates directly into the total energy expenditure and into the adequacy statement built on it: an inpatient intake of 600 kcal/day was recorded as 68% of total energy expenditure, and against the corrected value it is 55.4%. The parallel statement against the prescribed allowance, 41.7% of 1440 kcal/day, is unaffected because it does not use the equation.

Two things make this worth reporting rather than merely correcting. The first is that the error is invisible on inspection: 882 kcal/day is a plausible number for a 30 kg child and nothing in the form flags it. It becomes visible only when the equation is re-evaluated, which is why the recomputation of every printed derived quantity belongs in the routine audit of a nutritional care plan. The second is that predictive equations perform badly in exactly this population, so an error of this size sits on top of a method that is already imprecise. In 275 children and adolescents aged 6 to 18 years, the best-performing equation reached only 44.9% accuracy in the participants with obesity, and the smallest root mean squared error in that subgroup was 305.4 kcal/day against 136.2 kcal/day in the normal-weight participants.[54] In 109

hospitalised children the Schofield equation had the highest overall accuracy at 47.7%, and on Bland-Altman analysis every equation deviated from measured expenditure by more than 10% in every nutritional category.[55] In 556 adolescents with severe obesity, an equation derived specifically for them explained under half the variance in measured resting energy expenditure.[56] A study of children and adolescents with obesity found that only 60.6% were normometabolic by agreement with a single predictive equation, 25.5% being classified hypermetabolic and 13.9% hypometabolic.[57] Even an equation built and validated inside the population it is meant for carries a wide error: new equations derived in 184 boys and validated in 148 others gave mean errors of 51 ± 199 and 39 ± 193 kcal/day, biases of 4.7% and 3.9%, and accuracies of 61.2% and 66.2%.[58] The clinical implication is not that the equation should be abandoned but that its output should be treated as a starting estimate that is re-derived and re-checked, and never as a number to be quoted to three significant figures in a percentage of adequacy.

The same form contains a quieter inconsistency of denominator. Its energy prescription uses the ideal body weight, 24 kg × 60 kcal/kg/day, which is the correct method for a child with excess weight because it avoids feeding to sustain the excess. Its fluid requirement, on the same page, uses the measured weight: 30 kg × 80 mL/kg/day = 2400 mL/day, where the ideal weight would give 1920 mL/day. That figure also sits well above what the ward was actually prescribing on the same day, 1000 mL, so on hospital day 3 the chart carried a fluid requirement of 2400 mL/day on one form and a prescription of 1000 mL/day on another, and the admission order of 2700 mL/24 h had been a third figure two days earlier. One form, two denominators, and a number that no one acted on.

Haemoconcentration measured against an undeclared baseline

The haematocrit criterion for plasma leakage is expressed as a relative rise, and a relative rise requires a baseline. This child's peak haematocrit of 48% is a rise of 29.7% against the value drawn eleven hours earlier at the same institution, and 33.3% against both the referring hospital's value and her own convalescent value. The three candidate baselines converge, so in her case the conclusion is robust: the rise exceeds 20% however it is anchored. That convergence is worth stating because it is not the usual situation. A prospective Sri Lankan study that measured serial haematocrit with thrice-daily ultrasound in 43 patients found a mean haematocrit of 40.6% at baseline and 41.3% at the onset of the critical phase, with no significant difference, and **none of the participants reached a 20% rise at any point during the critical phase, the maximum observed being 17.0%.**[59] Those patients were already hospitalised and receiving fluid, which blunts haemoconcentration, and they were not children; but the finding shows how often the criterion fails when a true pre-illness baseline is unavailable, and it makes the present case unusual rather than typical. A laboratory study of 1141 blood counts from 163 adults with dengue adds a measurement caveat that applies directly here: the relationship between haemoglobin and haematocrit weakens as the red cell distribution width rises, the coefficient of determination falling from 0.92 to 0.78 above a width of 18, and the difference between calculated and analysed haematocrit is greatest at haemoglobin values below 12 g/dL and at 16 g/dL or above.[60] This child's peak haemoglobin was 16.1 g/dL, within the range where that discrepancy is largest. The three candidate baselines are all listed in Table 2 and the two curves are illustrated in Figure 2A.

Two features of her record deserve comment. First, the haemoglobin rose in parallel with the haematocrit, by 3.1 g/dL or 23.8%, and each of the three values above 15 g/dL was annotated as normal while the paired haematocrit on the same report was annotated as raised. Both cannot be right. The haemoglobin was in fact the more sensitive index of the same event, because it was measured directly rather than derived. Second, no weight was measured after admission. In a child in whom peripheral oedema is harder to see, serial weight is the cheapest available measure of net fluid accumulation during the reabsorption phase, and it was not obtained even once in five days. Table 2 records the paired values and Figure 2A plots both curves.

Plasma leakage was inferred and never imaged

The diagnosis of dengue with warning signs in this child rested on abdominal pain with epigastric tenderness, mucosal bleeding, and a rising haematocrit with a rapidly falling platelet count. One other warning sign was never sought by imaging: clinical fluid accumulation, which carries a negative predictive value of 69.3% in children in the Jakarta paediatric series. Hepatomegaly, for which that series reports a negative predictive value of 80.8% in infants and none for the age group this child belongs to,[3] was assessed by palpation and was absent. **No abdominal or chest ultrasonography and no chest radiograph were performed at any point in this admission.** In 132 Colombian children aged 6 months to 14 years with dengue with warning signs or severe dengue, ultrasound identified capillary leakage in 95.5%, comprising ascites in 83.3%, pleural effusion in 46.2%, hepatomegaly in 40.9% and gallbladder wall thickening in 39.4%.[61] In an adult emergency department series of 44 patients, gallbladder wall thickening separated severe dengue from dengue with warning signs with 90.5% sensitivity and 69.6% specificity, and identified the need for critical care with 100% sensitivity.[62] A hospital series using initial and follow-up abdominal and chest ultrasound found pericholecystic fluid significantly associated with severe disease and gallbladder wall oedema, ascites and any positive ultrasound finding associated with progression, with odds of 2.74, 2.04 and 2.619 respectively.[63] Lung ultrasound in 50 patients found at least one abnormality in 33 (66%), abnormalities being commoner in the critical and convalescent phase (30 of 41, 73%) than in the febrile phase (3 of 9, 33%, P = 0.047).[64] The omission matters more in a child than it would in an adult, because leakage is the manifestation that dominates paediatric severe dengue: in an emergency-department surveillance cohort of 1089 dengue patients in Puerto Rico, of whom 281 (26%) were severe, severe plasma leakage or shock accounted for 59% of severe disease in those aged 0 to 9 years and 86% in those aged 10 to 19 years, against 49% in adults.[65]

There is an irony worth naming. The single investigation that would have converted an inferred plasma leak into an observed one is ultrasonography, and excess subcutaneous tissue is exactly what makes ultrasonography technically harder. **The child in whom the test is most difficult is the child in whom the diagnosis is most often assumed, and in this record the test was never attempted.** Serum albumin, which is the other cheap surrogate for leakage and which was below 35 g/L in the case-control series from the Thai-Myanmar border with an adjusted odds ratio of 8.10 for severe dengue,[4] was measured once at 3.2 g/dL, eleven hours before the haematocrit peak, and never repeated. In 190 Bangladeshi children with dengue, a reduced serum albumin was present in 44.7%,[66] and in 556 Thai children a serum albumin below 3.5 g/dL was among the factors significantly associated with severe dengue.[5] A second albumin at the

haematocrit peak would have cost very little and would have told the clinicians whether the leak was still widening; the single value obtained is listed in Table 3.

Aminotransferases, electrolytes and calcium

The hepatic profile in this child was typical of dengue and was not severe. Aspartate aminotransferase of 125 U/L and alanine aminotransferase of 36 U/L give a ratio of 3.47, the aspartate-predominant pattern characteristic of the illness, at 3.4 and 1.4 times the respective laboratory upper limits. In 230 Vietnamese children an aspartate aminotransferase cut-off of 120 U/L predicted severe dengue with an area under the curve of 0.93 (95% confidence interval 0.90 to 0.96), 82.5% sensitivity and 87.3% specificity,[67] and in 304 Indonesian children alanine aminotransferase reached an area under the curve of 0.894, with 73% sensitivity and 90% specificity at a cut-off of 52 U/L.[68] In 325 Nepalese patients elevated aspartate aminotransferase carried an adjusted odds ratio of 2.40 (95% confidence interval 1.49 to 3.86) for dengue with warning signs, while elevated alanine aminotransferase did not.[69] This child's aspartate aminotransferase sits just above the Vietnamese cut-off and her alanine aminotransferase well below the Indonesian one, a discordance that is itself instructive about single-cut-off rules; both values were measured once and never repeated, so the peak is unknown. Adults with central obesity in a Sri Lankan cohort of 577 had higher aminotransferases, C-reactive protein and ferritin than leaner patients, though the same study found no association between oxidative stress markers and clinical severity.[12]

The electrolyte pattern was mild and multifactorial: sodium 133 mmol/L, potassium 3.3 mmol/L and chloride 99 mmol/L in a child with three days of watery diarrhoea, reduced oral intake and an active plasma leak. The corrected calcium of 8.24 mg/dL is the one value worth pursuing further. In 210 adults with dengue, mean corrected calcium fell across the severity groups from 8.39 ± 0.59 to 8.05 ± 0.62 to 7.61 ± 0.67 mg/dL ($P < 0.001$), hypocalcaemia below 8.5 mg/dL was present in 91.3% of severe cases, and hypocalcaemia independently predicted severe dengue with an adjusted odds ratio of 3.94 (95% confidence interval 1.98 to 7.84).[70] That study is in adults and its group sizes are not published, so it cannot be transferred to a 7-year-old without caution; but this child's value of 8.24 mg/dL falls between the means reported for the two least severe of those three groups, 8.05 and 8.39 mg/dL, and it was measured once and never repeated. The activated partial thromboplastin time of 41.6 seconds was annotated in the record as 1.5 times raised, which implies a comparator of 27.7 seconds that the record does not print; the prothrombin time was normal, and no fibrinogen or D-dimer was measured, so the coagulopathy was characterised on a single parameter. Table 3 presents the full electrolyte, coagulation and hepatic panel.

Three agents, and three different questions about each

Tranexamic acid was started at the referring hospital before the single bleeding episode and was continued at the referral centre until discharge. Its evidence base in gastrointestinal bleeding is one of the clearer negative results in modern therapeutics. In 12,009 adults randomised to tranexamic acid or placebo for significant gastrointestinal bleeding, death due to bleeding within five days occurred in 222 of 5994 (3.7%) against 226 of 6015 (3.8%), a risk ratio of 0.99 (95% confidence interval 0.82 to 1.18); venous thromboembolic events were more frequent with tranexamic acid (0.8% against 0.4%, risk ratio 1.85, 95% confidence interval 1.15 to 2.98) as were seizures (0.6% against 0.4%, risk ratio 1.73, 95% confidence interval 1.03 to 2.93).[71] A subsequent randomised trial in 600 adults with advanced cirrhosis and upper gastrointestinal bleeding did find less failure to control bleeding by day 5 with tranexamic acid, 19 of 300 (6.3%) against 40 of 300 (13.3%), $P = 0.006$, but with no difference in five-day or six-week mortality.[72] Neither trial enrolled children and neither concerns dengue, in which the platelet count falls as viraemia rises.[73] No fibrinolytic abnormality was demonstrated in this child: her prothrombin time was normal, her activated partial thromboplastin time was prolonged at 41.6 seconds, and neither fibrinogen nor D-dimer was measured, so the coagulation profile was never characterised well enough to identify a target for the drug. She bled on illness day 3, when the platelet count recorded at the referring hospital was still 195 × 10^3/mm^3. We are not aware of any trial evidence supporting tranexamic acid in paediatric dengue bleeding, the extrapolation available from adults is null or harmful in the non-cirrhotic setting, and in this child the delivered dose was between 6.67 and 8.33 mg/kg on the two candidate weights, and on neither of them the 10 mg/kg the chart claims. The bleeding was a single episode, and it occurred while she was already receiving tranexamic acid at the referring hospital, so it cannot be read as evidence that the drug worked; the platelet count went on falling for a further 33.1 hours after her arrival.

Ceftriaxone raises a different question. It was begun at the referring hospital at 600 mg twice daily and increased to 1200 mg twice daily at the referral centre, where it was continued to discharge, without a recorded bacterial indication, without a blood culture and with a stool culture that was planned but never reported. The increase deserves to be described accurately: 600 mg twice daily is 40 mg/kg/day on the measured weight, below every regimen the pharmacokinetic literature cited below supports, so if the drug was to be continued at all the referral team corrected an underdose rather than escalating unnecessarily. The case against a bacterial indication is not one-sided and should be put both ways. Against it: the leucocyte count was 2.60 × 10^3/mm^3 at the referring hospital and 3.88 × 10^3/mm^3 at its nadir here, the leukopenia characteristic of acute dengue; the later rise to 13.58 × 10^3/mm^3 on illness day 6 is the convalescent leucocytosis of the same illness; and the illness ran the course of dengue and no other. For it: the admission differential showed 12% band forms against a reference upper limit of 5%, a marked left shift, in a child with six to eight watery stools a day, abdominal pain, gastrointestinal bleeding and two documented episodes of typhoid fever, the most recent two months earlier, in a typhoid-endemic city. That is a defensible reason to start an antibiotic on illness day 2 and it is very probably why one was started. It is not a reason to continue one for six days without a culture. Antibiotic use in dengue is common and largely empirical: in 7086 Taiwanese children with confirmed dengue, 29.4% of those without a concomitant bacterial infection were prescribed antibiotics within the 14-day assessment period, with prescribing falling from 43.2% to 19.3% among inpatients once dengue was confirmed, and younger children more likely to receive them.[74] In 249 hospitalised adults the cumulative incidence of antibiotic use was 9.3%, predominantly empirical, and the only predictor of inappropriate use that study reports is the ward of admission, with an incidence rate ratio of 4.15 (95% confidence interval 1.16 to 14.82) against intensive care, on about 23 exposed patients.[75] Secondary bacterial infection does occur, and in 1597 Colombian children its frequency was 2.4% and 7.3% in two cohorts of children with dengue with warning signs, rising to 30.7% and 38.2% in severe disease, with raised leucocytes, C-reactive protein and interleukin-6 at admission as early indications in children under 48 months.[76] Prolonged

fever beyond illness day 7, which occurred in 11.06% of 253 Colombian children with warning signs or severe dengue, is a further recognised marker.[77] Of those markers, none applied here in a form that would justify treatment: this child was 89 months old and the biomarker signal in the Colombian secondary-infection cohort is confined to children under 48 months, she defervesced on illness day 5, and she had no organ dysfunction. Her leucocyte count did reach 13.58 × 10^3/mm^3 on illness day 6, marginally above the upper reference limit, on the day the erythematous rash of the recovery phase first appeared. No culture was ever sent. The exposure was neither justified in the record nor monitored, and a retrospective audit establishes what was written rather than what was thought. Its size was not the problem: at 80 to 100 mg/kg/day, depending on which weight is used, it is at or below the 100 mg/kg once-daily regimen that population pharmacokinetic work found adequate in critically ill children, a group this child never joined.[43] A defensible dose does not supply a recorded indication, and the indication is what a later reader needs in order to judge whether the exposure should have continued for six days.

Omeprazole is the third agent worth naming, for a subtler reason. Its annotated dose of 1 mg/kg/dose is the only one of the 3 annotated drug orders that the measured weight reproduces, and the modelling evidence suggests that total body weight is the wrong denominator for this class: obesity reduces pantoprazole clearance by an estimated 18%,[39] and a physiologically based model found the fixed weight-tiered regimen superior to either 1 mg/kg total body weight or 1.2 mg/kg lean body weight for keeping children within the reference exposure range.[40] In other words, the one order that used the measured weight consistently is the one for which the measured weight has the weakest justification. This is not a criticism of the prescriber; it is a demonstration that a silent denominator cannot be right by accident.

Secondary infection is the better-supported determinant of severity in this child

On illness day 8 both dengue IgM and IgG were positive. Taken with a positive NS1 antigen on illness day 3, the pattern is consistent with a secondary infection, and secondary infection has a far better-established relationship with severity than nutritional status does. In 220 Indian inpatients, all 22 with secondary dengue had hepatic dysfunction, severe in 9 of them, against 74.5% hepatic dysfunction overall.[78] Cytokine profiling of 64 Indonesian patients in Bali found tumour necrosis factor alpha, interleukin-6 and interleukin-17 higher and interleukin-10 lower in secondary than in primary infection, and the same pattern separated dengue haemorrhagic fever from dengue fever.[79] The relationship is not one-directional, however: a prospective multicentre study of 619 Indian children found that primary infections accounted for more than half of all clinical cases (344 of 619), of severe dengue cases (112 of 202) and of fatalities (5 of 7), although the composition of severe cases is not the risk of severe disease per infection and the denominators needed to convert one into the other are not published.[80] The relevant point for the present case is one of attribution. **This child had at least two candidate determinants of a more severe course operating simultaneously, excess weight and a probable secondary infection, and a single case cannot separate them.** The serological classification itself rests on a single IgM and IgG pair without a ratio, without paired acute and convalescent titres and without serotyping, which is a further limitation of the record rather than of the inference.

The diagnostic performance of the tests that were used is worth stating, since the diagnosis rests on them. A recent evaluation of an NS1 antigen rapid test against polymerase chain reaction in 316 symptomatic patients in Dhaka reported a sensitivity of 96.93% (95% confidence interval 95.03 to 98.83) and a specificity of 99.35%.[81] Older comparisons are less favourable and more variable: in 86 acute febrile sera, NS1 by enzyme-linked immunosorbent assay reached 82.4% sensitivity with 94.3% specificity, NS1 by rapid test 76.5%, and IgM by either method only 27.5% and 17.9% respectively, rising to 82.4% sensitivity and 100% specificity for a combined NS1 and IgM rapid test after threefold serum concentration.[82] A positive NS1 on illness day 3 followed by positive IgM and IgG on illness day 8 is as secure a laboratory diagnosis as this setting permits, and Table 3 records both results with the tests that were not performed.

The course she did not have, and what predicts it in children

It is worth stating plainly what did not happen, because the counterfactual is what makes the monitoring worthwhile. This child never became hypotensive, never had a capillary refill time above 2 seconds, never required oxygen and never needed a fluid bolus. In a Malaysian emergency department series of 254 paediatric dengue presentations, of whom 39 (15.4%) had severe dengue, the features independently associated with severe disease were lethargy, a systolic blood pressure below 90 mmHg, a capillary refill time longer than 2 seconds, ascites and hepatomegaly;[83] three of the five were absent here – her lowest recorded systolic pressure was 95 mmHg, her capillary refill time never exceeded 2 seconds and her liver was not palpable. A fourth, lethargy, was reported by her father as reduced activity, although the compound warning sign of lethargy with restlessness was not documented; and the fifth, ascites, was never looked for by imaging and so cannot be called absent. In 157 Indian children of whom 51 (32.5%) had severe dengue and 5 (3.2%) died, the independent predictors were a fever of 39 °C or above (adjusted odds ratio 4.06, 95% confidence interval 1.60 to 10.27), petechiae (9.91, 3.07 to 32.03), a prothrombin time above 14 seconds (37.21, 1.46 to 946.23) and a platelet count below 50,000/cu mm (12.83, 1.12 to 147.17).[84] Two of those four were present in this child – her peak recorded temperature at home was 41 °C and her platelet count fell to 36 × 10^3/mm^3 – while her prothrombin time was normal at 11.7 seconds and she had no petechiae, although her tourniquet test was positive. The confidence intervals on those two laboratory estimates are so wide that they carry little information about any individual child, and we do not use them as a risk statement. An older Indian series of 170 laboratory-confirmed children classified 30 (17.65%) as dengue fever, 106 (62.35%) as dengue with warning signs and 34 (20.0%) as severe dengue, and reported vomiting, altered sensorium, shock, hepatomegaly, splenomegaly, thrombocytopenia and raised urea and creatinine among the features associated with severe disease.[85] Analysis of 1857 Brazilian children aged 0 to 10 years with severe dengue found respiratory failure, melaena, haematemesis and altered consciousness the commonest manifestations, with 89.6% of those cases hospitalised.[86] This child had two of those four manifestations, and she did not progress; Figure 1 records the whole course from which that judgement is made. A course like hers is common where dengue is common: of 332 children hospitalised at a Vietnamese paediatric hospital over two years, 50 (15.1%) had dengue with warning signs and not one progressed to severe dengue.[10] The corollary is that prediction models built on such cohorts should be carried between settings with care; a model developed on seventeen years of paediatric data from two Thai hospitals reached an area under the receiver-operating-characteristic curve of 0.90 in a general hospital setting and 0.79 in a primary-care

setting without laboratory facilities, and lost performance significantly when trained at one of the two hospitals and tested at the other.[87]

Had she progressed, the Indonesian paediatric data describe what would have followed. In 146 children with dengue shock syndrome admitted to a single Indonesian paediatric intensive care unit, mortality was 5.5%, and percentage fluid overload, shock on admission, disseminated intravascular coagulation and acute kidney injury were independent predictors of death, with disseminated intravascular coagulation (risk ratio 15.26, 95% confidence interval 4.97 to 46.81) and nutritional status (16.47, 3.72 to 72.9) associated with a prolonged stay.[88] In a Yogyakarta cohort of children with dengue shock syndrome, a disseminated intravascular coagulation score of 5 or more was present in 25 of the cohort (73.5%), of whom 9 (36.0%) died, with a median platelet count of 20,500/μL and a median D-dimer of 832.5 ng/mL.[89] A prospective Indian series of 78 children with severe dengue in intensive care reported 20 deaths (25.6%), with a positive fluid balance above 10% at 24 hours as one of the independent predictors of mortality.[90] For the small group who remain refractory, a non-randomised comparison in children with fluid-refractory severe dengue reported survival of 97.1% with albumin added to standard therapy against 77.1% with standard therapy alone (P = 0.043), though with historical controls and unstated group sizes.[91] **Two features of that literature bear directly on the present argument: fluid overload and nutritional status both appear as predictors, and both are quantities that this record expresses per kilogram of an unnamed weight.** In a child who does deteriorate, the ambiguity documented here would propagate straight into the two variables that the intensive-care literature identifies as prognostic.

What this case adds to the obesity-and-dengue literature

The published association between excess weight and dengue severity is real but modest, heterogeneous in its exposure definition, and derived almost entirely from cohorts rather than from individual records. The Nicaraguan cohort's adjusted odds ratios of 1.21 for infection and 1.59 for symptomatic disease are the most robust estimates available;[7] the Sri Lankan survey's odds ratio of 2.33 for hospitalisation applies to a survey population with retrospectively reported hospitalisation;[8] the Vietnamese odds ratio of 2.3 applies to mechanical ventilation among children who already had shock;[9] and the Thai paediatric series found no difference in nutritional status across severity categories overall, the only signal being a higher proportion of overweight among the more severe plasma-leakage grades.[14] Two studies point the other way in children: in Ecuadorian arbovirus surveillance, each additional centimetre of mid-upper-arm circumference was associated with a 43% fall in hospitalisation odds among children, though the interval crossed unity (adjusted odds ratio 0.566, 95% confidence interval 0.252 to 1.019);[92] and a Colombian population survey found waist circumference positively associated with seropositivity in girls, while in adults it was height that was inversely associated with both seropositivity and a reported history of hospitalisation, and waist circumference that was inversely associated with seropositivity alone.[93] In hospitalised Taiwanese adults, obesity was associated with more petechiae, dyspnoea and severe hepatitis and a higher peak haematocrit, **but with no difference in severe dengue or mortality**.[15] A matched case-control study of comorbidities in 468 hospitalised adults found only hypertension independently associated with severe dengue, with no obesity-specific estimate reported.[94] An open-label trial of metformin in 120 patients with dengue and obesity found six cases of dengue shock syndrome in the metformin arm against five in the control arm, with more adverse events on metformin and 42% of the treated arm stopping the drug.[95] Against that literature, Figure 3B records the two answers this child's own record supports.

This case does not add to that evidence about outcome, and we make no claim that it does. The child recovered without shock, without a bolus and without transfusion, on a regimen that never exceeded the maintenance-plus-5%-deficit quota computed on ideal weight and that fell below ideal-weight maintenance within 42.1 hours, and a single favourable course tells us nothing about risk. What the case adds is upstream of the association: it shows that in a real record the exposure variable itself was not securely measured. **If the exposure in a study is 'obesity' and the exposure in the ward is a label whose stated criterion the child's own numbers do not satisfy, then the association and the clinical decision are not about the same thing.** The remedy is unglamorous and entirely within reach: state which reference and which criterion, record the z score or the percentage of the median rather than only the category, and take one measure of adiposity that is not a function of weight and height alone.

A second contribution is methodological. To our knowledge the interval argument used in the weight-indexed decision subsection has not been applied to a clinical record before. It requires nothing beyond the numbers already printed on the chart, it is fully reproducible, and it converts a soft observation that 'the dosing weight is unclear' into a demonstrable statement that no single weight is consistent with the chart. The method generalises to any record in which doses are annotated per kilogram, and it could be automated inside a prescribing system so that the inconsistency is caught at the moment of ordering rather than in a retrospective audit. In this child it would have fired on the tranexamic acid order, which is the shortest and lowest interval illustrated in Figure 4.

Inconsistencies in the source record, declared

Because the argument of this report is an audit of a record, the inconsistencies found in that record are stated here in full rather than silently corrected. **First**, the body mass index is printed as 19.8 kg/m^2 where the recorded weight and height give 19.20 kg/m^2; the printed value corresponds to a height of 123.1 cm. **Second**, the obesity classification is justified by a body mass index above the 95th centile, which neither the printed nor the recomputed value reaches. **Third**, the initial fluid order reads 110 mL/h annotated as 2700 mL over 24 hours, where 110 mL/h for 24 hours is 2640 mL; the corresponding day-1 order reads 100 mL/h annotated as 2500 mL where the product is 2400 mL. **Fourth**, the same initial infusion appears as 110 mL/h in the treatment plan and as 100 mL/h in the narrative case analysis. **Fifth**, the four per-kilogram labels on the fluid ladder are jointly consistent only with a weight between 24.6 and 28.0 kg, which is neither of the two weights the record contains. **Sixth**, tranexamic acid 200 mg is annotated as 10 mg/kg/dose but is 6.67 mg/kg on the measured weight and 8.33 mg/kg on the recorded ideal weight. **Seventh**, the Schofield equation is evaluated with the height in metres in a term whose coefficient is per centimetre, understating basal metabolic rate by 200 kcal/day. **Eighth**, the nutritional care form computes energy on ideal body weight and fluid on measured body weight on the same page. **Ninth**, a leucocyte count of 4.57 × 10^3/mm^3 is annotated as leukopenia although it lies inside the conventional paediatric reference interval used here, by 0.07 × 10^3/mm^3, a margin small enough that the

laboratory's own limit may well place it outside; while three haemoglobin values above 15 g/dL are annotated as normal alongside haematocrit values annotated as raised. **Tenth**, the erythrocyte count is printed as 4.85 × 10^3/mm^3 where the magnitude requires 10^6/mm^3, and the corrected calcium is printed without a unit. **Eleventh**, the nutritional annex records the diagnosis as dengue haemorrhagic fever grade II with warning signs, combining the 1997 grading with the 2009 classification, which are different systems. **Twelfth**, the narrative case analysis states that oedema of both lower limbs was found on physical examination as evidence of plasma leakage, whereas no oedema is documented in the admission examination or in any subsequent daily entry; that statement is not used anywhere in this report. **Thirteenth**, the activated partial thromboplastin time is annotated as 1.5 times raised, which implies a comparator of 27.7 seconds that the record does not print. **Fourteenth**, the daily fluid total written for hospital day 2, 1580 mL, cannot be reconciled with the hourly rates documented for the same day, which integrate to about 2004 mL. **Fifteenth**, the record labels as melaena a stool described in the same document as mixed with dark red blood, which is haematochezia. **Sixteenth**, the referral documentation records four days of fever before admission while the ward's own entry of day VI of fever on 30 January makes the admission day illness day 4, that is the fourth day of fever; the two counts differ by one day and this report uses the ward's. Correcting these does not change the diagnosis, the treatment given or the outcome; declaring them is what makes the rest of this report checkable.

A minimum data set for the child with excess weight and dengue

The audit above converges on a small set of measurements, each of which was either missing or ambiguous in this record and each of which is cheap. They are set out in Table 6 with the moment at which each should be obtained and the reason it is needed. Three of them deserve emphasis. **The dosing weight should appear in the order, not in the clinician's head.** A single field naming the weight and its basis converts every quotient on the chart from an assertion into a calculation that can be checked, and it is the only item on the list that costs nothing at all. **A body weight should be measured every day.** In a child in whom oedema is hard to see, serial weight is the cheapest measure of net fluid accumulation during the reabsorption phase, and it also fixes the denominator problem at source. **Ultrasonography should be performed at defervescence.** It is the only investigation that converts inferred plasma leakage into observed plasma leakage, and the published yield in children with warning signs is high enough that a negative study is itself informative.[61]

Table 6. A proposed minimum data set of 10 items for the child with excess weight and dengue, with the moment at which each should be obtained and the reason it is needed.

Measurement	Moment	Reason
1. The dosing weight, and its basis	In every order that contains a per-kilogram quantity, from admission onwards	Without it no quotient on the chart can be checked; in this record the fluid ladder, the paracetamol order and the tranexamic acid order imply mutually incompatible weights
2. Body weight	On admission and once daily until discharge	The cheapest measure of net fluid accumulation during reabsorption, and the only way to keep the dosing denominator current; no weight was measured after admission here
3. One adiposity measure independent of weight and height, such as waist circumference or skinfold thickness	At the moment the nutritional label is assigned	Body mass index for age and weight for height gave opposite classifications in this child, and neither settles whether she was adipose
4. A declared haematocrit baseline, stated as pre-illness, admission or convalescent	At the first haematocrit, and again at discharge	The 20% criterion for plasma leakage is meaningless without a named baseline, and a prospective series found no patient reaching 20% when none was available
5. Serum albumin, repeated	On admission and again at the haematocrit peak	It is the cheap surrogate for a widening leak; a single admission value cannot show a trend, and only one was taken here
6. Abdominal and chest ultrasonography	At defervescence, and repeated if the haematocrit rises further	The only investigation that converts inferred plasma leakage into observed leakage; excess subcutaneous tissue makes it harder, which is a reason to plan it rather than to omit it
7. Urine output in millilitres, alongside the per-kilogram figure and the weight used	With every fluid-balance entry	A figure in mL/kg/h alone cannot be converted back into a volume; the two readings of this record differ by 20%
8. A named indication, an intended duration, and a culture taken before the first dose, for any antibiotic	At the moment the antibiotic is started	Ceftriaxone was given throughout this admission with no documented indication, no blood culture and a stool culture that was never reported
9. Dengue serotype, and a dated convalescent serology	At diagnosis and at follow-up	Secondary infection is a better-established determinant of severity than body habitus; without serotyping the two cannot be separated in a single case
10. A follow-up weight, height and body-mass-index recheck, with a dietetic referral if the nutritional label stands	At the first outpatient review after discharge	A label that generated a diagnosis, a care plan and an energy prescription during the admission was acted on for five days and then abandoned; this record contains no follow-up of any kind

Every item was missing, ambiguous or unrecoverable in the present record. This table contains no measured value, so no cell is marked red or grey. The first item is the only one that costs nothing. Items 2 to 10 each require a single measurement, a single tube of blood, a single bedside study or a single outpatient appointment. None of the 10 would have altered the management this child received; all 10 would have made the record checkable.

The remaining items follow the same logic. A declared haematocrit baseline, whether pre-illness, admission or convalescent, is what makes the 20% criterion meaningful, and the Sri Lankan series shows how often the criterion fails without one.[59] A second serum albumin at the haematocrit peak costs one tube and answers whether the leak is widening.[4] Urine output recorded in millilitres alongside the per-kilogram figure removes an ambiguity that is otherwise irrecoverable. An adiposity measure independent of weight and height would narrow the nutritional classification that body mass index alone leaves open.[18] A named indication, an intended duration and a culture sent before the first dose would make any antibiotic decision auditable.[74] And a recorded dengue serotype with a dated convalescent

serology would allow the contribution of secondary infection to be separated from that of body habitus in future series. Table 6 presents every item with the moment at which each should be obtained, and items 1, 2 and 7 are the three that this record could not supply at all.

Limitations

This is a single case, described retrospectively from a clinical record that was not created for research, and every observation in it is subject to that constraint. The most important limitation is that **nothing here is evidence about outcome**. The child recovered fully, so no inference can be drawn about whether the undeclared dosing weights or the misapplied nutritional criterion would matter in a child who deteriorated; the argument is about the auditability of the record, not about harm. Second, the interval argument in the weight-indexed decision subsection assumes that each printed per-kilogram figure is a rounded representation of a genuine calculation. If a figure was instead copied from a protocol, typed in error or written from memory, the interval for that order is meaningless, although the wider point that the denominators are unrecoverable stands either way. Third, the anthropometric recomputation depends on a single pair of measurements taken once, and neither the weight nor the height was repeated or measured in duplicate; a height error of 1.9 cm would reconcile the recorded and recomputed body mass index, so we cannot say which of the two is the transcription error. Fourth, the classification depends on which growth reference is used, and we have used the Centers for Disease Control and Prevention 2000 reference because that is the reference the record's own weight-for-age, height-for-age and weight-for-height percentages reproduce; a World Health Organization classification would place her differently again.[29] Fifth, no adiposity measure independent of weight and height exists for this child, so the question of whether she was in fact adipose cannot be settled from the record at all. Sixth, the reference intervals used to mark abnormal values are conventional paediatric intervals, and the two aminotransferase upper limits were inferred from the multipliers the record itself prints rather than read from a laboratory handbook. Seventh, the serological classification of secondary infection rests on a single IgM and IgG pair without a ratio, paired titres or serotyping. Finally, several of the comparator studies cited here are in adults, in different populations and with different definitions of excess weight, and each has been named as such where it is used. None of these limitations affects items 1 and 7 of Table 6, which require only that a weight already known, or a volume already measured, is written down.

5. Conclusion

A 7-year 5-month-old Indonesian girl with dengue with warning signs, one episode of haematemesis and one of melaena, and a haematocrit that rose from 37% to 48% in 11.1 hours with a platelet nadir of 36 × 10^3/mm^3, recovered without shock, without a fluid bolus and without transfusion on a regimen that never exceeded the maintenance-plus-5%-deficit quota computed on ideal body weight and that fell below ideal-weight maintenance within 42.1 hours, and was discharged on illness day 8. She was labelled obese. Recomputing her measurements gives a body mass index of 19.20 kg/m^2, the 92.4th centile, which is overweight; the value printed in the record, 19.8 kg/m^2, is the 94.3th centile and also falls below the 95th centile that the record cites as its criterion. Weight for height of 123.4% of the median does make her obese by the Waterlow convention. **Two criteria, applied to the same two measurements on the same day, give two different answers, and the record names the criterion that its own numbers do not satisfy.**

Interval arithmetic on the per-kilogram figures printed in her chart shows that the four labelled steps of the fluid ladder are jointly consistent only with a body weight between 24.6 and 28.0 kg, which is neither her measured weight of 30 kg nor the ideal weight of 24 kg recorded on the same chart, and that the intervals implied by the paracetamol and the tranexamic acid annotations do not overlap. **No single body weight reproduces every per-kilogram figure this chart prints.** Separately, the Schofield equation was evaluated with the height in metres in a term whose coefficient is per centimetre, understating basal metabolic rate by 200 kcal/day, or 18.5%, and thereby overstating the adequacy of her inpatient intake as 68% of total energy expenditure where the corrected figure is 55.4%. The incompatible intervals are shown in Figure 4.

None of these findings changed what happened to this child, and none is offered as evidence about outcome. They are offered as evidence that in a paediatric dengue record the quantities most exposed to error are the ones with a body weight in the denominator, and that the weight is almost never written down. **The single change with the greatest return is also the cheapest: name the dosing weight, and its basis, in the order itself.** Beyond that, a daily weight, a declared haematocrit baseline, a second albumin at the haematocrit peak, ultrasonography at defervescence, urine output recorded in millilitres as well as per kilogram, one adiposity measure that is not a function of weight and height, a named indication and a culture before any antibiotic, and a recorded serotype with dated paired serology would together make a record of this kind fully auditable. Until the exposure variable is measured as carefully in the ward as it is defined in the cohort studies, the association between excess weight and dengue severity cannot be acted on at the bedside with any confidence. The measurements that would close that gap are listed in Table 6.

Declarations

Ethics approval

This report describes routine clinical care delivered in 2023 and involved no intervention, no additional investigation and no change to management. In accordance with institutional policy on single-case reports, the case was reviewed by the Health Research Ethics Committee of Dr. M. Djamil General Hospital and the Faculty of Medicine, Universitas Andalas, which issued a written determination that ethics-committee approval was not required for a de-identified retrospective description of care already given; the determination reference will be supplied to the editorial office on request and is reproduced in full in the accompanying ethics approval statement. Written informed consent for publication was obtained from the patient's father as her legal guardian, and age-appropriate assent was obtained from the child. All directly identifying information has been removed; no photograph, radiograph or other image of the patient appears in this report, and the figures are entirely author-generated from tabulated numerical data.

Consent for publication

Written consent for publication of this de-identified case, including all tables and figures, was obtained from the patient's father as her legal guardian. A copy of the signed consent form is available to the editorial office on request.

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 body-mass-index percentile and z score, the ideal body weight, the weight-for-height percentage, the haematocrit and platelet changes, the per-kilogram quotients, the weight intervals implied by the printed per-kilogram figures, and the recomputed basal metabolic rate – is reproducible from the values printed in Tables 1 to 5 together with the Centers for Disease Control and Prevention 2000 lambda-mu-sigma parameters for girls and the three coefficients of the metabolic-rate equation, which are printed in the energy-prescription subsection.

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. FIR: conceptualization, investigation, data curation, formal analysis, visualization, writing – original draft, project administration. RM: conceptualization, methodology, validation, supervision, writing – review and editing. RN: methodology, validation, resources, supervision, writing – review and editing. All 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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CARE guideline statement

This report was prepared in accordance with the CARE guideline for case reports.[96] A completed CARE checklist with page references accompanies the submission, and the patient and family perspective required by item 12 of that checklist appears in the Results section.

Use of artificial intelligence

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

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