

Case Report

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Septic shock mimicking dengue and typhoid: A case report

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ABSTRACT

Rationale: In dengue- and typhoid-endemic regions, a reactive IgM often anchors the diagnosis, yet severe sepsis can drive polyclonal B-cell activation and low-specificity antibodies that yield false-positive results.

Patient concerns: A 57-year-old previously healthy male patient presented with six days of fever, crampy abdominal pain, and mucoid diarrhea, followed by progressive weakness and circulatory collapse.

Diagnosis: Septic shock with multiple organ dysfunction (SOFA 10), thrombocytopenia, markedly raised procalcitonin, acute kidney injury, conjugated hyperbilirubinemia, and pancreatic edema. Dengue IgM was positive and *Salmonella* TUBEX was borderline positive, but both reverted to negative on repeat testing. The final diagnosis was culture-negative septic shock of presumed gastrointestinal origin with secondary acute pancreatitis.

Interventions: Fluid resuscitation, norepinephrine and vasopressin, empirical ceftriaxone, nutritional support, and conservative pancreatitis care.

Outcomes: Cultures obtained after antibiotics were sterile; procalcitonin fell from 7.95 to 0.15 ng/mL by day 7, and the patient recovered and was discharged on day 13.

Lessons: In septic shock, an isolated reactive IgM should be interpreted cautiously and integrated with biomarker trends, cultures, and organ-dysfunction patterns.

KEYWORDS: IgM misleads; Septic shock; Dengue; Typhoid

1. Introduction

In dengue- and typhoid-endemic regions, a reactive IgM often anchors the diagnosis. Severe sepsis; however, drives polyclonal

B-cell activation and low-specificity antibodies that produce false-positive results[1]. We present a male patient with culture-negative gastrointestinal septic shock and secondary pancreatitis. Initial serology returned falsely positive IgM results for both dengue and *Salmonella*, and repeat testing showed resolution of these positive findings. Dual false seropositivity occurring together during sepsis is seldom documented. Earlier publications mainly report isolated cross-reactivity between dengue and typhoid serological tests, not concurrent false positivity for both pathogens[2].

2. Case presentation

A 57-year-old man, previously healthy, had no known autoimmune disease, hematological disorder, malignancy, chronic liver disease, alcohol use, immunodeficiency, or long-term medication, including immunosuppressive therapy. He had no arthralgia, rash, or other clinical features suggesting systemic autoimmune disease; dedicated autoimmune testing was not performed.

The illness began on 14 February 2026 with fever, crampy abdominal pain, and mucoid diarrhea (3–5 stools/day), which resolved by day 5. On 19 February (day 6), he presented in shock, with epigastric pain radiating to the back and no jaundice. He

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Table 1. Serial hematological parameters during hospitalization.

Parameters	19 Feb. (Emergency room)	20 Feb. (Ward)	21 Feb. (Ward)	22 Feb. (Ward)	23 Feb. (Ward)	25 Feb. (Ward)	27 Feb. (Ward)	2 Mar. (Ward)	Reference range
WBC, $\times 10^3/\mu\text{L}$	8.1	6.6	11.72	9.42	10.04	10.15	10.05	8.18	3.37-10.0
Eosinophils, %	0.1	0.2	0.4	0.8	0.9	0.8	0.9	0.5	0.6-5.4
Basophils, %	0.2	0.3	0.3	0.2	0.6	0.7	0.7	0.6	0.3-1.4
Neutrophils, %	94.5	89.7	86.7	88.1	77.3	81.5	83.4	80.0	39.8-70.5
Lymphocytes, %	3.0	3.6	5.8	7.3	13.9	11.2	9.3	13.3	23.1-49.9
Monocytes, %	2.2	6.2	6.8	3.6	7.3	5.8	5.7	5.6	4.3-10.0
RBC, $\times 10^6/\mu\text{L}$	4.27	3.46	3.49	3.70	3.74	4.07	4.13	3.78	3.69-5.46
Hemoglobin, g/dL	13.0	10.7	10.9	11.5	11.8	12.6	12.7	11.4	11.0-14.7
Hematocrit, %	37.2	30.3	30.3	32.0	34.1	37.2	38.2	35.2	35.2-46.7
MCV, fL	87.1	87.6	86.8	86.5	91.2	91.4	92.5	93.1	86.7-102.3
MCH, pg	30.4	30.9	31.2	31.1	31.6	31.0	30.8	30.2	27.1-32.4
MCHC, g/dL	34.9	35.3	36.0	35.9	34.6	33.9	33.2	32.4	29.7-33.1
RDW-CV, %	12.8	13.1	13.2	13.0	13.4	13.4	13.6	13.3	12.2-14.8
Platelet count, $\times 10^3/\mu\text{L}$	48	33	34	57	63	126	223	342	150-450
Immature granulocytes, %	0.6	0.9	1.0	1.8	5.0	8.6	11.1	2.1	0-0.5

WBC: White blood cell, RBC: Red blood cell, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, RDW-CV: Red cell distribution width-coefficient of variation.

was conscious (GCS 15) with blood pressure 82/54 mmHg, heart rate 132/min, respiratory rate 24/min, and SpO₂ 97% on room air. Vasopressors were required despite fluid resuscitation (septic shock); the SOFA score was 10.

Investigations showed thrombocytopenia ($48 \times 10^3/\mu\text{L}$) and neutrophilia (94.5%) with a left shift; the peripheral smear showed immature granulocytes and giant platelets without atypical lymphocytes or blasts. Procalcitonin was 7.95 ng/mL, creatinine 2.76 mg/dL, with conjugated hyperbilirubinemia and raised amylase/lipase (341/1039 U/L). Blood gas showed a high-anion-gap metabolic acidosis (anion gap 16.3; albumin-corrected ≈ 19 ; base excess -5.2) with respiratory alkalosis, compatible with systemic illness and tissue hypoperfusion; the PaO₂/FiO₂ ratio was 395 (Supplementary Table 1). Stool microscopy showed fecal leucocytes (5-10/HPF) and erythrocytes (3-5/HPF) without amoeba, an inflammatory pattern. Urinalysis showed no significant pyuria (leucocytes 15.6/ μL , 2.8/HPF) and no nitrituria, with proteinuria and hyaline casts consistent with sepsis-associated acute kidney injury of a largely prerenal (hypoperfusion) nature, in keeping with the rapid renal recovery (Supplementary Table 2). Dengue IgM (immunochromatographic, ICT) was positive with negative IgG, and *Salmonella* TUBEX was positive (score 4); NS1 and RT-PCR were unavailable. Malaria rapid test and leptospira IgM were negative, and HBsAg, anti-HCV, and HIV were non-reactive. Chest radiography was normal, with clear lungs and sharp costophrenic angles (no effusion) (Supplementary Figure 1), and the ECG was normal; echocardiography was not performed. Ultrasound showed pancreatic edema without necrosis or gallstones (Supplementary Figure 2). Blood and urine specimens were obtained after antibiotic initiation; the urine and blood culture reports, available on 22 and 25 February, respectively, showed no growth, and repeat serology on 21 February was negative for both dengue IgM and TUBEX. Serial laboratory

data appear in Table 1 and Supplementary Table 3.

Initial resuscitation comprised two 500 mL crystalloid boluses over 2 hours, followed by colloid for persistent instability and maintenance crystalloid at 2000 mL/24 h. Ceftriaxone 2 g intravenously twice daily was started empirically on 19 February for suspected gastrointestinal sepsis and stopped on 28 February after a 10-day course. Norepinephrine was initiated at 0.05 $\mu\text{g/kg/min}$ and titrated to a maximum of 0.15 $\mu\text{g/kg/min}$. Vasopressin was added at 0.015 units/m on 20 February, reduced to 0.01 units/h on 21 February, and stopped on 22 February. Norepinephrine was subsequently weaned and stopped on 24 February. Urine output and hemodynamic recovery are detailed in Supplementary Table 4; no renal replacement therapy was required. Slight jaundice was noted on 25 February, coinciding with the peak bilirubin. With nutritional support, the patient recovered, with amylase, lipase, and bilirubin declining towards normal by discharge on 3 March 2026. The day-by-day course, investigations, and management are detailed in Supplementary Table 5, and the disease course and proposed causal sequence in Figure 1.

Blood gas analysis demonstrated mixed high-anion-gap metabolic acidosis (HCO₃⁻ 18.8 mmol/L, base excess -5.2 mmol/L; anion gap 16.3, albumin-corrected 19 mmol/L) together with concurrent respiratory alkalosis (pCO₂ 27 mmHg). The PaO₂/FiO₂ ratio was 395 (arterial pO₂ 83 mmHg/FiO₂ 0.21), corresponding to a SOFA respiratory sub-score of 1. Internal consistency was confirmed (Henderson equation: calculated [H⁺] 34.5 nmol/L, pH 7.46 *vs.* measured 7.45; TCO₂=HCO₃⁻+0.03 $\times$ pCO₂=19.6 mmol/L; Winter's formula expected pCO₂ 34-38 mmHg, the lower measured value indicating a superimposed respiratory alkalosis). FiO₂, fractional inspired oxygen; SO₂, oxygen saturation; TCO₂, total carbon dioxide; SOFA, Sequential Organ Failure Assessment.

The specimen was uncontaminated (normal epithelial cell counts). There was no pyuria; proteinuria with hyaline casts is consistent with

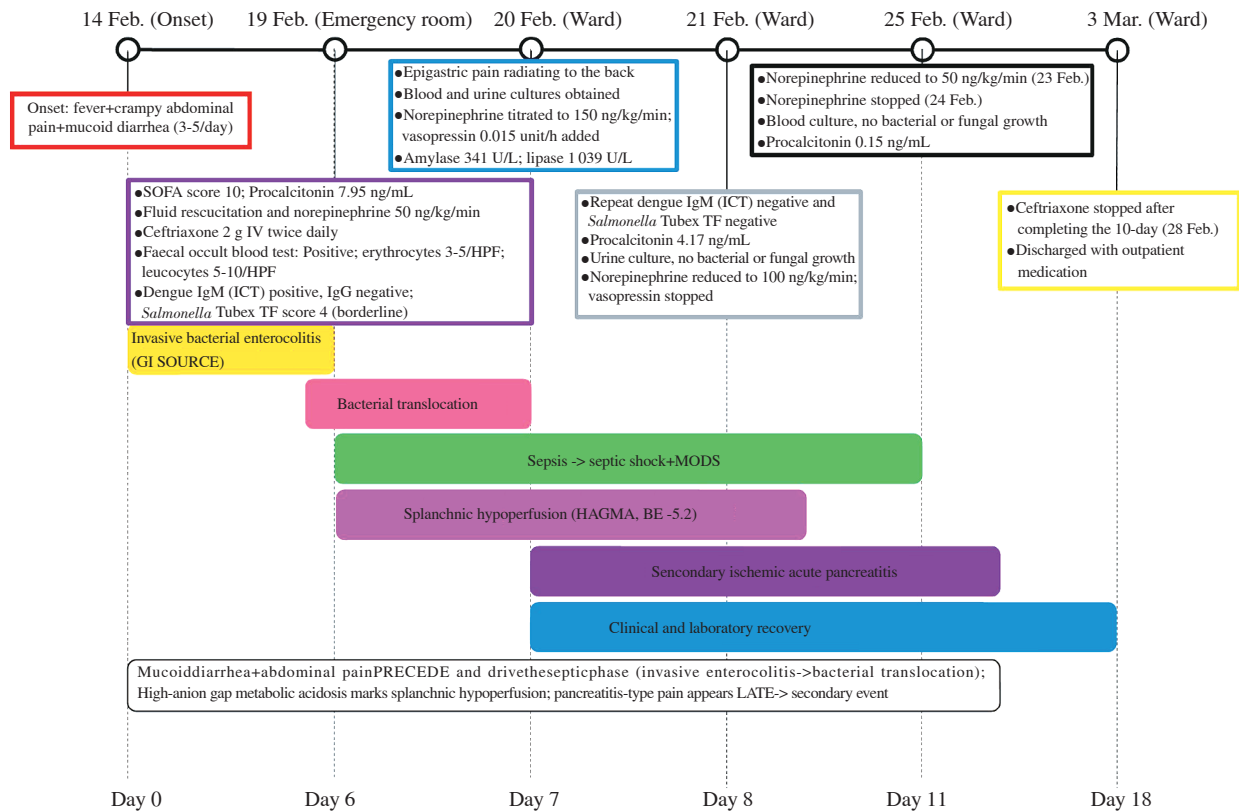


Figure 1. Timeline of the clinical course and the proposed causal cascade of culture-negative septic shock of presumed gastrointestinal origin with secondary acute pancreatitis in a 57-year-old man. An invasive bacterial enterocolitis (gastrointestinal source, presenting with mucoid diarrhea and abdominal pain) progressed through bacterial translocation to sepsis and septic shock with multiple organ dysfunction and splanchnic hypoperfusion (high-anion-gap metabolic acidosis, base excess -5.2), culminating in secondary ischaemic pancreatitis and followed by clinical and laboratory recovery. Dengue IgM and *Salmonella* TUBEX, drawn during shock, reverted to negative on repeat testing.

sepsis-associated acute kidney injury with a prerenal/hypoperfusion component. Microscopic bacteriuria without pyuria and with a subsequently sterile urine culture argues against urinary tract infection.

3. Discussion

We considered several explanations for the dengue IgM result rather than attributing it to a single mechanism. Acute dengue was not confirmed and was considered less likely because there was no haemoconcentration, pleural effusion, or ascites to support clinically significant plasma leakage[3], and the initially reactive IgM became negative on repeat testing. Nevertheless, NS1 antigen, RT-PCR, paired quantitative serology, and neutralization testing were unavailable; therefore, acute dengue could not be definitively excluded. Other flaviviral infections were assessed clinically and epidemiologically. Yellow fever and West Nile virus are not endemic in Indonesia, and there was no relevant travel or vaccination history. Zika virus infection was considered less likely in the absence of rash or conjunctivitis, while Japanese encephalitis was clinically unlikely because there

was no encephalopathy. Because confirmatory flavivirus testing was unavailable and the immunochromatographic assay was not independently validated in this case, the transient reactivity was interpreted as a probable non-specific or cross-reactive result rather than being attributed solely to sepsis-associated immune dysregulation.

Sequential infection, co-infection, secondary bacterial infection, serological cross-reactivity, and non-specific immune activation were all considered. The chronology—an enteric prodrome followed by shock, with both serological results becoming negative—was most consistent with culture-negative septic shock of suspected gastrointestinal origin accompanied by transient low-specificity serological reactivity; however, neither the causative pathogen nor a bacterial etiology was microbiologically confirmed. TUBEX overdiagnoses enteric fever in Indonesia[4]. Cultures were negative but drawn after ceftriaxone, supporting culture-negative sepsis[5]. Malaria, leptospirosis, amoebiasis, viral hepatitis, and HIV were excluded; scrub typhus and melioidosis testing was unavailable and could not be fully excluded. Procalcitonin was markedly elevated but was not treated as a standalone confirmatory marker of bacterial sepsis, because it can also rise in severe viral infection, major surgery

or trauma, and non-infectious systemic inflammation. The value of 7.95 ng/mL supported, but did not confirm, a bacterial process when interpreted alongside the neutrophilic left shift, inflammatory stool findings, organ dysfunction, and decline to 0.15 ng/mL by hospital day 7. Acute kidney injury and severe systemic inflammation may also have contributed to the elevation. Pre-antibiotic blood cultures, fecal culture, and bone-marrow culture were not obtained.

A structured assessment favored distributive shock. Cardiogenic shock was considered less likely because the ECG and chest radiograph showed no acute cardiac abnormality or pulmonary congestion and there were no clinical signs of heart failure; however, myocarditis or other myocardial dysfunction could not be definitively excluded because cardiac biomarkers and echocardiography were not obtained. Obstructive shock was clinically less likely in the absence of respiratory compromise or suggestive radiographic findings. Hypovolaemia from preceding diarrhea may have contributed initially, but persistent vasopressor dependence after fluid resuscitation argued against a purely hypovolaemic mechanism. Acute pancreatitis was identified after hemodynamic collapse and may itself have contributed to systemic inflammation and circulatory instability. Conversely, pancreatic edema without necrosis, gallstones, or marked hypertriglyceridemia could represent pancreatic injury secondary to septic splanchnic hypoperfusion[9,10]. The temporal and causal relationship therefore remains uncertain. The main limitations are the unconfirmed pathogen, unavailable virological and neutralization testing, cultures obtained after antibiotics, and the absence of echocardiography and cardiac biomarkers.

In patients with septic shock in endemic regions, reactive rapid serology should be interpreted cautiously and integrated with clinical chronology, epidemiology, microbiology, biomarker trends, and organ-dysfunction patterns. Repeat and, whenever available, confirmatory molecular or paired serological testing are required before classifying a transient IgM result as either acute infection or false positivity.

Conflict of interest statement

The authors declare that there is no conflict of interest.

Ethics approval and patient's consent

This study received an ethics exemption from the Health Research Ethics Committee of Dr. Soetomo General Academic Hospital, Surabaya, Indonesia (Ref. No. 2351/LOE/301.4.2/IV/2026; 30 April 2026). The study procedures adhered to the ethical principles of the

Declaration of Helsinki. Written informed consent for publication was obtained from the patient.

Data availability

Available from the corresponding author on request.

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The authors received no extramural funding for the study.

Authors' contributions

SR: conceptualization, investigation, data curation, writing-original draft, and visualization. A: conceptualization, supervision, validation, and writing-review and editing. Both authors approved the final manuscript.

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Supplementary Table 1. Arterial blood gas analysis (20 February 2026).

Parameter	Result (20/2/2026)	Reference range
pH	7.45	7.35–7.45
pCO ₂ (mmHg)	27	35–45
pO ₂ (mmHg)	83	80–100
HCO ₃ [−] (mmol/L)	18.8	22–26
TCO ₂ (mmol/L)	19.6	23–30
Base excess (mmol/L)	−5.2	−2 to +2
SO ₂ (%)	97	94–98
Temperature (°C)	37	—
FiO ₂ (%)	21	—
PaO ₂ /FiO ₂ ratio	395	≥400

Supplementary Table 2. Urinalysis and urine sediment microscopy on admission (19 February 2026).

Parameter	Result	Reference range
Dipstick and physicochemical analysis		
Specific gravity	1.011	1.003–1.030
pH	5.50	4.5–8.0
Colour	Yellow	Yellow
Clarity	Clear	Clear
Protein	3+	Negative
Glucose	Negative	Negative
Ketone	Negative	Negative
Bilirubin	Negative	Negative
Urobilinogen	Normal	Normal
Blood	Negative	Negative

Parameter	Result	Reference range
Leucocyte esterase	Negative	Negative
Nitrite	Negative	Negative
Albumin-to-creatinine ratio (mg/g)	≥ 300	<30
Protein-to-creatinine ratio (g/g)	≥ 0.5	<0.15
Sediment (automated microscopy)		
Erythrocytes (/μL)	2.2	0–4.36
Erythrocytes (/HPF)	0.4	0–2
Leucocytes (/μL)	15.6	0–14
Leucocytes (/HPF)	2.8	0–5
Non-squamous epithelial cells (/LPF)	1	0–1
Squamous epithelial cells (/LPF)	9	0–10
Hyaline casts (/LPF)	11	0–10
Pathological casts (/LPF)	Negative	Negative
Crystals (/LPF)	None	Negative
Bacteria (/LPF)	18.6	0–20
Bacteria (×10 ⁴ /mL)	10.3	0–0.99
Yeast/fungi (/LPF)	0.018	Negative

Note: HPF, high-power field; LPF, low-power field

Supplementary Table 3. Serial clinical chemistry parameters during hospitalization.

Parameter	19 Feb (ER)	20 Feb (Ward)	22 Feb (Ward)	23 Feb (Ward)	24 Feb (Ward)	25 Feb (Ward)	27 Feb (Ward)	2 March (Ward)	3 March (Ward)	Reference Range
Blood urea	40.3		74.5		31.6					7–18

Parameter	19 Feb (ER)	20 Feb (Ward)	22 Feb (Ward)	23 Feb (Ward)	24 Feb (Ward)	25 Feb (Ward)	27 Feb (Ward)	2 March (Ward)	3 March (Ward)	Reference Range
nitrogen (mg/dL)										
Serum creatinine (mg/dL)	2.76				0.99					0.6–1.3
Albumin (g/dL)	2.98		2.34		2.59	2.59	3.12			3.4–5.0
Direct bilirubin (mg/dL)	1.34		1.11			2.79		2.00	0.89	<0.2
Total bilirubin (mg/dL)	1.58		1.31			3.27		2.35	1.17	0.2–1.0
Amylase (U/L)		341		133		383	694	345	115	25–100
Lipase (U/L)		1039		424		593	690	504	168	16–77
Triglycerides (mg/dL)			175							<150
Total cholesterol			111							<200

Parameter	19 Feb (ER)	20 Feb (Ward)	22 Feb (Ward)	23 Feb (Ward)	24 Feb (Ward)	25 Feb (Ward)	27 Feb (Ward)	2 March (Ward)	3 March (Ward)	Refere nce Range
	(mg/dL)									

Supplementary Table 4. Timeline of the clinical course, investigations, and management.

Day / Date	Clinical findings	Investigations	Management
19 February 2026 (Day 1, admission)	1. Chief complaints: fever and fatigue 2. Fully conscious (GCS 15) 3. BP 82/54 mmHg; HR 132/min; RR 24/min; SpO ₂ 97% on room air 4. Septic shock (SOFA score 10): 5. Cardiovascular 3 (MAP maintained on norepinephrine 50 ng/kg/min) 6. Coagulation 3 (platelets 48 ×10 ³ /μL) 7. Renal 2 (creatinine 2.76 mg/dL) 8. Hepatic 1 (total bilirubin 1.58 mg/dL)	1. Hematology: neutrophilia 94.5%; thrombocytopenia 48 ×10 ³ /μL 2. Blood film: shift to the right with immature granulocytes; no atypical lymphocytes; no blasts 3. Inflammatory marker: procalcitonin 7.95 ng/mL 4. Biochemistry: total bilirubin 1.58, direct bilirubin 1.34 mg/dL; albumin 2.98 g/dL; BUN 40.3, creatinine 2.76 mg/dL; sodium 126.3 mmol/L (hypovolaemic hyponatremia) 5. Serology: dengue IgM (ICT) positive, IgG negative; Salmonella Tubex TF score 4 (positive); malaria ICT negative;	1. Fluid resuscitation: crystalloid 500 mL over 1 h, repeated once; colloid added for persistent instability. 2. Norepinephrine commenced at 50 ng/kg/min 3. Empirical broad-spectrum ceftriaxone 2 g IV twice daily 4. Admitted to the high-care unit

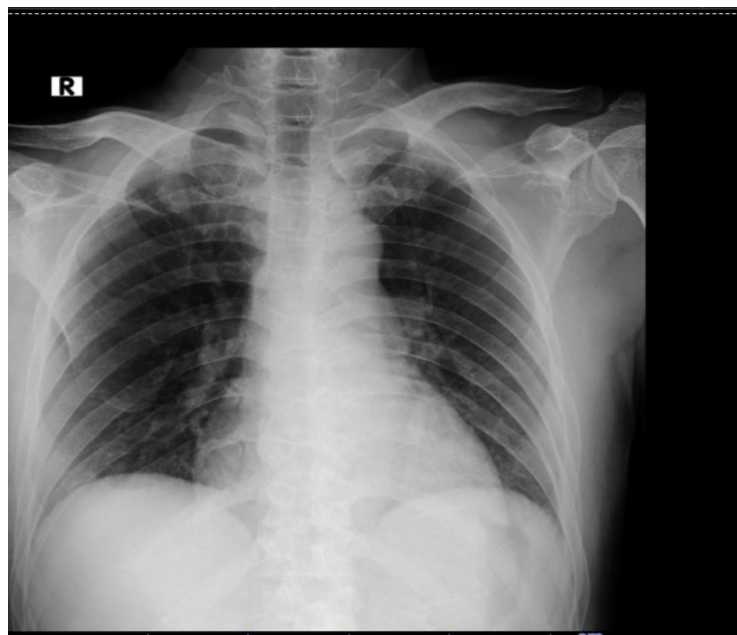
Day / Date	Clinical findings	Investigations	Management
	9. Respiratory 1 (PaO ₂ /FiO ₂ 395)	leptospira IgM (ICT) negative; HBsAg 0.32, anti-HCV 0.0478, HIV 0.179 all non-reactive (CLIA) 6. Urinalysis: proteinuria 7. Stool: faecal occult blood test positive; erythrocytes 3–5/HPF; leucocytes 5–10/HPF 8. ECG: sinus tachycardia (132/min) 9. Chest radiograph: heart and lungs normal	
20 February 2026 (Day 2)	1. Epigastric pain radiating to the back 2. Oliguria (urine output 100 mL/10 h) 3. Hemodynamic deterioration (MAP <65 mmHg)	1. Biochemistry: amylase 341 U/L; lipase 1039 U/L 2. Microbiology: blood and urine cultures obtained (after antibiotic initiation)	1. Maintenance crystalloid 2000 mL/24 h 2. Norepinephrine titrated to 150 ng/kg/min; vasopressin 0.015 unit/m added 3. Tramadol 100 mg IV every 12 h for analgesia 4. Nil by mouth; total parenteral

Day / Date	Clinical findings	Investigations	Management
			nutrition planned
21 February 2026 (Day 3)	1. Urine output improved to 700 mL/12 h 2. Hemodynamically stable (MAP >65 mmHg)	1. Hematology: leucocytosis $11.72 \times 10^3/\mu\text{L}$ 2. Serology: repeat dengue IgM and Salmonella Tubex TF negative 3. Inflammatory marker: procalcitonin 4.17 ng/mL	1. Parenteral nutrition via central venous catheter (Clinimix 1000 mL, Amino fluid 500 mL, RD5 500 mL/24 h) 2. Vasopressin reduced to 0.01 unit/m
22 February 2026 (Day 4)	Urine output 2700 mL/24 h; BP 128/82 mmHg	1. Haematology: leucocytes $9.42 \times 10^3/\mu\text{L}$; platelets $57 \times 10^3/\mu\text{L}$ 2. Biochemistry: creatinine 2.61 mg/dL; total bilirubin 1.31, direct bilirubin 1.11 mg/dL; sodium 136 mmol/L 3. Microbiology: urine culture, no bacterial or fungal growth	1. Norepinephrine reduced to 100 ng/kg/min; vasopressin stopped 2. Albumin 20% and potassium chloride 50 mEq infusions
23 February 2026 (Day 5)	1. Reduced abdominal pain 2. Urine output 3900 mL/24 h; improving hemodynamics	Biochemistry: amylase 133 U/L; lipase 424 U/L	Norepinephrine reduced to 50 ng/kg/min

Day / Date	Clinical findings	Investigations	Management
24 February 2026 (Day 6)	1. Improved abdominal pain 2. Urine output 2200 mL/24 h; haemodynamically stable	1. Biochemistry: creatinine 0.99 mg/dL; albumin 2.59 g/dL; potassium 4.0 mmol/L 2. Imaging: abdominal ultrasound, pancreatic edema without peripancreatic fluid	1. Norepinephrine stopped 2. Early enteral nutrition commenced; intravenous nutrition tapered; tramadol stopped.
25 February 2026 (Day 7)	1. Urine output 1900 mL/24 h; hemodynamically stable without vasopressor support 2. Slight jaundice noted (coinciding with peak bilirubin)	1. Microbiology: blood culture, no bacterial or fungal growth 2. Inflammatory marker: procalcitonin 0.15 ng/mL	
26 February 2026 (Day 8)	Urine output 3100 mL/24 h		Transferred to the low-care ward
27 February 2026 (Day 9)	1. No abdominal pain; improved wellbeing and oral intake 2. Urine output 2200 mL/24 h	1. Hematology: platelets $223 \times 10^3/\mu\text{L}$ 2. Biochemistry: albumin 3.12 g/dL	Antibiotic therapy planned to complete a 10-day course
28 February	Tolerating oral intake		Ceftriaxone stopped after

Day / Date	Clinical findings	Investigations	Management
2026 (Day 10)			completing the 10-day course.
3 March 2026 (Day 13)	Asymptomatic; no complaints	1. Hematology: platelets recovered to $342 \times 10^3/\mu\text{L}$ 2. Biochemistry: amylase 115, lipase 168 U/L; total bilirubin 1.17 mg/dL (all declining)	Discharged with outpatient medication

Note: BP, blood pressure; HR, heart rate; RR, respiratory rate; SpO₂, peripheral oxygen saturation; GCS, Glasgow Coma Scale; SOFA, Sequential Organ Failure Assessment; MAP, mean arterial pressure; PaO₂/FiO₂, ratio of arterial oxygen partial pressure to fractional inspired oxygen; ICT, immunochromatographic test; CLIA, chemiluminescent immunoassay; HPF, high-power field; IV, intravenous.



Supplementary Figure 1. Chest radiograph showing clear lung fields with sharp costophrenic angles and no pleural effusion or cardiomegaly.



Supplementary Figure 2. Abdominal ultrasonography showing pancreatic oedema without peripancreatic fluid collection or necrosis.