Scientific Research Publishing

Open Journal of Internal Medicine, 2026, 16(2), 255-281
https://www.scirp.org/journal/ojim
ISSN Online: 2162-5980
ISSN Print: 2162-5972

Peritoneal Dialysis as Sole RRT in Severe Paediatric MODS with AKI Stage 3 and Late Stenotrophomonas Maltophilia Bloodstream Infection: PD Prescription Deviation and Neurocritical Complications in a Resource-Limited PICU

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How to cite this paper: Najiaghdam, Y.T. (2026) Peritoneal Dialysis as Sole RRT in Severe Paediatric MODS with AKI Stage 3 and Late Stenotrophomonas Maltophilia Bloodstream Infection: PD Prescription Deviation and Neurocritical Complications in a Resource-Limited PICU. *Open Journal of Internal Medicine*, **16**, 255-281.
https://doi.org/10.4236/ojim.2026.162021

Received: April 20, 2026
Accepted: May 26, 2026
Published: May 29, 2026

Abstract

Background: Multi-organ dysfunction syndrome (MODS) in infants carries mortality exceeding 50% in resource-limited settings. Acute kidney injury (AKI) Stage 3 requiring renal replacement therapy (RRT) is particularly challenging, where continuous RRT (CRRT) is unavailable. Peritoneal dialysis (PD) remains the sole accessible RRT in most sub-Saharan African paediatric intensive care units (PICUs). We report a case of survival from severe sepsis-induced MODS with AKI Stage 3 and ischaemic hepatitis managed with PD as sole RRT, with concurrent documentation of a PD prescription deviation and its quantified clinical impact, followed by late neurocritical complications temporally associated with Stenotrophomonas maltophilia bloodstream infection with suspected, but unconfirmed, catheter association. **Case Presentation:** A 9-month-old female (7.7 kg) presented with bacterial gastroenteritis, febrile convulsions, and rapid progression to septic shock with MODS. Admission procalcitonin (PCT) was 26.49 ng/mL with a normal white cell count. She required mechanical ventilation for 13 days, three simultaneous vasoactive agents, and PD for AKI Stage 3 with peak creatinine 302.6 μmol/L and peak AST 4274 U/L. A nursing prescription deviation—extending the prescribed 20-minute dwell time to 60 - 120 minutes without authorisation—reduced dialysate exposure by 55% - 60% and delayed the onset of creatinine decline by approximately 48 hours. Following correction, creatinine normalised to 74.5 μmol/L by Day 16, and the PD catheter was removed. PCT at Day 15 was 1.02 ng/mL—

a 96% reduction from admission. On Day 18, new severe sepsis developed (PCT 9.28 rising to 52.83 ng/mL), complicated by sepsis-associated coagulopathy suggestive of disseminated intravascular coagulation (DIC; APTT 16.3 seconds), right parietal intracerebral haemorrhage (7.1 × 6.6 cm), bilateral cerebral infarction, and hydrocephalus on CT brain. Blood culture confirmed Stenotrophomonas maltophilia—sensitive only to levofloxacin, resistant to cotrimoxazole, and intrinsically resistant to carbapenems, including meropenem, used for 17 days. **Conclusion:** This case documents AKI Stage 3 recovery via PD in a resource-limited PICU, a quantified PD prescription deviation, and late Stenotrophomonas bloodstream infection (BSI) with presumed catheter origin causing neurocritical complications—representing a notable triple clinical and patient safety contribution to the literature. Carbapenem stewardship, strict PD dwell time compliance, and early CVC removal are key preventable factors.

Keywords

Peritoneal Dialysis, AKI Stage 3, Mods, Stenotrophomonas Maltophilia, Bloodstream Infection, Candiduria, Intracranial Haemorrhage, Sepsis-Associated Coagulopathy, Paediatric ICU, Resource-Limited, Sub-Saharan Africa, Antibiotic Stewardship, Patient Safety

1. Introduction

Multi-organ dysfunction syndrome (MODS) represents the most severe spectrum of sepsis and carries a mortality rate of 40% - 60% in paediatric patients, rising substantially in resource-constrained settings [1]. Acute kidney injury (AKI) is common in critically ill children with sepsis and independently increases mortality, prolongs ICU stay, and confers long-term renal sequelae [2]. When AKI reaches Stage 3 by Kidney Disease: Improving Global Outcomes (KDIGO) criteria—creatinine exceeding 3 times baseline, sustained oliguria, or the need for RRT—active renal support is mandatory.

Continuous renal replacement therapy (CRRT) is the preferred modality for haemodynamically unstable paediatric AKI in high-income settings [3]. However, CRRT remains inaccessible across most sub-Saharan African PICUs owing to prohibitive equipment costs, the absence of trained personnel, and constrained healthcare infrastructure. Acute peritoneal dialysis (PD) constitutes the only routinely accessible RRT modality in the majority of African PICUs [4]. The physiological rationale for short-cycle acute PD rests on solute transport kinetics: peritoneal equilibration of small solutes reaches 80% - 90% of maximum within the first 20 - 25 minutes of dwell, after which the concentration gradient is substantially exhausted [5]. Extension of dwell time beyond this saturation plateau reduces cycle frequency—the primary determinant of total daily delivered PD dose—without proportional gain in per-cycle clearance.

A second but underappreciated threat in resource-limited PICUs is suspected

catheter-related bloodstream infection (CRBSI) from central venous catheters, though definitive catheter origin is often difficult to confirm in resource-limited settings where differential time-to-positivity and quantitative catheter tip cultures may not be available. Stenotrophomonas maltophilia is an emerging nosocomial Gram-negative bacillus characterised by intrinsic resistance to carbapenems—the agents most commonly used for empirical sepsis coverage—and is increasingly selected by prolonged broad-spectrum antibiotic exposure in ICU patients [6]. Its propensity to cause haematogenous seeding, including cerebral septic emboli, renders it particularly dangerous in the post-MODS recovery phase when patients are physiologically vulnerable.

We present a case of a 9-month-old infant who survived severe sepsis-induced MODS with AKI Stage 3 and ischaemic hepatitis managed exclusively with PD as sole RRT in a resource-limited Kenyan PICU, complicated by a documented and quantified nursing prescription deviation, and subsequently by Stenotrophomonas maltophilia bloodstream infection of presumed catheter origin causing disseminated intravascular coagulation, intracranial haemorrhage, bilateral cerebral infarction, and hydrocephalus. Based on a PubMed search conducted in March 2026 using the terms "peritoneal dialysis" AND "acute kidney injury" AND "paediatric" AND "Africa" (limited to case reports and English language), no published case from sub-Saharan Africa was identified documenting all three components simultaneously: PD efficacy as sole RRT in paediatric MODS, a quantified PD dwell time prescription deviation with biochemical validation, and late Stenotrophomonas bloodstream infection with neurocritical complications. While individual components have been reported separately—Ademola *et al.* described acute PD outcomes in Nigerian children, and Brooke and Looney *et al.* have reviewed Stenotrophomonas nosocomial infections—their simultaneous occurrence in a single paediatric case has not been previously documented.

2. Case Presentation

2.1. History and Presentation

A 9-month-old female weighing 7.7 kg was referred from a community health facility in Kikuyu, Kenya, on 20 March 2026 (**Figure 1**). The referring physician recorded four episodes of loose, mucoid stools and one episode of vomiting over two days, an active tonic-clonic convulsion lasting two minutes at the referring facility, and associated cough, lethargy, and tachypnoea. Vital signs at the referring facility: temperature 40.4˚C, heart rate 169 beats per minute, respiratory rate 55 breaths per minute, oxygen saturation 96% on room air. Prior to transfer, the patient received intravenous Ringer's lactate 210 ml over one hour, rectal diazepam 3.5 mg, and paracetamol 125 mg.

On arrival at Lifecare Hospitals Kikuyu, examination revealed a markedly unwell, febrile infant with a bulging anterior fontanelle, lower chest wall in-drawing, and bilateral chest crepitations. No rash, petechiae, hepatosplenomegaly, or meningism was identified. The infant was alert but markedly irritable. Developmental

history revealed pre-existing delayed motor milestones. Immunisation status was appropriate for age. Blood group was B Rhesus positive.

A 9-month-old female infant, during the acute critical phase. Orotracheal tube secured with tape and eyes taped for corneal protection during cisatracurium blockade. Multiple peripheral IV lines delivering concurrent vasopressors, sedation, and electrolyte replacement. Five-lead cardiac monitoring and a peripheral SpO_2 probe are visible. This image was obtained during the period of maximal multi-organ support—mechanical ventilation, three vasoactive agents, and peritoneal dialysis—between ICU Days 2 and 6.

Figure 1. Patient during peak multi-organ support.

2.2. Initial Investigations

Admission investigations on 20 March 2026 demonstrated procalcitonin (PCT) 26.49 ng/mL—markedly elevated, consistent with severe bacterial sepsis—while C-reactive protein was 1.60 mg/L, reflecting the characteristic temporal dissociation between these markers in early bacteremia (PCT rises within 2 - 6 hours; CRP lags 24 - 48 hours) [7]. White blood cell count was 11.15×10^9/L with a neutrophil fraction of 53.2%—within the laboratory reference range despite massively elevated PCT, reflecting overwhelming sepsis before adequate bone marrow response. Haemoglobin was 11.3 g/dL with a microcytic pattern (MCH 24.8 pg, MCV 76.8 fL), consistent with pre-existing iron deficiency anaemia. Platelets were 221×10^9/L.

Stool analysis demonstrated loose, mucoid, green-coloured stools with 3 - 8 pus cells per high-power field, no erythrocytes, no parasites, and negative Rota virus antigen—consistent with bacterial gastroenteritis as the primary sepsis source. No causative organism was identified on blood culture at this stage. Vitamin D tested on Day 7 was 13 ng/mL, classified as insufficient, contributing to persistent ionised hypocalcaemia throughout the admission. Renal ultrasonography on Day 6 confirmed bilaterally normal kidneys with preserved corticomedullary differentiation, appropriate PD catheter positioning in the pelvic cavity, and a mild right pleural effusion (16.3 mm maximum depth). Venous Doppler of the right upper

limb demonstrated no deep vein thrombosis.

The earliest documented creatinine was 72.2 μmol/L on Day 1, representing the pre-AKI baseline. This rose to 252.7 μmol/L by Day 3, 279.6 μmol/L by Day 4, and reached a peak of 302.6 μmol/L on Day 6—confirming AKI Stage 3 by KDIGO criteria (creatinine exceeding 3 × baseline: 3 × 72.2 = 216.6 μmol/L). Urea peaked concurrently at 106.84 mg/dL. Liver function testing on Day 2 revealed AST 3342 U/L; repeat on Day 3 confirmed a true AST peak of 4274 U/L and ALT peak of 1060 U/L, consistent with severe ischaemic hepatitis from septic shock. Serial biochemical data are presented in Tables 1-3.

Table 1. Serial kidney function tests—21 March to 15 April 2026.

Date	Day	Na (mmol/L)	K (mmol/L)	Cr (μmol/L)	Urea (mg/dL)	PD Phase
21-Mar	1	151	4.5	72.2 (baseline)	39.64	Pre-PD
23-Mar	3	151 - 152	3.9 - 4.5	174.9 - 252.7	73.99 - 99.81	PD Correct
24-Mar	4	151 - 152	3.9 - 4.5	174.9 - 279.6	73.99 - 101.9	PD Correct
26-Mar	6	137 - 146	4.5 - 5.0	**302.6 ← PEAK**	**106.84 ← PEAK**	Deviation corrected
27-Mar	7	135	4.1	277.7	84.6	Corrected PD
31-Mar	11	128 - 129	2.6 - 3.9	240.8 - 245.0	58.44 - 62.22	Corrected PD
02-Apr	13	152	4.5	219.0	82.3	PD Holiday
04-Apr	15	148	4.7	105.0	79.5	PD Holiday
05-Apr	**16**	146	3.7	**74.5 ✓ Normal**	69.49	Catheter removed
11-Apr	**22**	133	3.9	**27.2 ‡**	**26.8 ✓**	Post-BSI; PCT 11.75
15-Apr	**26**	—	—	**19.7 ‡**	**27.09 ✓**	Late inpatient recovery phase

✓ Within normal reference range (53 - 123 μmol/L). ‡ Below baseline—consistent with critical illness sarcopenia.

Table 2. Serial liver function tests.

Date	Day	Albumin (g/dL)	AST (U/L)	ALT (U/L)	GGT (U/L)	Direct Bili (mg/dL)
22-Mar	2	3.25	3342	810	8.3	0.10
23-Mar	3	3.30	**4274 ← PEAK**	**1060 ← PEAK**	32.1	0.07
24-Mar	4	2.9	675	566	105	1.6
26-Mar	6	2.49	1485	579	219	0.11
31-Mar	11	2.71	121.5	15.4 ✓	188.7	0.15
04-Apr	**15**	**3.1**	**130.8**	**19.3 ✓**	177.4	**0.10 ✓**

✓ Within or approaching normal reference range.

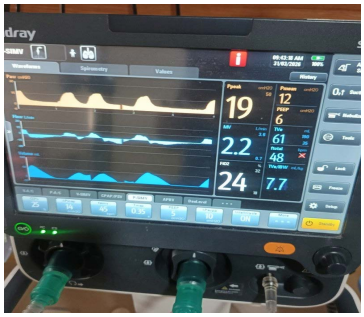
Table 3. Serial blood gas results.

Date	Day	pH	pCO_2 (mmHg)	HCO_3 (mmol/L)	Lactate (mmol/L)	iCa (mmol/L)
26-Mar	6	7.223	43.5	17.5	3.5	1.03
27-Mar	7	7.369	30.0	16.9	3.0	1.27
31-Mar	11	7.250	45.0	19.3	1.6	1.19
03-Apr	14	~7.36	52.3	**24.2 ✓**	1.6	1.19
04-Apr	**15**	**7.381 ✓**	35.2	21.4	1.8	0.87

✓ Within normal reference range.

2.3. Management—Phase 1: Critical Resuscitation (Days 1 - 6)

Orotracheal intubation was performed on Day 1 following rapid deterioration with worsening hypoxia and haemodynamic instability. Mechanical ventilation was initiated in pressure-controlled assist-control mode (PC-ACV) with target tidal volumes of 5 - 6 ml/kg, PEEP 5 - 6 cmH_2O, and respiratory rates titrated by Winter's formula. Cisatracurium was selected for neuromuscular blockade over rocuronium and vecuronium, as Hofmann elimination renders it independent of hepatic or renal clearance—a critical distinction given the concurrent severe ischaemic hepatitis (peak AST 4274 U/L) [8]. Three concurrent vasoactive agents were required: adrenaline (epinephrine), vasopressin, and dobutamine, consistent with current paediatric sepsis guidelines [9]. Meropenem was selected as the primary empirical antibiotic for suspected Gram-negative enteric sepsis, given the clinical presentation of bacterial gastroenteritis with rapid progression to septic shock, and the dosing interval was extended from 8-hourly to 12-hourly during maximal renal impairment (**Figure 2**). The referring facility had administered ceftriaxone and gentamicin; escalation to meropenem was warranted, given the severity of organ dysfunction and the possibility of extended-spectrum beta-lactamase (ESBL)-producing organisms. Azithromycin was added on Day 16 following post-extubation chest radiography demonstrating bilateral reticulonodular opacities consistent with ventilator-associated atypical pneumonia, to provide coverage for atypical organisms (Mycoplasma, Chlamydia, Legionella). Vancomycin was commenced on Day 18 to cover potential Gram-positive nosocomial pathogens, including methicillin-resistant Staphylococcus aureus (MRSA), given the new sepsis episode with an indwelling CVC. Metronidazole was added concurrently for anaerobic coverage and potential Clostridioides difficile colitis in the context of prolonged broad-spectrum antibiotic exposure. Fluconazole was initiated on Day 20 following identification of heavy candiduria (yeast cells ++++) on urinalysis, in the context of evolving neutropenia and prolonged broad-spectrum antibiotic exposure [10]. Following Stenotrophomonas maltophilia identification on Day 19, meropenem was discontinued (intrinsically inactive), and levofloxacin commenced as the sole agent with confirmed activity.



Mindray P-SIMV mode: PEEP 6 cmH_2O, FiO_2 24%, peak inspiratory pressure 19 cmH_2O, TV 61 ml (7.7 ml/kg), total RR 48 bpm. Low FiO_2 of 24% on Day 10 confirms adequate oxygenation and progress toward weaning parameters.

Figure 2. Ventilator screen—day 10 (31-Mar-2026).

Peritoneal dialysis was commenced on Day 2 (22 March 2026) following percutaneous PD catheter insertion, confirmed sonographically in the pelvic cavity. The prescribed protocol was: fill volume 20 ml/kg (150 ml), fill time 10 minutes, dwell time 20 minutes, drain time 25 - 30 minutes, yielding approximately 26 cycles per 24 hours with approximately 3900 ml total daily dialysate exposure. Heparin 500 units/L was added to each dialysate bag. Initial dialysate concentration was 2.5% dextrose, adjusted to 1.5% dextrose as fluid balance improved (Figure 3, Figure 4).

Dialysate fill bags suspended from an IV pole with effluent draining by gravity into a calibrated collection bag. Manual clamp operation by nursing staff controlled all three phases. Improvised collection basins on the floor served as secondary volume measurement vessels. The absence of automated cycling equipment is representative of acute PD delivery in the majority of sub-Saharan African PICUs and was the context in which the prescription deviation occurred.

Figure 3. Bedside gravity PD setup—resource-limited PICU.

Single-cycle drain volume approximately 500 ml, demonstrating amber/orange discoloration consistent with bilirubin staining from ischaemic hepatitis (peak AST 4274 U/L; conjugated bilirubin 1.6 mg/dL on Day 4). Clear fluid without turbidity or fibrin—no peritonitis throughout the 14-day PD course.

Figure 4. PD effluent with amber discoloration—bilirubin staining.

2.4. Prescription Deviation—Dwell Time Error and Quantified Clinical Impact

On Day 5 of peritoneal dialysis (ICU Day 6), review of the bedside PD chart at ward round revealed that nursing staff had extended the prescribed dwell time from 20 minutes to 60 minutes and, in subsequent cycles, to approximately 120 minutes without physician authorization. This alteration was not documented in the medical record and had been ongoing for approximately four to five days. Fill volumes had also been reduced from the prescribed 150 ml to approximately 110 ml per cycle, compounding the prescription deviation.

Three complementary analytical approaches quantify the clinical impact. First, cycle frequency analysis: the reduction from 26 to 14 - 16 cycles per 24 hours represents a 42% decrease in dialysate delivery opportunities. Second, dialysate volume analysis: total daily dialysate exposure fell from 3900 ml to approximately 1540 - 1760 ml—a 55% - 60% volumetric reduction. Third, mass transfer modelling incorporating the peritoneal equilibration plateau: at 20 minutes, the membrane achieves approximately 85% of maximum small solute equilibration; at 90 minutes (mean deviation dwell), approximately 95% is reached. Applying this correction—prescribed clearance $26 \times 0.85 = 22.1$ arbitrary units; deviation clearance $15 \times 0.95 = 14.2$ units—yields an estimated net reduction in small solute clearance of 35% - 40%. The academically preferred figure is therefore an estimated 35% - 40% net clearance reduction, while the 55% - 60% dialysate volume reduction most accurately characterises the prescription failure in volumetric terms. These estimates are derived from proportional modelling based on published peritoneal equilibration kinetics [5] and assume linear solute transport, uniform membrane permeability, and constant ultrafiltration—assumptions that are reasonable approximations but were not validated by direct Kt/V measurement in this patient. The 48-hour delay in creatinine decline is a clinical correlation rather than a controlled comparison.

Clinical Correlation:

Creatinine continued rising during the deviation period to peak 302.6 μmol/L on Day 6. Consistent creatinine decline commenced only within 48 hours of dwell correction—a clinical correlation that validates the kinetic analysis. The onset of creatinine decline was delayed by approximately 48 hours relative to the projected trajectory had the correct prescription been maintained—with normalisation by Day 16 rather than a projected Day 13 - 14 (**Table 4**).

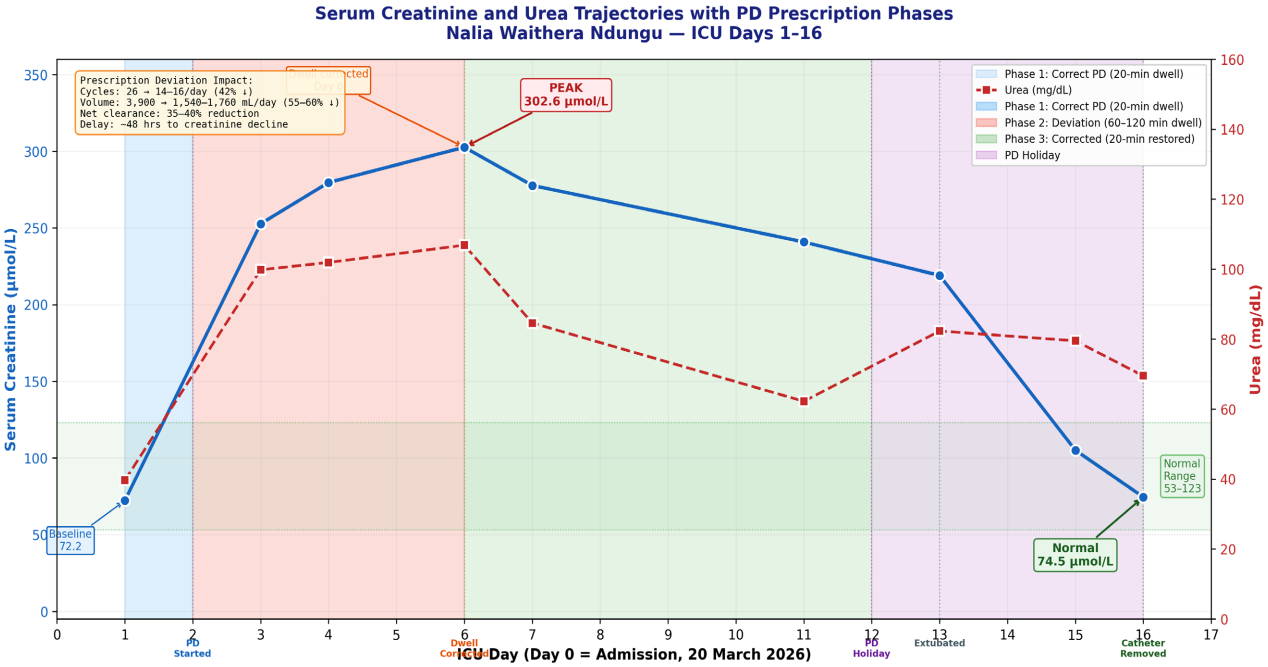
Table 4. Quantification of PD prescription deviation impact.

Parameter	Prescribed	During Deviation	Difference
Fill Volume	150 ml (20 ml/kg)	~110 ml	−40 ml/Cycle
Dwell Time	20 Minutes	60 - 120 Minutes	3 - 6× Prescribed
Cycles per 24 Hours	~26	~14 - 16	**42% Reduction**

Continued

Total Dialysate/24 hrs	~3900 ml	~1540 - 1760 ml	**55% - 60% Reduction**
Net Clearance Reduction	**100%**	**35% - 40% (Mass Transfer Model)**	**Academically Preferred Estimate**
Delay in Creatinine Decline	Expected from Day 4 - 5	Began Day 7	**~48-Hour Delay; Normalisation Day 16 vs Projected Day 13 - 14**

Mass transfer model: prescribed clearance 26 × 0.85 = 22.1 units; deviation clearance 15 × 0.95 = 14.2 units; reduction 35.5%. Reference: Twardowski *et al.* [5].

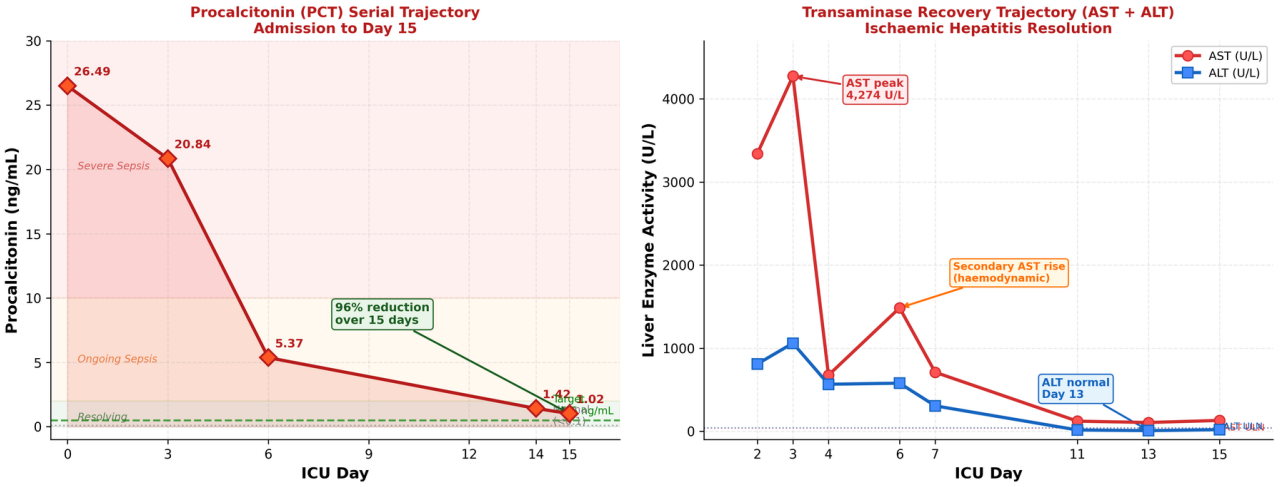


Phase 1 (Days 1 - 2, blue): correct 20-min dwell. Phase 2 (Days 2 - 6, red): deviation to 60 - 120 min—42% cycle reduction, 55% - 60% dialysate volume reduction, 35% - 40% net clearance reduction. Phase 3 (Days 7 - 16, green): corrected dwell—consistent creatinine decline to normalisation.

Figure 5. Serum creatinine trajectory by ICU day with PD prescription phases.

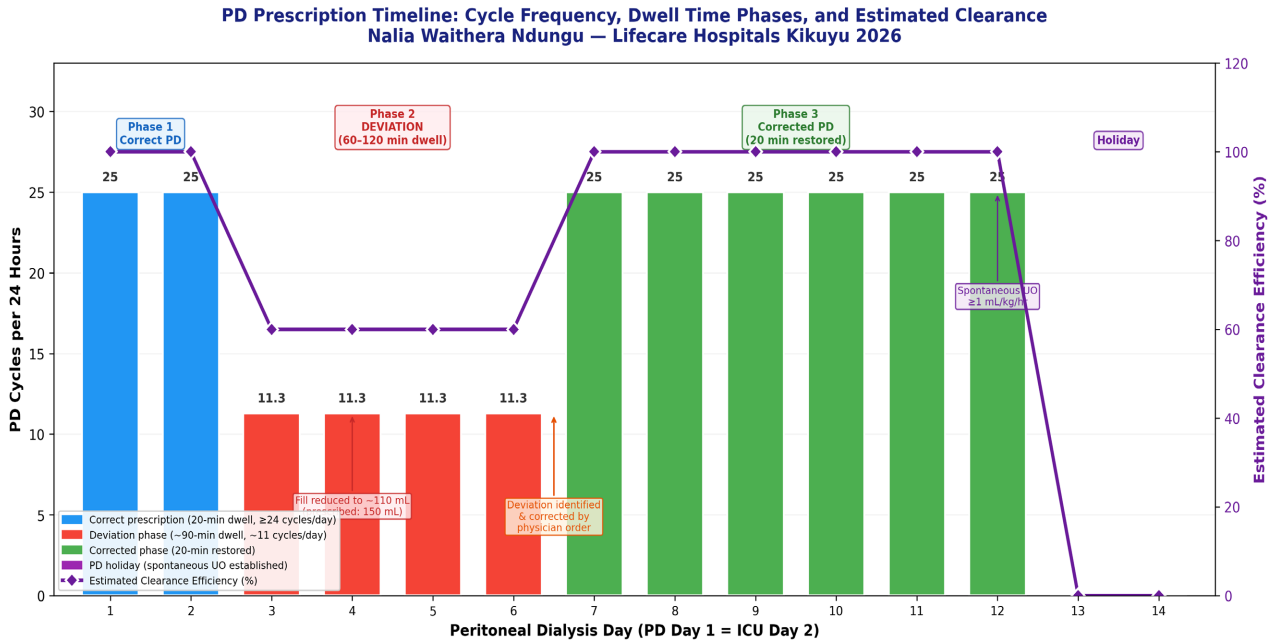
2.5. Phase 2: Recovery and Extubation (Days 7 - 16)

Following dwell time correction, creatinine demonstrated its first consistent decline: 277.7 µmol/L (Day 7), 240.8 µmol/L (Day 11), 177.9 µmol/L (Day 14), 105.0 µmol/L (Day 15), and 74.5 µmol/L (Day 16)—within the normal reference range. AST fell from 4274 U/L to 121.5 U/L by Day 11 and 130.8 U/L by Day 15. ALT normalised to 7.8 U/L by Day 13. Bicarbonate reached 24.2 mmol/L on Day 14—the first normal value since admission. Lactate fell from 3.5 mmol/L at peak to 1.6 mmol/L by Day 14.



Left: PCT 26.49 → 1.02 ng/mL—96% reduction over 15 days. Right: AST peak 4274 U/L → 130.8 by Day 15; ALT normalised Day 13. Secondary AST rise on Day 6 reflects haemodynamic compromise during peak AKI severity.

Figure 6. PCT and hepatic transaminase recovery.



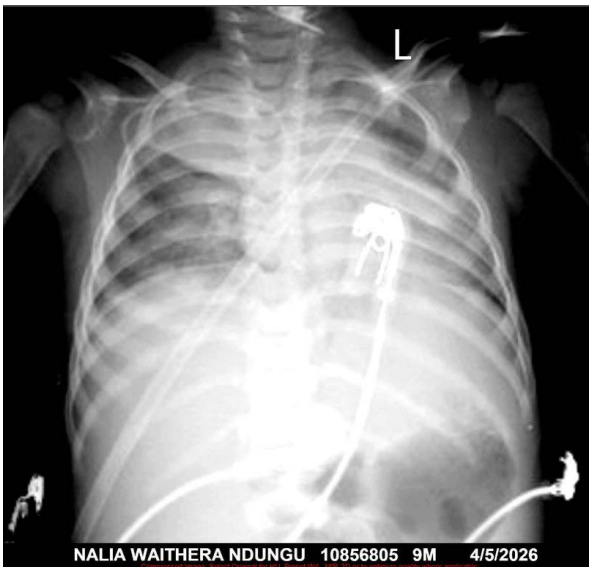
Purple line: estimated clearance efficiency. Deviation phase (orange, PD Days 3 - 6): cycles fell to ~14 - 16/day (mean ~15/day used for clearance modelling), estimated 35% - 40% net clearance reduction. PD holiday Day 12 after spontaneous UO ≥ 1 ml/kg/hr confirmed.

Figure 7. PD prescription timeline. Bars: actual cycle frequency/day.

Vasopressin was discontinued on Day 11—the first vasopressor weaned. A spontaneous breathing trial on Day 12 demonstrated SpO_2 100% on a non-rebreather mask at 10 L/min with RR 28 - 33 bpm; the patient was extubated on Day 13. Total ventilation duration was 13 days. PD holiday commenced on Day 12 after spontaneous urine output exceeded 1 ml/kg/hour. The PD catheter was re-

moved on Day 16 (creatinine 74.5 μmol/L, pH 7.381). No peritonitis was documented throughout the 14-day PD course [11]. PCT at Day 15 was 1.02 ng/mL—a 96% reduction from the admission value of 26.49 ng/mL. (**Figure 6**, **Figure 7**)

Post-extubation chest radiography on Day 16 demonstrated right apical consolidation and bilateral fine reticulonodular opacities—consistent with ventilator-associated atypical pneumonia (VAP). Azithromycin was added for atypical coverage (**Figure 8**).



Right apical consolidation and bilateral fine reticulonodular opacities with perihilar infiltrates—consistent with VAP/atypical pneumonia. PD catheter visible in abdomen (removed same day). No pleural effusion or pneumothorax. Left lateral marker confirms projection. Reported by Dr. Onkoba V., Lifecare Hospitals.

Figure 8. Post-extubation CXR—Day 16 (05-Apr-2026).

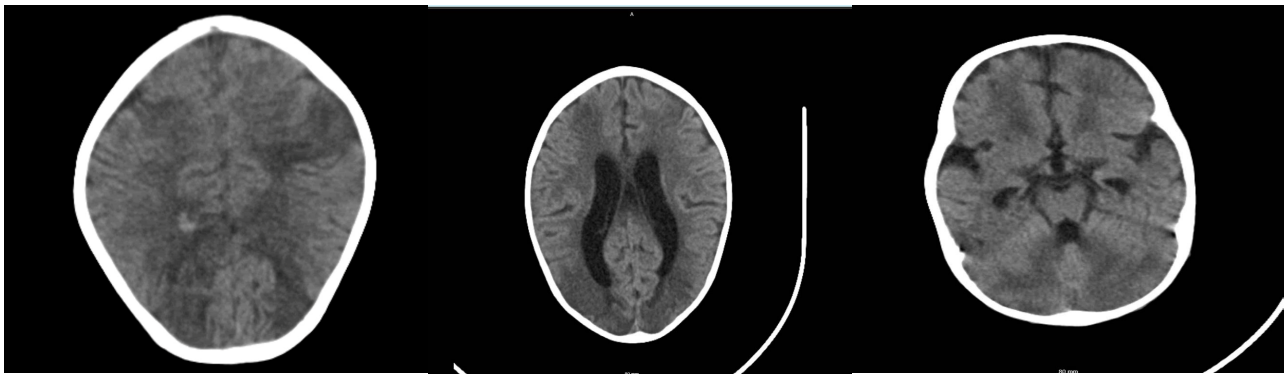
2.6. Phase 3: Late Complication—Stenotrophomonas Bloodstream Infection and Neurocritical Events (Days 17 - 19)

On Day 17, morning biochemistry demonstrated a severe electrolyte crisis: magnesium 0.40 mmol/L (critically low), total calcium 1.58 mmol/L (critically low), and potassium 3.2 mmol/L—all attributable to furosemide-driven renal wasting compounded by failure to administer prescribed magnesium sulfate on the preceding day. Creatinine continued to fall to 64.6 μmol/L—below the pre-AKI baseline—reflecting critical illness-associated muscle wasting rather than renal dysfunction.

On Day 18, PCT rose acutely to 9.28 ng/mL—a nine-fold increase from Day 15—followed by 52.83 ng/mL on Day 19. This trajectory was incompatible with the prior resolving course and confirmed a new, distinct sepsis event. Coagulation testing revealed a critically shortened APTT of 16.3 seconds (reference 27 - 43 seconds), consistent with sepsis-associated coagulopathy suggestive of the hypercoagulable consumption phase of disseminated intravascular coagulation (DIC),

with INR 1.14, suggesting relative preservation of the extrinsic pathway. Formal DIC scoring (ISTH criteria) was not possible as fibrinogen and D-dimer were not available. The subclavian CVC, *in situ* for 12 days, was removed, and the tip was sent for culture. Blood cultures were obtained simultaneously.

A non-enhanced CT brain performed on Day 18 revealed three significant findings: a right parietal intracerebral haemorrhage measuring 7.1 × 6.6 cm without perilesional oedema and without midline shift; a left parietal subacute cerebral infarct estimated 3 - 10 days old; and Benign Enlargement of Subarachnoid Spaces in Infancy (BESS) with periventricular leukomalacia (PVL)—findings consistent with pre-existing hypoxic-ischaemic injury corresponding to the delayed motor milestones at admission. Review of additional CT slices at the ventricular level demonstrated markedly enlarged bilateral lateral ventricles consistent with hydrocephalus, and slices at the temporal level revealed bilateral temporal lobe and cerebellar hypodensity—establishing diffuse hypoxic-ischaemic encephalopathy as the unifying radiological diagnosis (Figure 9).



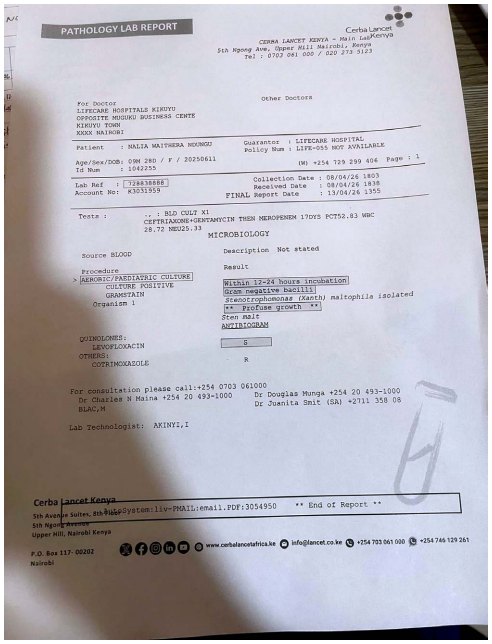
(A) (B) (C)

(A) Widened subarachnoid spaces (BESS), diffuse white matter hypodensity (PVL), and focal hyperdense haemorrhagic lesion. No midline shift. (B) Markedly enlarged bilateral lateral ventricles (communicating or ex vacuo hydrocephalus), severe periventricular white matter hypodensity indicating extensive leukomalacia, and thin cortex. Midline central. (C) Bilateral temporal lobe hypodensity, bilateral cerebellar hypodensity, and prominent Sylvian fissures. Brainstem preserved. Pattern consistent with diffuse hypoxic-ischaemic encephalopathy from prolonged vasopressor-dependent septic shock.

Figure 9. (A) CT brain—high vertex—BESS + PVL + haemorrhage; (B) CT brain—ventricular level—hydrocephalus + severe PVL; (C) Temporal/posterior fossa—diffuse bilateral ischaemia.

Blood culture collected on Day 19 at Cerba Lancet Kenya confirmed profuse growth of Stenotrophomonas (Xanthomonas) maltophilia within 12 - 24 hours of incubation (Figure 10). Antibiogram revealed sensitivity to levofloxacin and resistance to cotrimoxazole. The organism is intrinsically resistant to all carbapenems. Meropenem—administered for 17 days prior to culture confirmation—had zero microbiological activity against this organism throughout the treatment period.

Meropenem was discontinued, and levofloxacin 10 mg/kg once daily (77 mg IV) commenced as the sole antibiotic with confirmed activity against the identified organism, following QTc verification. Vancomycin and metronidazole were continued. Fluconazole was subsequently added following identification of heavy



Aerobic/paediatric blood culture. Collection 08-Apr-2026 18:03. Positive within 12 - 24 hours. Organism: Stenotrophomonas (Xanthomonas) maltophilia—profuse growth. Levofloxacin: Sensitive. Cotrimoxazole: Resistant. Context on report: Meropenem 17 days, PCT 52.83 ng/mL, WBC 28.72 × 10^9/L.

Figure 10. Blood culture final report—Cerba Lancet Kenya (13-Apr-2026).

candiduria (yeast cells ++++) on urinalysis (Day 20) in the context of evolving neutropenia. A note regarding PCT interpretation: following initiation of dexamethasone for cerebral oedema management, PCT fell from 52.83 to 12.0 ng/mL. This decline represents pharmacological suppression of cytokine-mediated PCT induction by corticosteroids and does not reflect microbiological clearance, as the Stenotrophomonas bacteraemia remained untreated until levofloxacin was started. PCT values obtained during corticosteroid therapy must be interpreted with significant caution.

Table 5. Clinical milestones—complete admission timeline.

Date	Day	Milestone
20-Mar	0	Admission—Temp 40.4˚C, HR 169, active tonic-clonic seizure, PCT 26.49 ng/mL, WBC 11.15 × 10^9/L
21-Mar	1	Orotracheal intubation—respiratory failure and septic shock; 3 vasopressors commenced
22-Mar	2	PD commenced—AST 3342 U/L (ischaemic hepatitis)
23-Mar	3	True AST peak 4274 U/L—PCT 20.84 ng/mL—AKI Stage 3 confirmed
26-Mar	6	Creatinine peak 302.6 µmol/L—PD dwell error identified and corrected by physician order
27-Mar	7	pH 7.369—first acid-base improvement after ventilator correction

Continued

31-Mar	11	Vasopressin discontinued—first vasopressor weaned
01-Apr	12	PD holiday commenced—spontaneous UO ≥ 1 ml/kg/hr confirmed
02-Apr	**13**	**Successful extubation—13 days of mechanical ventilation**
03-Apr	14	Bicarbonate 24.2 mmol/L—metabolic acidosis fully resolved
05-Apr	**16**	**PD catheter removed—Creatinine 74.5 µmol/L NORMAL—PCT 1.02 ng/mL**
07-Apr	18	PCT 9.28→52.83 ng/mL—CT brain: ICH + bilateral infarcts + hydrocephalus—APTT 16.3
08-Apr	**19**	**Stenotrophomonas maltophilia confirmed—levofloxacin started—meropenem stopped**
13-Apr	**24**	PCT 1.56 ng/mL (on dexamethasone)—CRP 8.78 mg/L—levofloxacin ongoing
15-Apr	**26**	**Cr 19.7 µmol/L (sarcopenia)—WBC 5.07 × 10^9/L—ANC 1.86 (neutropenia)—PLT 202—MCV 88.6 fL**

Mass transfer model: prescribed clearance 26 × 0.85 = 22.1 units; deviation clearance 15 × 0.95 = 14.2 units; reduction 35.5%. Reference: Twardowski *et al.* [5].

The subclavian CVC catheter tip, collected on Day 18 (07 April 2026) and processed at Cerba Lancet Kenya, demonstrated no bacterial growth on aerobic culture after 48 hours and no anaerobes isolated (Table 5). Gram stain showed no bacteria and no fungal elements. This sterile catheter tip result, in the context of profuse Stenotrophomonas maltophilia growth on simultaneous blood culture, is noteworthy. While the clinical presentation is consistent with a CVC-related bloodstream infection, the negative catheter tip culture introduces diagnostic uncertainty: the source may represent primary bacteraemia from gastrointestinal translocation in a critically ill patient with prolonged broad-spectrum antibiotic exposure, rather than classical catheter colonisation with secondary bacteraemia. Differential time-to-positivity analysis was not performed.

Urinalysis on Day 20 (09 April 2026) demonstrated turbid urine with 3 - 5 pus cells/HPF, >10 RBCs/HPF (microscopic haematuria), protein +, blood ++, and critically, yeast cells ++++—a heavy fungal burden strongly suggestive of candiduria. In the context of severe neutropenia (ANC nadir 0.72 × 10^9/L), prolonged broad-spectrum antibiotics (ceftriaxone, gentamicin, meropenem 17 days, levofloxacin), and an indwelling urinary catheter, this finding raises significant concern for invasive candidiasis. The microscopic haematuria (RBCs > 10/HPF) with proteinuria may reflect ongoing renal parenchymal injury, urinary catheter-related trauma, or fungal-mediated mucosal inflammation. Urine culture for fungal speciation was not available. Empirical fluconazole was commenced, given the heavy urinary yeast burden in the context of severe neutropenia and prolonged broad-spectrum antibiotic exposure [10].

Table 6. Follow-up inflammatory and haematological markers—09 to 15 April 2026.

Date	Day	WBC ($\times 10^9$/L)	Neut %	ANC ($\times 10^9$/L)	Hb (g/dL)	MCV (fL)	PLT ($\times 10^9$/L)	PCT (ng/mL)	CRP (mg/L)	Clinical Context
09-Apr	**20**	13.04	—	6.76	—	—	156	—	—	Yeast ++++; CVC tip sterile
11-Apr	**22**	4.74	—	2.49	—	—	—	11.75	—	Cr 27.2 (sarcopenia); Na 133
13-Apr	**24**	—	—	—	—	—	—	1.56	8.78	On levofloxacin + dexamethasone
15-Apr	**26**	**5.07** ↓	**36.5** ↓	**1.86** ↓	11.4	**88.6** ↑	202	—	—	Neutropenia; MCV shift 76.8 → 88.6

↓ Below reference range. ↑ Above reference range. PCT on Day 24 confounded by concurrent dexamethasone (see Section 3.4). Admission WBC 11.15 × 10^9/L; Day 19 WBC 28.72 × 10^9/L.

2.7. Follow-Up Investigations (Days 24 - 26)

Follow-up investigations obtained on Days 24 - 26 (13 - 15 April 2026) provided important data on the trajectory of the Stenotrophomonas bacteraemia and the systemic consequences of prolonged critical illness. Procalcitonin on Day 24 was 1.56 ng/mL—a substantial decline from the peak of 52.83 ng/mL on Day 19, though interpretation remains confounded by concurrent dexamethasone therapy (see Section 3.4). CRP on Day 24 was 8.78 mg/L—elevated above the admission value of 1.60 mg/L—consistent with ongoing systemic inflammation from the Stenotrophomonas BSI despite levofloxacin initiation.

Kidney function testing on Day 26 demonstrated creatinine 19.7 µmol/L and urea 27.09 mg/dL. The creatinine value was markedly below the pre-AKI baseline of 72.2 µmol/L and does not represent supranormal renal function; rather, it reflects critical illness-associated sarcopenia with depleted creatinine-generating skeletal muscle mass following 26 days of critical illness, including 13 days of mechanical ventilation with neuromuscular blockade. Urea normalisation to 27.09 mg/dL (from a peak of 106.84 mg/dL) confirmed resolution of the uraemic state.

Full haemogram on Day 26 revealed significant post-sepsis haematological changes. WBC had fallen from 28.72 × 10^9/L on Day 19 to 5.07 × 10^9/L with neutropenia (ANC 1.86 × 10^9/L, neutrophil fraction 36.5%)—consistent with post-sepsis bone marrow recovery lag, potentially compounded by levofloxacin-associated myelosuppression. Relative lymphocytosis (53.1%) accompanied the neutropenia. MCV had shifted from 76.8 fL at admission (microcytic, consistent with iron deficiency) to 88.6 fL—a macrocytic shift potentially attributable to folate or vitamin B12 depletion during prolonged critical illness, reticulocytosis, or critical illness dyserythropoiesis. Haemoglobin remained stable at 11.4 g/dL. Platelets recovered to 202 × 10^9/L following the coagulopathy episode. RDW was elevated at 16.5%, reflecting anisocytosis from mixed erythropoietic populations during re-

covery. These follow-up haematological and inflammatory marker data are presented in Table 6.

2.8. Echocardiographic Assessment (Day 28)

Transthoracic echocardiography was performed on Day 28 (17 April 2026) using a Mindray system with an S4-1 phased array transducer. Standard paediatric views were obtained, including apical four-chamber, parasternal long axis (PLAX), parasternal short axis (PSAX), and subcostal views, with color Doppler interrogation of the valvular planes (Figure 11).

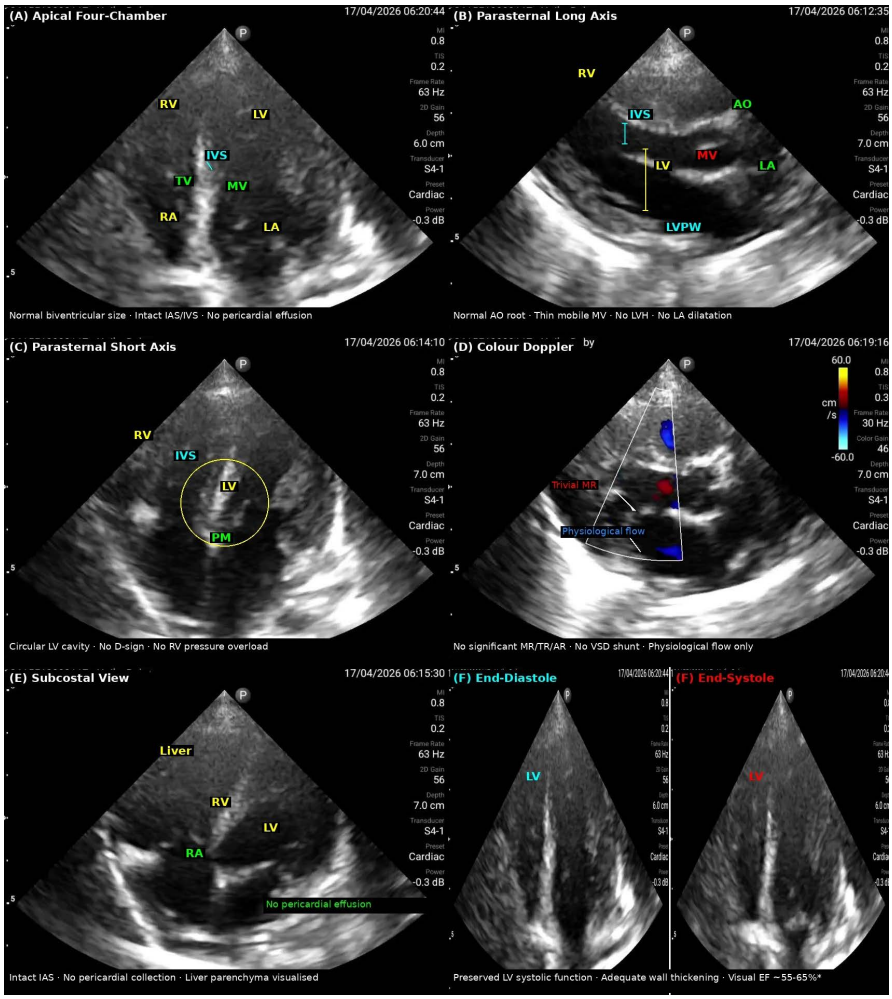


Figure 11. Annotated transthoracic echocardiography—Day 28 (17-Apr-2026). (A) Apical four-chamber: normal biventricular size, labeled LV, RV, LA, RA, IVS, MV, TV. Intact septa, no pericardial effusion. (B) Parasternal long axis: normal aortic root, thin mobile mitral valve, no LVH or LA dilatation. IVS and LVPW measurement lines are shown. (C) Parasternal short axis at papillary muscle level: circular LV cavity (yellow outline) confirming no D-sign and no RV pressure overload. (D) Color Doppler: trivial MR only, no significant valvular regurgitation, physiological transvalvular flow. (E) Subcostal: intact IAS, no pericardial effusion, liver parenchyma visualised. (F) End-diastole vs end-systole: preserved LV systolic function with adequate wall thickening; visual EF ~55% - 65%.* Mindray S4-1 transducer, cardiac preset. *Visual estimate—formal Simpson's biplane not performed.

Left ventricular systolic function was preserved with a hyperdynamic circulation pattern—small LV cavity size with vigorous systolic wall thickening across all segments, consistent with low systemic vascular resistance. No regional wall motion abnormalities were identified. The interventricular septum and LV posterior wall thickness were within normal limits for age, with no evidence of hypertrophy. The mitral valve leaflets appeared structurally normal with appropriate excursion; the aortic root was normal in calibre. Color Doppler demonstrated only physiological or trivial valvular regurgitation—no haemodynamically significant mitral regurgitation, tricuspid regurgitation, or aortic regurgitation was identified. The interatrial and interventricular septa appeared intact with no evidence of ASD or VSD. No pericardial effusion was identified.

PSAX views demonstrated a circular LV cavity without septal flattening, arguing against significant RV pressure overload at the time of imaging. The RV appeared proportionate on apical four-chamber views, though formal TAPSE measurement was limited by the absence of dedicated M-mode acquisition at the tricuspid annulus. Subcostal views confirmed no pericardial effusion and partially visualised the IVC.

The echocardiographic haemodynamic phenotype was consistent with a post-septic distributive circulation: preserved to hyperdynamic LV systolic function with low systemic vascular resistance (vasoplegia), rather than primary cardiogenic dysfunction [12] (**Figure 12**). This distinction is clinically significant as it defines the haemodynamic management strategy—vasopressor optimisation and SVR augmentation rather than inotropic support. The absence of pericardial effusion, significant valvular disease, and structural cardiac abnormality was reassuring following prolonged vasopressor-dependent septic shock requiring three simultaneous vasoactive agents (**Figure 13**). M-mode at the PLAX LV level demonstrated a fractional shortening of approximately 45% (normal 28% - 44%)—quantitatively hyperdynamic, confirming the 2D visual impression. LVIDd was approximately 20 mm (small for age; normal ~22 - 28 mm for a 10-month-old), consistent with low SVR and relative intravascular hypovolaemia. IVSd and LVPWd were both approximately 4 mm—normal, with no hypertrophy. PW Doppler at the mitral valve tips demonstrated E wave velocity approximately 45 - 50 cm/s and A wave approximately 30 - 35 cm/s, yielding an E/A ratio of approximately 1.4 - 1.5—within the normal range for age and excluding significant diastolic dysfunction. PW Doppler with color interrogation confirmed trivial MR and trivial AR on spectral analysis, with no haemodynamically significant regurgitation (**Figure 14**). Tissue Doppler imaging was not available on this system; formal e', s', and TAPSE measurements are recommended on follow-up.

Clinical note: Amlodipine was being administered at the time of echocardiography. Given the echocardiographic confirmation of vasoplegia with low SVR, calcium channel blockade is haemodynamically counterproductive, and its discontinuation was recommended.

Distributive (vasoplegic) circulation confirmed: preserved/hyperdynamic LV

function with low SVR, proportionate RV without D-sign, no significant valvular or structural abnormality. Key management implication: discontinuation of amlodipine (calcium channel blockade worsens vasoplegia), vasopressor optimisation, and restrictive fluid strategy. Follow-up echo recommended with M-mode (FS%, TAPSE), PW Doppler (E/A), TDI (e', s'), and IVC collapsibility index.

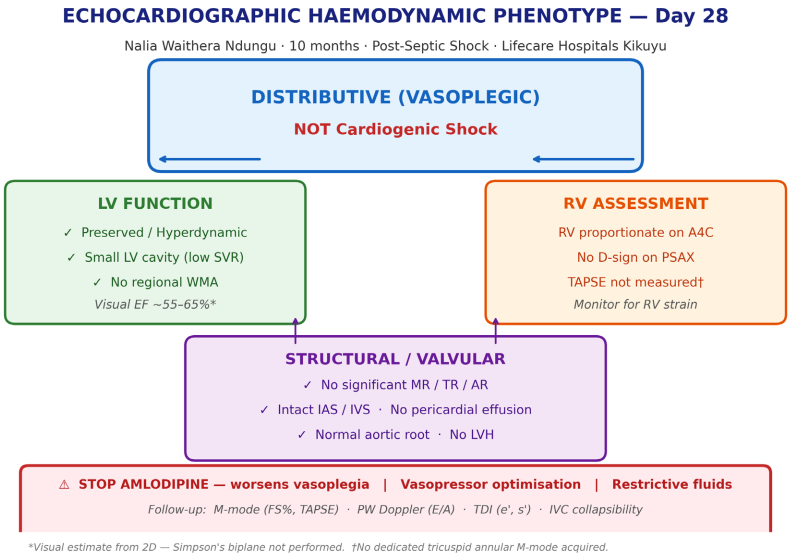
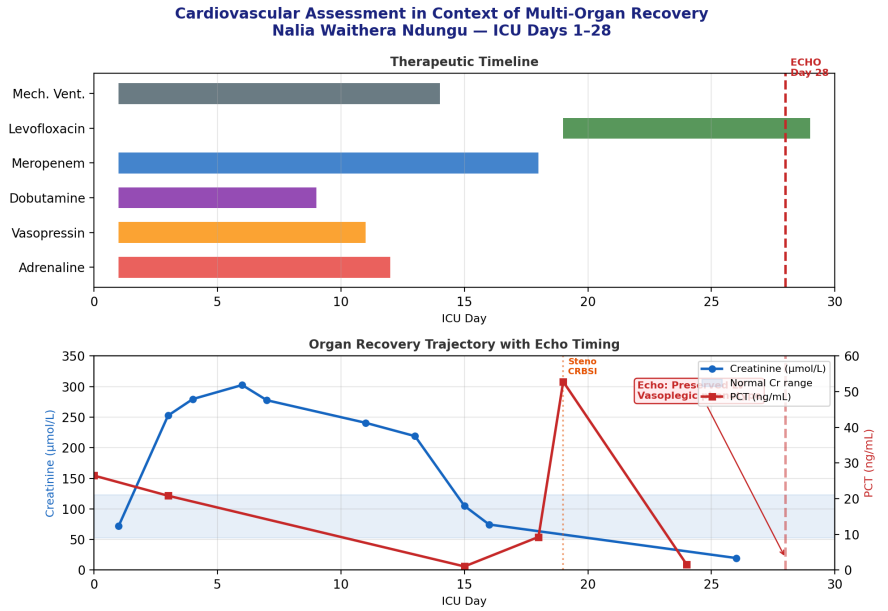


Figure 12. Echocardiographic haemodynamic phenotype summary—Day 28.

Table 7. Echocardiographic findings summary—Day 28 (17 April 2026).

Parameter	Finding	Clinical Significance
LV Systolic Function	Preserved/Hyperdynamic	Excludes Cardiogenic Shock
LV Cavity Size	Small	Low SVR/Vasoplegia
Visual EF*	~55% - 65%	Adequate Pump Function
Regional WMA	None Identified	No Focal Ischaemia
IVS/LVPW Thickness	Normal for Age	No LVH
RV Size	Proportionate	No RV Dilatation
PSAX LV Geometry	Circular	No D-Sign → No RV Pressure Overload
TAPSE	Not Measured†	Requires M-Mode on Follow-Up
Mitral Valve	Thin, Mobile	Structurally Normal
Colour Doppler	Trivial MR Only	No Significant Regurgitation
IAS/IVS	Intact	No ASD/VSD
Pericardial Effusion	None	No Tamponade Physiology
Aortic Root	Normal Calibre	No Aortic Pathology
IVC	Partially Visualised	Formal IVCCI Not Calculated

Structured tabulation of all parameters assessed, findings, and clinical significance. *Visual estimate from 2D echo. †No dedicated tricuspid annular M-mode acquired.



Top: Therapeutic timeline showing vasopressor duration, antibiotic courses, and mechanical ventilation period, with echocardiography timing at Day 28. Bottom: Organ recovery trajectory plotting creatinine (blue) and PCT (red) with echo finding annotation. The echo at Day 28 confirmed preserved LV function despite prior triple vasopressor requirement, with a vasoplegic haemodynamic phenotype. Stenotrophomonas BSI event (Day 19) and the associated PCT spike are highlighted.

Figure 13. Cardiovascular assessment in the context of multi-organ recovery.

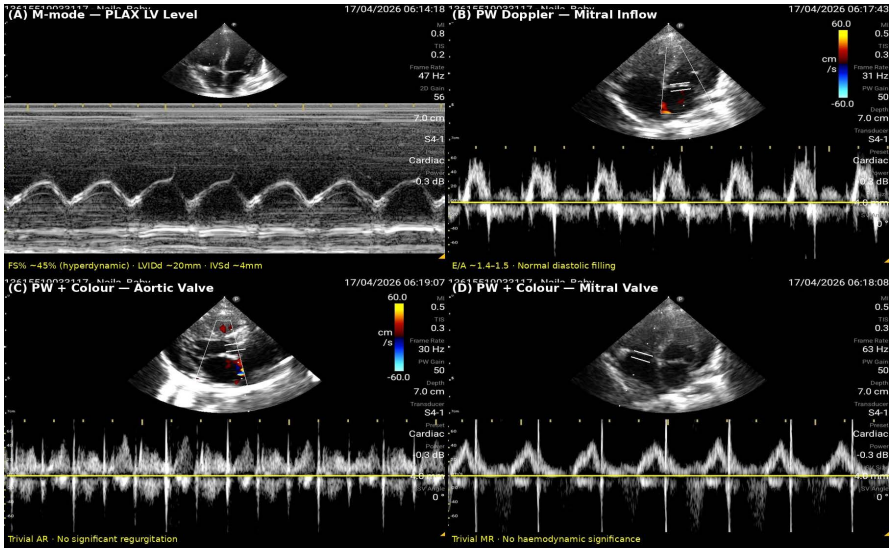
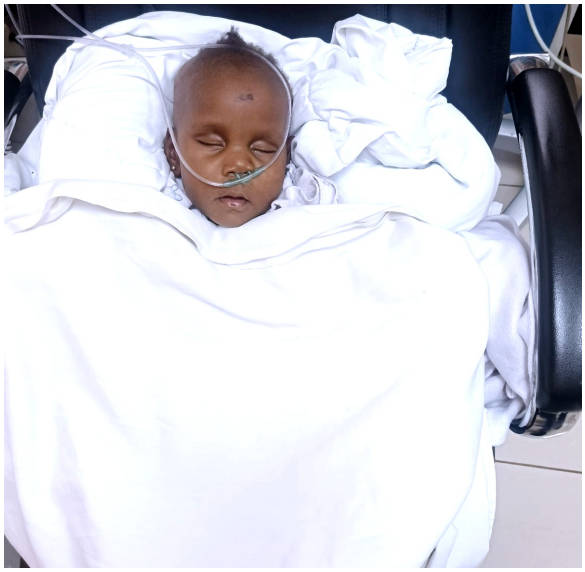


Figure 14. M-mode and PW Doppler—Day 28 (17-Apr-2026). (A) M-mode at PLAX LV level: fractional shortening ~45% (hyperdynamic; normal 28% - 44%), LVIDd ~20 mm (small for age), IVSd ~4 mm, and LVPWd ~4 mm (normal). Quantitative confirmation of hyperdynamic LV function with low SVR. (B) PW Doppler mitral inflow: E wave ~45 - 50 cm/s, A wave ~30 - 35 cm/s, E/A ratio ~1.4 - 1.5 (normal for age)—no diastolic dysfunction, normal filling pattern. (C) PW Doppler with color at the aortic valve: trivial AR only. (D) PW Doppler with color at mitral valve: trivial MR confirmed on spectral trace. Mindray S4-1, cardiac preset, SV 4.0 mm.



The patient was following successful extubation (Day 13) and completion of peritoneal dialysis (Day 16). The patient is breathing comfortably on low-flow nasal cannula oxygen, wrapped in standard hospital linen. Contrast with Figure 1 (peak multi-organ support phase): orotracheal tube, multiple peripheral IV lines, concurrent vasopressor infusions, neuromuscular blockade, and invasive monitoring have all been discontinued, reflecting clinical stabilisation after resolution of MODS with AKI Stage 3 and ischaemic hepatitis. This image was obtained during the post-extubation recovery phase prior to the onset of the late Stenotrophomonas maltophilia bloodstream infection.

Figure 15. Post-recovery—patient on nasal cannula oxygen.

2.9. Final Clinical Outcome and Follow-Up Plan

At last clinical assessment on Day 28, the patient was haemodynamically stable without vasopressor support, tolerating enteral feeds via nasogastric tube, and breathing spontaneously on low-flow nasal cannula oxygen (Figure 15) (1 L/min, FiO_2 24%) (Table 7). Neurological examination demonstrated persistent generalised hypotonia, absent head control, inconsistent visual fixation to light, and intermittent myoclonic jerks of the upper limbs controlled with phenobarbitone. The anterior fontanelle was full but soft. The modified paediatric Glasgow Coma Scale was 10/15 (E3 V3 M4). These findings were consistent with the extensive periventricular leukomalacia, bilateral temporal and cerebellar hypodensity, and ex vacuo hydrocephalus documented on CT brain. Renal function had recovered completely with creatinine 19.7 μmol/L and urea 27.09 mg/dL on Day 26 (creatinine 27.2 μmol/L at discharge on Day 32); the low creatinine relative to the admission baseline (72.2 μmol/L) reflected critical illness sarcopenia rather than ongoing renal dysfunction. Platelet count had recovered to 202×10^9/L following the coagulopathy episode. The patient was discharged on Day 32 (21 April 2026) in stable condition on oral phenobarbitone, oral levofloxacin (to complete a 14-day course), oral fluconazole, and nasogastric feeds. A paediatric neurology and neurosurgery referral was arranged for assessment of the hydrocephalus and consideration of ventriculoperitoneal shunt insertion if progressive. Repeat CT brain was

scheduled at 4 - 6 weeks to monitor ventricular size. Physiotherapy and neurodevelopmental supportive care were commenced with an outpatient review. Nephrology follow-up was scheduled at 3 months for renal function surveillance and chronic kidney disease screening, given the severity and 16-day duration of AKI Stage 3. The family received counseling regarding the guarded neurodevelopmental prognosis.

3. Discussion

3.1. Peritoneal Dialysis as Sole RRT in Paediatric MODS

This case demonstrates survival from severe sepsis-induced MODS with AKI Stage 3, ischaemic hepatitis with peak AST 4274 U/L, and 13 days of mechanical ventilation in a resource-limited sub-Saharan African PICU without access to CRRT. The case supports and extends existing evidence for PD as an effective sole RRT in paediatric AKI. Rewa and Bagshaw demonstrated that PD can achieve adequate small solute clearance when delivered with short dwell times and high cycle frequencies [4]. Studies from sub-Saharan Africa have reported PD as the predominant RRT modality in paediatric AKI, with outcomes comparable to CRRT when prescription fidelity is maintained [13]. The resource-limited context of paediatric critical care in sub-Saharan Africa, where cautious fluid resuscitation strategies are essential [14], further underscores the importance of optimising available RRT modalities.

The concurrent occurrence of severe ischaemic hepatitis—rarely documented in the published African paediatric PD literature—introduced additional pharmacological complexities. The selection of cisatracurium over rocuronium and vecuronium, both of which undergo substantial hepatic and renal elimination, represents a deliberate pharmacological decision guided by the degree of hepatic impairment, and is consistent with evidence-based guidance on drug selection in multi-organ failure [8]. Vitamin D insufficiency (13 ng/mL), identified on Day 7, contributed to persistent ionised hypocalcaemia and may have impaired innate immune function, representing a potentially modifiable risk factor in critically ill African infants where vitamin D deficiency is prevalent [15].

3.2. The PD Prescription Deviation—A Patient Safety Contribution

The prescription deviation documented in this case—unauthorised extension of PD dwell time from 20 to 60 - 120 minutes—represents, based on our PubMed search, one of the first paediatric case reports providing biochemical quantification of the clinical impact of such an error in a resource-limited African setting. Recent paediatric PD literature from sub-Saharan Africa [16] [17] documents comparable acute PD outcomes but does not describe quantified dwell-time deviations, supporting the rarity but not the uniqueness of this observation. This claim remains limited by the non-systematic nature of the search. The physiological argument against extended dwell time in acute PD is grounded in peritoneal mem-

brane transport kinetics. Twardowski and colleagues demonstrated that 80% - 90% of maximum small solute equilibration occurs within 20 - 25 minutes of dwell, after which the peritoneal concentration gradient is substantially exhausted [5]. Extension beyond this plateau yields minimal additional per-cycle clearance while reducing the achievable cycle frequency—the primary driver of total daily PD dose.

Three analytical estimates were applied to quantify the impact of the deviation. Cycle frequency analysis yielded a 42% reduction. Dialysate volume analysis yielded a 55% - 60% reduction. Mass transfer modelling incorporating the plateau effect yielded an estimated 35% - 40% net clearance reduction—the most academically defensible figure, as it accounts for the fact that longer dwells are not entirely without clearance benefit per cycle. The five-day period of subtherapeutic dialysis delivery prolonged the patient's exposure to uraemia, acidosis, and the systemic inflammatory burden of under-dialysed AKI during the period of maximum haemodynamic compromise. These clearance estimates are model-based projections rather than measured outcomes: they assume stable peritoneal membrane permeability, negligible ultrafiltration-driven solute removal, and constant endogenous solute generation rates across all phases—conditions that were not independently verified but represent standard pharmacokinetic approximations for acute PD in this clinical context.

The human factors dimension is central to the patient safety message. In resource-limited PICUs, nursing staff managing PD are frequently not specialist-dialysis-trained, and the intuitive assumption that longer dwell time equates to greater dialysis efficacy—while physiologically incorrect—is a predictable cognitive error in the absence of targeted training. Prevention requires structural interventions: prominently displayed bedside prescriptions specifying dwell time in large font, nursing competency frameworks specific to PD dwell time physiology, timed cycle documentation with nurse signature, and physician-led dwell time verification at every clinical assessment.

3.3. Stenotrophomonas Maltophilia Bloodstream Infection—Antibiotic Stewardship Lesson

The identification of Stenotrophomonas maltophilia on blood culture on Day 19 provides the unifying pathological explanation for the neurocritical complications arising on Day 18. Stenotrophomonas maltophilia is a non-fermentative Gram-negative aerobic bacillus characterised by intrinsic resistance to all carbapenems through chromosomal L1 metallo-beta-lactamase and L2 serine-beta-lactamase [6] [18]. It is an opportunistic nosocomial pathogen associated with prolonged ICU stay, indwelling central venous catheters, mechanical ventilation, and extended broad-spectrum antibiotic exposure—all of which were present in this patient.

Seventeen days of meropenem—while initially appropriate for the Gram-negative enteric sepsis—created ideal selective pressure for Stenotrophomonas while

eliminating susceptible competing flora. The organism was recovered from a bacteraemic episode with profuse growth, with the subclavian CVC (*in situ* for 12 days) representing the most likely but unconfirmed portal of entry, given the sterile catheter tip culture. The absence of a positive catheter tip culture and the lack of differential time-to-positivity testing preclude definitive IDSA CRBSI confirmation, supporting a stewardship approach that treats the CVC as a suspected rather than proven source. Recent paediatric series report S. maltophilia bloodstream infection mortality of 33% - 42% in immunocompromised children [17], underscoring the severity of this organism in the paediatric ICU population. PCT rising from 1.02 ng/mL at Day 15 to 52.83 ng/mL at Day 19—the highest inflammatory marker of the entire admission. The resulting Gram-negative bacteraemia was temporally associated with sepsis-associated coagulopathy suggestive of DIC (APTT 16.3 seconds), with simultaneous microvascular thrombosis (left parietal infarct, bilateral temporal and cerebellar ischaemia) and haemorrhage (right parietal ICH 7.1 × 6.6 cm). This paradoxical simultaneous thrombosis and haemorrhage is a clinical hallmark suggestive of the mixed consumption phase of DIC.

The antibiogram confirmed levofloxacin as the only active agent—cotrimoxazole was resistant, and carbapenems were intrinsically ineffective. This case illustrates a critical antibiotic stewardship lesson: prolonged carbapenem therapy in patients with central venous catheters and prolonged ICU admission selects for carbapenem-resistant nosocomial organisms. De-escalation from broad-spectrum coverage to the narrowest spectrum consistent with clinical response should be initiated at the earliest safe opportunity, guided by serial PCT, clinical response, and culture results.

3.4. PCT Interpretation under Corticosteroid Therapy

The PCT decline from 52.83 to 12.0 ng/mL following dexamethasone initiation illustrates a clinically important but underappreciated confound: corticosteroids suppress cytokine-mediated PCT induction at the transcriptional level, causing pharmacological PCT reduction independent of bacterial load [7] [19]. This PCT decline cannot be interpreted as microbiological clearance of the Stenotrophomonas bacteraemia, as no effective antibiotic had yet been administered. Clinicians managing patients on corticosteroids must use alternative markers—particularly blood culture clearance, WBC trend, clinical haemodynamic signs, and CRP—rather than relying on PCT as the primary treatment response marker.

3.5. The Renal-Respiratory Interdependence

An important mechanistic relationship in this case was the direct impact of renal recovery—or its delay—on ventilator weaning. Under-dialysis during the prescription deviation period perpetuated metabolic acidosis (pH 7.223 on Day 6), fluid retention, uraemic toxin accumulation, and hypokalaemia—each of which independently impeded ventilator liberation. Conversely, optimal PD delivery from Day 7 onward enabled acid-base normalisation, fluid removal, and the haemody-

namic stability that made the successful spontaneous breathing trial possible on Day 12. This renal-respiratory interdependence supports the concept that in paediatric MODS, renal replacement adequacy is not merely a renal therapy but a systemic stabilisation strategy that enables recovery of other organ systems.

3.6. Limitations

This case has limitations inherent to its resource-limited setting. The causative organism of the initial bacterial gastroenteritis was not identified on culture. Formal illness severity scoring (PELOD-2, PRISM III) was not performed prospectively. Kt/V was not directly measured; clearance estimates are derived from proportional modelling. Fibrinogen and D-dimer were not obtained during the coagulopathy episode. MRI brain—the preferred modality for characterising hypoxic-ischaemic injury and distinguishing acute from subacute infarction—was not available. Repeat blood culture following levofloxacin initiation was not obtained; microbiological clearance of the Stenotrophomonas bacteraemia is therefore not confirmed. The subclavian CVC catheter tip culture was sterile (no bacterial growth after 48 hours); definitive IDSA diagnostic criteria for catheter-related bloodstream infection were therefore not met, and the catheter origin of the Stenotrophomonas bacteraemia remains presumptive rather than confirmed. These limitations reflect the real-world constraints of paediatric critical care delivery in sub-Saharan Africa and are documented transparently to contextualise the clinical decisions made.

4. Conclusions

This case report documents three simultaneous and interconnected contributions to the paediatric critical care literature. First, it demonstrates that peritoneal dialysis can serve as effective sole renal replacement therapy in severe paediatric MODS with AKI Stage 3, achieving complete renal recovery with creatinine normalisation over 16 days in a resource-limited sub-Saharan African PICU [20]. Second, it provides a rare example of model-based biochemical quantification of a PD dwell-time prescription deviation in the paediatric literature—an estimated 35% - 40% net reduction in small solute clearance and an approximately 48-hour delay in the onset of creatinine decline, based on proportional modelling rather than directly measured Kt/V—with direct preventable patient safety implications for PICUs globally. Third, it documents a late Stenotrophomonas maltophilia bloodstream infection with suspected but unconfirmed catheter association after prolonged carbapenem exposure, temporally associated with sepsis-associated coagulopathy suggestive of DIC, intracranial haemorrhage, bilateral cerebral infarction, and hydrocephalus—a rare and severe neurocritical complication sequence with an important antibiotic stewardship lesson.

The four actionable recommendations arising from this case are: strict adherence to PD dwell time prescriptions with nurse-level competency assessment and physician verification at every round; de-escalation of broad-spectrum carbapenem

therapy at the earliest clinically safe opportunity to prevent nosocomial selection of resistant organisms; prompt removal of central venous catheters when clinically feasible in the post-resuscitation phase; and cautious interpretation of PCT values in patients receiving corticosteroid therapy.

Data Availability

All clinical data supporting the findings of this case report are presented within the published article. No additional datasets were generated or analysed. This case report was prepared in accordance with the CARE (CAse REport) guidelines checklist (Gagnier *et al.*). A completed CARE checklist is available from the corresponding author upon request.

Patient Consent

Written informed consent for publication of this case report and all accompanying clinical images was obtained from the parent and legal guardian of the patient prior to submission. A signed copy is retained in the patient's medical record.

Ethics Approval

This case report was conducted in accordance with the Declaration of Helsinki (2013 revision). Institutional ethics review was obtained from the Lifecare Hospitals Research and Ethics Committee, Nairobi, Kenya.

Author's Contributions

YTN: primary intensivist and nephrologist; clinical management; conceptualization; data collection and verification; manuscript drafting, figure generation, and revision. The author read and approved the submitted version.

Acknowledgements

The author thanks the nursing staff of the Paediatric Intensive Care Unit at Lifecare Hospitals Kikuyu for their dedication throughout this prolonged admission; the laboratory teams at Lifecare Hospitals and Cerba Lancet Kenya for timely investigation processing; Dr. Onkoba V. for radiological interpretation; and the patient's family for their trust and consent to publication.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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