KJMHS KORDOFAN JOURNAL OF MEDICAL AND HEALTH SCIENCES

KJMHS 2026; Vol. 3 (Issue 1) **Online ISSN: 3027-5601** https://doi.org/ 10.70946/KJMHS3-1-26P1

Do We Need to Follow International Guidelines for Empirical Antibiotics Selection in Critically Sick Children? A Case series

Anasalwogud Ahmed[1], Alddai Alnair[1], Aaqib Ahmed[1], Maged Nabawi[1], Ayman Gouda[1], Mohamed Babiker[2]

[1]Pediatric Intensive Care, Al Qassimi Hospital for Women and Children (AQWCH), United Arab Emirates,
[2]Department of Pediatric, Al Qassimi Hospital for Women and Children (AQWCH), United Arab Emirates

Abstract

Background: Sepsis is a leading cause of pediatric critical illness and mortality. Choosing empiric antibiotics based solely on international guidance may be inadequate where local resistance patterns differ. **Methods** (Case Series Description): We describe two infant cases with rapidly progressive septic shock, purpuric/necrotic skin lesions, and multi-organ failure. **Results:** Blood/CSF/lesion cultures in both cases grew Pseudomonas aeruginosa resistant to initial ceftriaxone+vancomycin; one isolate was imipenem-susceptible after escalation. Despite maximal organ support, both infants died. **Conclusion:** While international guidelines standardize care, empiric therapy in critically ill children should be tailored to local epidemiology and AMR surveillance, ensuring early coverage for Pseudomonas where prevalence warrants.

Correspondence to: Anasalwogud Ahmed: anasalwogud@gmail.com

Keywords: *International Guidelines, Empirical Antibiotics, Critically Sick, Children*

Introduction

Sepsis continues to be the primary cause of serious illness that leads to death in children who need intensive care worldwide [1]. The timely selection and proper use of empirical antibiotics stands as a fundamental treatment approach that directly shapes patient results [2, 5]. International guidelines establish general recommendations for initial antibiotic therapy that rely on typical bacterial agents alongside their susceptibility patterns [3, 6]. Local epidemiological patterns and increasing antimicrobial resistance cause significant therapeutic issues because initial treatment may prove insufficient [9]. This paper describes two fatal cases of infant sepsis from Pseudomonas aeruginosa which showed resistance to standard empirical antibiotics to underscore the challenges of treating severe pediatric infections and to examine the need for updating current empirical antibiotic protocols.

Case Series

Case 1 Presentation

The emergency department received a 4-month-old infant with fever and poor feeding alongside purplish skin lesions that spread quickly during one week. The patient showed signs of septic shock with poor perfusion and hypotension (BP 52/39(43)mmHg) and hypoxia (SPO2 84%) and lethargy when first received in the hospital. The patient displayed skin lesions which were purpuric across the entire body while presenting severe hypoglycemia with RBS 1mmol/L (figure 1). The patient received septic shock resuscitation according to guidelines which included intubation and mechanical ventilation. The patient underwent full septic workup and started intravenous antibiotics of ceftriaxone and vancomycin. The medical staff transferred the patient to the Pediatric Intensive Care Unit (PICU) where a central venous catheter was placed.

KJMHS KORDOFAN JOURNAL OF MEDICAL AND HEALTH SCIENCES
KJMHS 2026; Vol. 3 (Issue 1) **Online ISSN: 3027-5601** https://doi.org/ 10.70946/KJMHS3-1-26P1

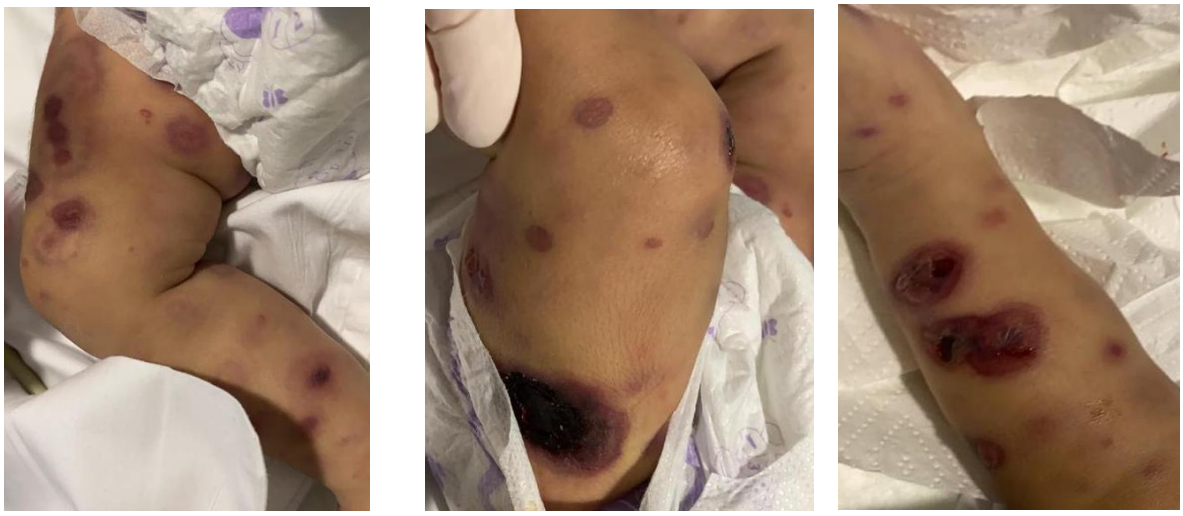

Figure 1 (case 1). Generalized purpuric skin lesions of varying size distributed over the entire body at initial presentation, which rapidly evolved within hours to confluent areas with necrotic changes, consistent with fulminant septic vasculopathy.

The patient needed high-dose inotropic support through epinephrine and norepinephrine alongside hydrocortisone for treating refractory shock. The patient presented with leucopenia and lymphopenia and anemia which suggested bone marrow suppression according to initial test results (Table 1). The patient displayed systemic inflammation. Coagulation tests showed severe abnormalities, indicating coagulopathy consistent with disseminated intravascular involvement.

Table 1 (Case 1). The Laboratory Results Summary

Category	Test	Result	Reference Range
Hematology	WBC	2.82 ×10³/µL	5.0 − 15.0 ×10³/µL
Hematology	ANC	1.83 ×10³/µL	1.5 − 8.0 ×10³/µL
Hematology	Lymphocytes	0.54 ×10³/µL	2.0 − 8.0 ×10³/µL
Hematology	Hemoglobin (Hb)	8.2 g/dL	11.0 − 14.0 g/dL
Hematology	Platelet Count	175 ×10³/µL	150 − 450 ×10³/µL
Inflammatory Marker	CRP	177.60 mg/L	<5 mg/L
Coagulation Profile	PT	44.4 seconds	11.0 − 13.5 seconds
Coagulation Profile	PTT	57.7 seconds	25.0 − 35.0 seconds
Coagulation Profile	INR	3.75	0.8 − 1.2
Renal Function	BUN	11.47 mmol/L	2.5 − 7.0 mmol/L
Renal Function	Creatinine	69.30 µmol/L	18 − 35 µmol/L (infants)
Liver Function	Bilirubin	34.60 µmol/L	<17 µmol/L
Liver Function	AST	95 U/L	10 − 40 U/L

Initial investigations revealing profound leucopenia, anemia, and coagulopathy, together with evidence of acute kidney and liver injury. The laboratory abnormalities highlight multi-organ dysfunction in parallel with the rapid clinical progression to septic shock and disseminated intravascular coagulation (DIC) within the first 15 hours of hospital admission.

The laboratory tests indicated acute kidney injury through elevated BUN and creatinine levels. The liver function tests revealed elevated AST and hyperbilirubinemia which

KJMHS 2026; Vol. 3 (Issue 1) **Online ISSN: 3027-5601** https://doi.org/ 10.70946/KJMHS3-1-26P1

suggested liver involvement. The combination of severe sepsis/meningitis with multi-organ dysfunction syndrome included circulatory and respiratory failure and AKI and pancytopenia and coagulopathy. The PICU provided multiple organ support to the patient who continued worsening until a cardiorespiratory arrest occurred. The medical team started CPR according to PALS protocol yet failed to save the patient. The hospital stay lasted 15 hours. The follow up laboratory cultures results confirmed that Pseudomonas aeruginosa infection from peripheral and central blood cultures and cerebrospinal fluid (CSF) and skin lesion swab culture. The test results revealed that the isolated bacteria demonstrated resistance to the prescribed empirical antibiotics at first.

Case 2 Presentation

A 6-month-old male infant presented with initial symptoms of fever, cough, vomiting, diarrhea, and poor oral intake. Subsequently, he developed a progressive generalized purplish skin rash that evolved into necrotic skin lesions over time (Fig.2). Sepsis was suspected, and a full septic workup was done before initiating empirical antibiotics (ceftriaxone and vancomycin). After first 12 hours in pediatric ward, the patient transfer to the PICU due to low BP 58/31mmHg and features of uncompensated shock which was unresponsive to fluid resuscitation.

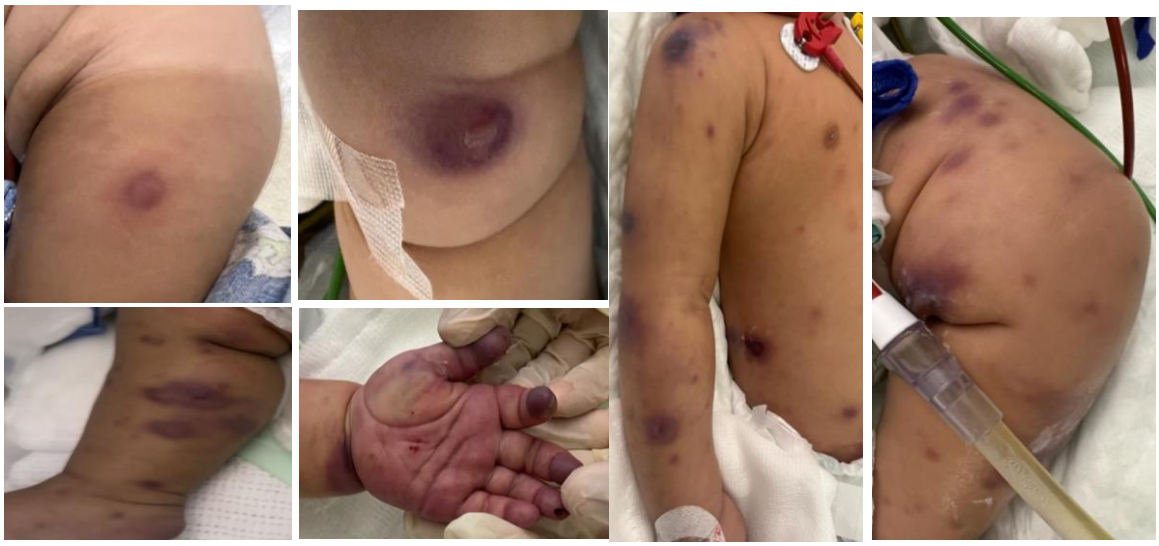

Figure 2 Generalized purpuric skin lesions of varying sizes at presentation, which progressed over 48 hours to necrotic lesions with deep discoloration and tissue breakdown, reflecting the rapid evolution of septic vasculopathy.

The patient in the PICU showed signs of neurological depression through sleepiness and irritability while his GCS score reached 10/15. He needed elective intubation and received high-dose inotropic support (epinephrine and norepinephrine together with calcium infusion and steroids for treating refractory catecholamine-resistant septic shock). The patient developed supraventricular tachycardia (SVT) showed in the ECG which returned to normal after receiving his third dose of adenosine. The echocardiogram showed that left ventricular function was moderate and PVR was elevated while the ejection fraction measured at 50%. The patient showed major myocardial damage through elevated Troponin I levels and extremely high BNP measurements (Table 2). The hematological tests showed bone marrow suppression together with anemia and severe thrombocytopenia. The coagulation studies indicated fulminant DIC because INR levels were elevated while PT and

KJMHS 2026; Vol. 3 (Issue 1) **Online ISSN: 3027-5601** https://doi.org/ 10.70946/KJMHS3-1-26P1

PTT were prolonged and fibrinogen was low and D-Dimer levels were high. The inflammatory markers CRP, IL-6 and procalcitonin showed extreme elevation which indicated both hyperinflammation and probable sepsis. The cerebrospinal fluid analysis revealed slightly elevated glucose levels and normal protein concentration together with lymphocytic cell predominance and gram-negative rod identification which indicated bacterial meningitis.

Table 2 (Case 2). The Laboratory Results Summary

Category	Test	Result	Reference Range
Cardiac Enzymes	Troponin I	366 ng/L	0 – 60 ng/L
Cardiac Enzymes	BNP	35,000 pg/mL	0 – 125 pg/mL
Hematology	Hemoglobin (Hb)	7.9 g/dL	11.0 – 14.0 g/dL
Hematology	Platelet Count	33 ×10⁹/L	150 – 450 ×10⁹/L
Coagulation Profile	INR	>3	0.8 – 1.2
Coagulation Profile	PT	>100 seconds	11 – 13.5 seconds
Coagulation Profile	PTT	>100 seconds	25 – 35 seconds
Coagulation Profile	Fibrinogen	0.5 g/L	2 – 4 g/L
Coagulation Profile	D-Dimer	8.1 mg/L	0 – 0.5 mg/L
Inflammatory Markers	CRP	285 mg/L	0 – 3 mg/L
Inflammatory Markers	IL-6	>5500 pg/mL	≤4 pg/mL
Inflammatory Markers	Procalcitonin	>10 µg/L	0 – 0.1 µg/L
CSF Analysis	Glucose	5.2 mmol/L	2.2 – 4.4 mmol/L
CSF Analysis	Protein	286 mg/L	150 – 450 mg/L
CSF Analysis	Cell Count	6 /mm³ (85% lymphocytes)	0 – 5 /mm³
CSF Analysis	Culture	Gram-negative rods	Not applicable
Renal Function	Creatinine	128 → 200 µmol/L	18 – 35 µmol/L (infants)
Renal Function	Urea	Elevated	2.5 – 7.0 mmol/L
Renal Function	Urine Output	<0.5 mL/kg/hr	≥1 mL/kg/hr
Liver Function	Albumin	13.6 g/L	21 – 49 g/L
Liver Function	ALT	2087 U/L	15 – 40 U/L
Liver Function	AST	3579 U/L	15 – 37 U/L
Liver Function	Glucose	2 – 3 mmol/L	3.5 – 5.5 mmol/L

Key laboratory findings during PICU admission demonstrating multi-organ dysfunction and fulminant disseminated intravascular coagulation (DIC). Values include markedly elevated inflammatory markers (CRP, IL-6, Procalcitonin), cardiac injury biomarkers (Troponin I, BNP), coagulopathy indices, and progressive renal and hepatic failure. Trends illustrate worsening parameters in parallel with rapid clinical deterioration over the 3-day PICU course.

The PICU team initiated antibiotic dose escalation during the second day of admission after discovering these results and when the patient's condition worsened while patient use intravenous imipenem and vancomycin and clindamycin at renal-adjusted doses. The medical staff gave IVIG to the patient because their condition kept deteriorating. The patient developed acute kidney injury (AKI) with acid base and electrolyte imbalance required correction and further developed renal shutdown that required continuous renal replacement therapy (CRRT). Acute liver injury evidence existed with hypoalbuminemia and elevated ALT and AST levels and continuous hypoglycemia (2–3 mmol/L) which needed a high glucose infusion rate (GIR) of 10 mg/kg/minute.

KJMHS 2026; Vol. 3 (Issue 1) **Online ISSN: 3027-5601** https://doi.org/ 10.70946/KJMHS3-1-26P1

The patient continued to experience severe sepsis/meningitis alongside multiple organ failure despite remaining in a critical state. The skin rash displayed growing deterioration because it expanded in both number and size while developing necrotic features and deepened coloration. The patient received a multidisciplinary team support, while the patient's condition continued to deteriorate until he experienced a fatal cardiorespiratory arrest. The application of High-quality CPR through PALS protocol failed to produce successful results. The patient spent a total of three days inside the PICU. The post-mortem microbiology tests revealed that Pseudomonas aeruginosa spread through peripheral and central blood cultures as well as CSF and skin lesion swab culture. The isolate demonstrated susceptibility to imipenem which became part of the PICU treatment plan during day 2. The bacteria showed resistance to the initial combination of antibiotics that healthcare providers selected.

Discussion

These cases demonstrate the fatal progression of Pseudomonas aeruginosa sepsis in young infants while demonstrating difficulties in choosing appropriate antibiotics at the start of treatment. The two infants demonstrated a fast progression of septic shock and multi-organ failure while their skin developed worsening purpuric lesions. The initial empirical treatment consisting of ceftriaxone and vancomycin according to pediatric sepsis guidelines for common pathogens (e.g. Staphylococcus aureus, Streptococcus pneumoniae, Escherichia coli) [3,6] failed to detect Pseudomonas aeruginosa. This pathogen, though uncommon in community-acquired infections, exhibits intrinsic and adaptive resistance, complicating management [4,7].

The identification delay of pathogen together with delayed antibiotic escalation demonstrated the shortcomings of conventional cultures in critical time-sensitive situations during fulminant sepsis.

International guidelines face challenges when managing atypical or resistant organisms which emerge in certain settings according to these reported cases. Empirical antibiotic protocols should incorporate local resistance patterns and epidemiological data to provide better protection against dangerous pathogens such as Pseudomonas aeruginosa [5,8].

The improvement of severe pediatric sepsis outcomes requires enhanced clinical vigilance together with quick diagnostic tests and ongoing antimicrobial surveillance to optimize empirical treatment approaches [9]:

- In pediatric ICUs with documented Pseudomonas prevalence or rising MDR trends, initial empiric therapy for critically ill infants should consider early antipseudomonal coverage (e.g., a carbapenem or antipseudomonal β-lactam) pending cultures.
- Establish and regularly update a local AMR antibiogram for pediatrics; embed it in sepsis bundles and order sets.

Recommendations

- Implement rapid diagnostics (e.g., blood culture identification panels) to shorten time to targeted therapy.
- Conduct post-prescription review within 24–48 hours to de-escalate or escalate based on microbiology and clinical trajectory.
- Provide unit-level education on selecting empiric therapy that balances timeliness, coverage breadth, and stewardship principles.

Conclusion

While *international guidelines* are vital for standardizing care and preventing delays in sepsis management, local resistance data must update empiric antibiotic choices. The use of broad empiric coverage becomes necessary for critically ill children who are free of prior illness when Pseudomonas or multidrug-resistant prevalence increases in the setting because initial inappropriate treatment may cause fatal outcomes.

KJMHS 2026; Vol. 3 (Issue 1) **Online ISSN: 3027-5601** https://doi.org/ 10.70946/KJMHS3-1-26P1

Acknowledgements
The authors would like to thank the medical and nursing staff of the Pediatric Intensive Care Unit, microbiology laboratory staff,and Emergency Department for their dedicated care of these patients.

References

1. Weiss, S. L., et al. "Surviving Sepsis Campaign International Guidelines for the Management of Septic Shock and Sepsis-Associated Organ Dysfunction in Children." *Pediatric Critical Care Medicine*, vol. 21, no. 2, 2020, pp. e52-e106.
2. Dellinger, R. P., et al. "Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock: 2012." *Critical Care Medicine*, vol. 41, no. 2, 2013, pp. 580-637.
3. Goldstein, B., et al. "International Pediatric Sepsis Consensus Conference: Definitions for Sepsis and Organ Dysfunction in Pediatrics." *Pediatric Critical Care Medicine*, vol. 8, no. 4, 2005, pp. 317-329.
4. Bassetti, M., et al. "Pseudomonas aeruginosa: a pathogen that needs to be taken seriously." *Clinical Microbiology and Infection*, vol. 22, no. 1, 2016, pp. 1-2.
5. Sánchez-Pinto, L. N., et al. "Pediatric Sepsis: a Summary of Current Definitions and Management Recommendations." *Current Pediatrics Reports*, vol. 11, no. 2, 2023, pp. 69-79.
6. de Souza, C. M. A., et al. "Antimicrobial Therapy in Pediatric Sepsis: What Is the Best Strategy?" *Frontiers in Pediatrics*, vol. 10, 2022, p. 830276.
7. Li, Y., et al. "Carbapenem-resistant *Pseudomonas aeruginosa* infections in critically ill children: Prevalence, risk factors, and outcomes." *Frontiers in Public Health*, vol. 11, 2023, p. 1088262.
8. Pediatric Sepsis Diagnosis, Management, and Sub-phenotypes. *Pediatrics*, vol. 153, no. 1, 2023, pp. e2023062967.
9. Matoria, R., & Soni, P. "Antibiotic Strategies for Neonatal Sepsis: Navigating Efficacy and Emerging Resistance Patterns." *European Journal of Pediatrics*, 2025. (As reported in *Times of India*, July 20, 2025).