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Acute Kidney Injury With Septic Shock and Carbapenem-Resistant Acinetobacter baumannii Peritonitis in Infancy: A Rare Case Report

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Abstract

Acinetobacter baumannii (*A. baumannii*) is an opportunistic multidrug-resistant Gram-negative pathogen commonly associated with healthcare-associated infections; peritonitis due to this organism in infants is exceptionally rare, particularly when presenting with septic shock and acute kidney injury (AKI) requiring peritoneal dialysis. We report the case of a previously healthy two-month-old male infant who presented with progressive respiratory distress, oliguria, generalized edema, and septic shock, necessitating mechanical ventilation and intensive care support. Laboratory investigations revealed severe AKI with metabolic derangements, and renal ultrasound demonstrated bilateral increased parenchymal echogenicity with pelvic fullness and perinephric fat stranding. Continuous peritoneal dialysis was initiated after insertion of a double-cuffed Tenckhoff catheter. Peritoneal fluid culture subsequently grew carbapenem-resistant *A. baumannii* (CRAB) complex (carbapenemase producer) that was resistant to most conventional antibiotics while retaining susceptibility to minocycline and colistin. Antimicrobial therapy was escalated to ceftazidime-avibactam, tigecycline, and intraperitoneal antibiotics according to susceptibility results and infectious disease recommendations. Despite aggressive management including dialysis, broad-spectrum antimicrobials, blood transfusion, and mechanical ventilation, the patient's condition deteriorated with persistent septic shock and multiorgan dysfunction; owing to financial constraints and a guarded prognosis, the family opted for discharge against medical advice. This case emphasizes that *A. baumannii* peritonitis is an extremely uncommon but devastating cause of severe sepsis and AKI in infancy and highlights the critical importance of prompt microbiological diagnosis, antimicrobial stewardship, early renal replacement therapy, and heightened awareness of multidrug-resistant pathogens in critically ill infants.

Categories: General Surgery, Internal Medicine, Gastroenterology
Keywords: acinetobacter baumannii, acute kidney injury, carbapenem-resistant, infant, multidrug-resistant infection, peritoneal dialysis, peritonitis, septic shock

Introduction

Acute kidney injury (AKI) in infancy remains a major contributor to morbidity and mortality in pediatric intensive-care units. Etiologies range from congenital anomalies and hypovolemia to sepsis-induced endothelial injury, cytokine-mediated inflammation (the release of signaling molecules that amplify the host inflammatory response), and microvascular thrombosis. Septic AKI is especially concerning because hypotension and these inflammatory cascades can rapidly progress to multiorgan dysfunction [1-3].

Acinetobacter baumannii (*A. baumannii*) has emerged globally as a leading nosocomial pathogen capable of surviving for prolonged periods on environmental surfaces and rapidly acquiring resistance determinants, including carbapenemases (enzymes that hydrolyze carbapenem antibiotics, rendering them ineffective). While it commonly causes ventilator-associated pneumonia, bloodstream infection, and catheter-related infections in critically ill patients, peritoneal infection is far less frequent and has been reported predominantly in adults receiving chronic peritoneal dialysis [4,5]. Spontaneous or secondary *A. baumannii* peritonitis in previously healthy infants is exceedingly rare [6,7].

Therapeutic options are severely limited once carbapenem resistance develops. Isolates frequently retain susceptibility only to polymyxins, tigecycline, minocycline, or newer β-lactam/β-lactamase-inhibitor combinations. Delay in appropriate therapy is strongly associated with adverse outcomes [8,9]. We describe a rare case of severe septic shock and dialysis-requiring AKI caused by CRAB peritonitis in a two-month-old infant and discuss diagnostic and management challenges in a resource-constrained environment.

Case Presentation

A two-month-old male infant (birth weight 2500 g), born at term and reportedly healthy thereafter, was brought to the emergency department of a tertiary-care hospital in Lucknow with progressive respiratory distress, poor feeding, oliguria, and generalized swelling. According to the mother, the infant had remained well until two days before admission, when difficulty in breathing appeared. Respiratory distress worsened over the ensuing 48 hours and was accompanied by a marked reduction in urine output (from 4-5 voids/day to 1-2 voids/day) and progressive facial, periorbital, and lower-limb edema. The child had been managed at multiple peripheral facilities with symptomatic measures; endotracheal intubation was performed before transfer because of worsening respiratory failure.

On arrival, the infant weighed 3.5 kg and measured 52 cm. He was mechanically ventilated and critically ill. Vital signs showed tachycardia (heart rate: 134 beats/min), tachypnea (respiratory rate: 62 breaths/min prior to ventilation), temperature of 98°F, and oxygen saturation of 98% once stabilized on the ventilator. Peripheral edema was prominent. Cardiovascular examination revealed normal heart sounds without murmurs; respiratory examination showed bilateral air entry without added sounds; the abdomen was soft and nondistended without organomegaly. The last urine had been passed several hours earlier, and the last stool earlier the same day. Vaccination status was age-appropriate.

Laboratory investigations demonstrated severe AKI with metabolic derangements, hyperglycemia, anemia, and systemic inflammation. Marked abnormalities included a serum creatinine of 2.4 mg/dL (reference: 0.2-0.4), arterial pH of 7.18, serum bicarbonate of 12 mEq/L, potassium of 6.1 mEq/L, and C-reactive protein of 48 mg/L. The complete set of laboratory findings is summarized in Table *1*.

Parameters	Patient values	Reference range
Hemoglobin (g/dL)	**8.2**	**9.5-13.5**
Total leukocyte count (/µL)	18 400	6 000-17 500
Platelet count (/µL)	156 000	150 000-400 000
C-reactive protein (mg/L)	48	<10
Blood urea (mg/dL)	86	5-18
Serum creatinine (mg/dL)	2.4	0.2-0.4
Serum sodium (mEq/L)	128	135-145
Serum potassium (mEq/L)	6.1	3.5-5.5
Serum chloride (mEq/L)	98	98-107
Serum bicarbonate (mEq/L)	12	20-28
Arterial pH	7.18	7.35-7.45
Serum calcium (mg/dL)	7.1	8.8-10.8
Serum phosphate (mg/dL)	7.8	4.8-8.2
Random blood glucose (mg/dL)	286	70-140
Serum albumin (g/dL)	2.6	3.5-5.0
Total bilirubin (mg/dL)	0.8	0.2-1.0
AST (U/L)	54	15-60
ALT (U/L)	38	13-45
International normalized ratio	1.3	0.9-1.2

TABLE 1: Laboratory investigations at presentation

Renal ultrasound demonstrated bilateral increased parenchymal echogenicity, fullness of both renal pelves, and bilateral perinephric fat stranding, consistent with acute renal parenchymal injury. These sonographic findings are shown in Figure *1*.

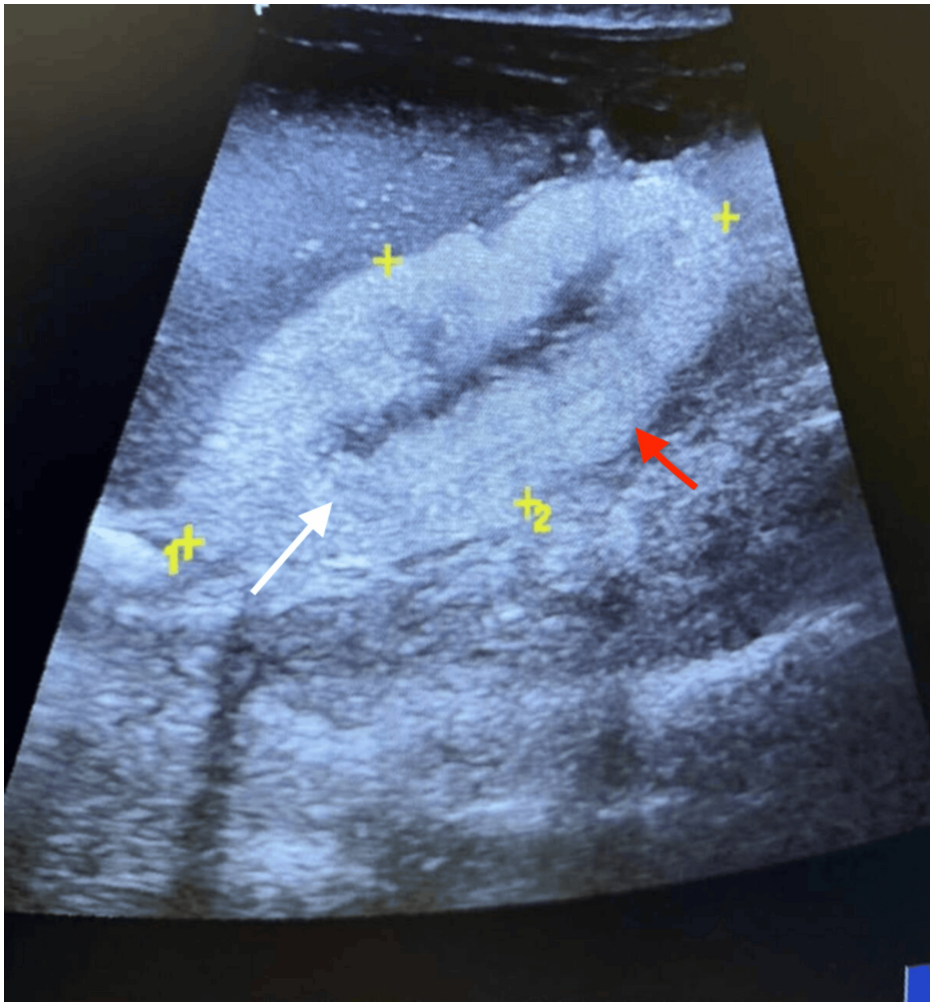


FIGURE 1: Renal ultrasound of the infant demonstrating features of acute renal parenchymal injury, including increased cortical echogenicity of the renal parenchyma (red arrow), perinephric fat stranding (white arrow), and measurements of renal parenchymal thickness and pelvic dimensions using yellow calipers (+1 and +2), revealing mild fullness of the renal pelvis

The infant was admitted to the pediatric intensive-care unit (PICU). Aggressive resuscitation included mechanical ventilation, intravenous fluids, vasoactive support, correction of electrolyte abnormalities, insulin infusion for hyperglycemia, calcium supplementation, sodium bicarbonate, broad-spectrum antimicrobials (meropenem and teicoplanin), analgesia, and nasogastric nutritional support.

Because of persistent oliguria and progressive renal failure, continuous peritoneal dialysis was chosen as the renal-replacement modality. On hospital day 2, a double-cuffed Tenckhoff catheter (37 cm) was inserted under sterile conditions and local anesthesia into the peritoneal and subcutaneous planes. Continuous PD was initiated with warmed dialysate, an initial dwell time of one hour, and careful monitoring of fluid balance, hemodynamics, and residual urine output. The infant initially tolerated dialysis; however, after the second cycle (approximately 24-36 hours after catheter insertion), the peritoneal effluent became cloudy. Cell count of the effluent showed >1000 white blood cells/μL with neutrophil predominance, and Gram stain demonstrated Gram-negative bacilli. Culture of the fluid subsequently grew drug-resistant *A. baumannii* complex extensively. No clinical or laboratory features of peritonitis were present at the time of catheter placement. Temporary interruption of PD and optimization of ventilatory parameters (SIMV mode, FiO_2 100%, PEEP 8 cm H_2O, rate 48/min) were required because of intensified respiratory distress.

Microbiological examination of peritoneal dialysis effluent demonstrated heavy growth of *A. baumannii* complex. Automated susceptibility testing identified an extensively drug-resistant (XDR), carbapenemase-producing isolate. The minimum inhibitory concentrations (MICs) and interpretive categories are presented in Table *2*.

Antimicrobial agent	MIC (µg/mL)	Interpretation
Piperacillin/tazobactam	**≥128**	**Resistant (R)**
Ceftazidime	≥64	Resistant (R)
Cefoperazone/sulbactam	≥32	Resistant (R)
Imipenem	≥16	Resistant (R)
Meropenem	≥16	Resistant (R)
Amikacin	≥64	Resistant (R)
Gentamicin	≥16	Resistant (R)
Ciprofloxacin	≥4	Resistant (R)
Levofloxacin	4	Intermediate (I)
Minocycline	1	Susceptible (S)
Colistin	≤0.5	Susceptible (S)
Trimethoprim/Sulfamethoxazole	≥320	Resistant (R)

TABLE 2: Antimicrobial susceptibility of Acinetobacter baumannii complex isolated from peritoneal fluid

As shown in Table 2, the isolate was resistant to piperacillin-tazobactam, ceftazidime, cefoperazone-sulbactam, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin, and trimethoprim-sulfamethoxazole. It retained susceptibility to minocycline (MIC: 1 µg/mL) and colistin (MIC: ≤0.5 µg/mL) and intermediate susceptibility to levofloxacin. Therapy was promptly escalated according to infectious-disease recommendations to include intravenous ceftazidime-avibactam and tigecycline, with continuation of selected intraperitoneal antibiotics once culture results were finalized. Although ceftazidime-avibactam is not listed in the susceptibility table, it was chosen on the basis of institutional protocol and local susceptibility patterns for carbapenemase-producing *Acinetobacter* in critically ill infants. Blood transfusion (160 mL packed red cells) was administered for anemia.

Despite these measures, the infant remained in refractory septic shock with multiorgan dysfunction. After detailed counseling regarding the guarded prognosis and the substantial ongoing financial burden, the parents elected to take the child home against medical advice (LAMA). Final diagnoses recorded were AKI, septic shock, right-sided pneumonia, respiratory failure requiring mechanical ventilation, and bacterial peritonitis due to *A. baumannii*.

Discussion

A. baumannii peritonitis is well-documented in adults on chronic peritoneal dialysis, where it accounts for a minority of Gram-negative episodes and is associated with higher rates of catheter loss and technique failure, especially when multidrug-resistant strains are involved [6,7]. In contrast, reports in infants and young children are scarce and usually occur in the context of prolonged hospitalization, invasive devices, or underlying immunodeficiency. The present case is notable because the infant was previously healthy, the community-acquired presentation progressed rapidly to septic shock and dialysis-requiring AKI, and the isolate was carbapenemase-producing [4,6].

The pathogenesis of septic AKI involves a combination of hemodynamic insults, endothelial activation, tubular epithelial injury, and inflammatory mediator release [1,2]. In this infant, the combination of systemic sepsis and direct peritoneal infection likely amplified the renal insult. Peritoneal dialysis remains the preferred continuous renal-replacement modality in small infants in many low- and middle-income settings because of technical simplicity, hemodynamic stability, and lower cost compared with extracorporeal circuits. Insertion of a soft Tenckhoff catheter under sterile conditions is recommended by current pediatric guidelines [10].

Management of CRAB peritonitis is challenging. The 2024 International Society for Peritoneal Dialysis (ISPD) pediatric guidelines suggest combination therapy for CRAB, ideally including high-dose ampicillin-sulbactam (even when in vitro resistance is present) plus a second active agent such as minocycline, tigecycline, a polymyxin, or cefiderocol, with consultation from an infectious-diseases specialist. Duration is

generally three weeks [11,12]. In the present case, ceftazidime-avibactam and tigecycline were selected on the basis of local availability and susceptibility data; colistin remained an option given documented susceptibility [8,9].

Despite appropriate escalation, clinical deterioration continued. Contributing factors likely included delayed presentation after multifacility transfers, the intrinsic virulence and resistance of the isolate, and limited supportive resources. The decision for LAMA reflects a common and tragic reality in many resource-limited settings where prolonged intensive care imposes unsustainable financial hardship on families.

This case highlights several practical lessons. First, unexplained oliguria and edema in a septic infant should prompt early consideration of AKI and timely initiation of renal-replacement therapy [3,10]. Second, peritoneal fluid must be cultured promptly whenever PD is commenced in a critically ill child; rapid identification of MDR organisms allows timely antimicrobial adjustment [11,12]. Third, infection-prevention measures-strict asepsis during catheter insertion and handling-are essential to reduce nosocomial acquisition of Acinetobacter [4,5]. Finally, antimicrobial stewardship programs and ready access to newer agents or combination regimens are urgently needed in regions with high prevalence of carbapenem-resistant Gram-negative pathogens [8,9].

Limitations of this report include the absence of long-term follow-up after LAMA and incomplete documentation of serial laboratory trends. Nevertheless, the detailed clinical, imaging, and microbiological data provide a clear illustration of the diagnostic and therapeutic difficulties posed by CRAB peritonitis in infancy.

Conclusions

CRAB isolated from peritoneal fluid represents a rare and potentially life-threatening infection in infancy, particularly in the setting of septic shock and severe AKI requiring renal replacement therapy. This case highlights the clinical challenges associated with multidrug-resistant Gram-negative infections and the potential for rapid progression to multiorgan dysfunction in critically ill infants. Although the microbiological findings establish the presence of resistant *A. baumannii* in the peritoneal fluid, its precise contribution to the initial onset of septic shock and AKI remains uncertain. The case underscores the importance of early microbiological investigation, timely culture-directed antimicrobial therapy, appropriate organ support, and strengthened infection-control and antimicrobial stewardship practices. Improved access to critical care and effective therapies is particularly important in resource-limited settings.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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References

1. Lameire NH, Bagga A, Cruz D, et al.: Acute kidney injury: an increasing global concern . Lancet. 2013, 382:170-9. 10.1016/S0140-6736(13)60647-9
2. Sutherland SM, Ji J, Sheikhi FH, Widen E, Tian L, Alexander SR, Ling XB: AKI in hospitalized children: epidemiology and clinical associations in a national cohort. Clin J Am Soc Nephrol. 2013, 8:1661-9. 10.2215/CJN.00270113
3. Basu RK, Zappitelli M, Brunner L, et al.: Derivation and validation of the renal angina index to improve the prediction of acute kidney injury in critically ill children. Kidney Int. 2014, 85:659-67. 10.1038/ki.2013.349
4. Peleg AY, Seifert H, Paterson DL: Acinetobacter baumannii: emergence of a successful pathogen . Clin Microbiol Rev. 2008, 21:538-82. 10.1128/CMR.00058-07
5. Wong D, Nielsen TB, Bonomo RA, Pantapalangkoor P, Luna B, Spellberg B: Clinical and pathophysiological overview of Acinetobacter infections: a century of challenges. Clin Microbiol Rev. 2017, 30:409-47. 10.1128/CMR.00058-16
6. Zhang W, Wu YG, Qi XM, Dai H, Lu W, Zhao M: Peritoneal dialysis-related peritonitis with Acinetobacter baumannii: a review of seven cases. Perit Dial Int. 2014, 34:317-21. 10.3747/pdi.2012.00198
7. Chao CT, Lee SY, Yang WS, et al.: Acinetobacter peritoneal dialysis peritonitis: a changing landscape over time. PLoS One. 2014, 9:e110315. 10.1371/journal.pone.0110315
8. Paul M, Carrara E, Retamar P, et al.: European Society of Clinical Microbiology and Infectious Diseases (ESCMID) guidelines for the treatment of infections caused by multidrug-resistant Gram-negative bacilli (endorsed by European society of intensive care medicine). Clin Microbiol Infect. 2022, 28:521-47. 10.1016/j.cmi.2021.11.025
9. Tamma PD, Heil EL, Justo JA, Mathers AJ, Satlin MJ, Bonomo RA: Infectious Diseases Society of America 2024 guidance on the treatment of antimicrobial-resistant Gram-negative infections. Clin Infect Dis. 2024, ciae403. 10.1093/cid/ciae403
10. Nourse P, Cullis B, Finkelstein F, Numanoglu A, Warady B, Antwi S, McCulloch M: ISPD guidelines for peritoneal dialysis in acute kidney injury: 2020 Update (paediatrics). Perit Dial Int. 2021, 41:139-57.
11. Warady BA, Same R, Borzych-Duzalka D, et al.: Clinical practice guideline for the prevention and management of peritoneal dialysis associated infections in children: 2024 update. Perit Dial Int. 2024, 44:303-64. 10.1177/08968608241274096
12. Borzych-Dużałka D, Same R, Neu A, et al.: Best practice of peritoneal dialysis-associated Gram-negative peritonitis in children: insights from the International Pediatric Peritoneal Dialysis Network Registry. Kidney Int Rep. 2024, 9:1654-63. 10.1016/j.ekir.2024.03.031