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# Infantile Meningitis Due to Multidrug-Resistant Acinetobacter Baumannii: A Case Report Highlighting Treatment Challenges

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## Abstract

*Acinetobacter baumannii* (*A. baumannii*), a multidrug-resistant (MDR) bacterium that has become a significant healthcare-associated pathogen, can infrequently cause severe infections of newborns' central nervous systems. Management of these conditions becomes difficult due to poor clinical outcomes with increasing resistance of organisms to commonly used antibiotics. We discuss a case of a one-month-old male infant who presented with fever, respiratory distress, and an enlarging head circumference as a complication of bacterial meningitis. On examination, he was found to be lethargic and febrile and had a head circumference of 38 cm. A computed tomography (CT) brain scan showed gross hydrocephalus, after which he underwent a diagnostic and therapeutic ventricular tap. The culture grew MDR *A. baumannii*, and he was treated with intrathecal colistin administered at each ventricular tap for almost 1.5 months. This case report highlights the rarity of *A. baumannii* meningitis in infants and the therapeutic challenges posed by increasing multidrug resistance, particularly in resource-limited settings, with implications for infection control and antibiotic stewardship.

**Categories:** Neurosurgery, Infectious Disease
**Keywords:** acinetobacter baumannii, developing country, infant, lmic, meningitis, multiple drug resistance, pakistan, rare

## Introduction

Bacterial meningitis is a severe, life-threatening disease that is widespread in low- and middle-income nations and is mostly associated with *Listeria monocytogenes*, *Streptococcus agalactiae*, *Klebsiella pneumoniae*, and *Escherichia coli* in neonates and infants. Early diagnosis and prompt antibiotic therapy are necessary to prevent permanent neurological damage and death. However, antimicrobial resistance makes management increasingly difficult. *Acinetobacter baumannii*, a multidrug-resistant (MDR) opportunistic pathogen, emerges as a rare but major cause of infant meningitis with a high mortality rate of 15%-71% [1-3]. Nearly 50% of all healthcare-associated neonatal meningitis cases in Iran were caused by *Acinetobacter* species, showing their clinical importance in this region [2].

*A. baumannii* is a Gram-negative, non-fermentative, catalase-positive, and oxidase-negative bacterium. Its high antibiotic-resistance profile, including resistance to aminoglycosides, carbapenems, and colistin, has also limited treatment options [4]. Acinetobacter meningitis in infants is associated with long-term hospitalization and neurosurgery complications [5]. Carbapenems and cephalosporins appear to be ineffective, and in some cases, treatment with polymyxins or tigecycline may be required. Case series of post-neurosurgical meningitis have consistently reported poor outcomes, with most patients being diagnosed with *A. baumannii* infection. This makes its MDR ability a serious threat [6].

*Acinetobacter* is a well-recognized pathogen contributing to bacteremia and ventilator-associated pneumonia, both of which can result in prolonged hospital stays [7]. Although Acinetobacter meningitis is rare in infants, it is a severe condition that can lead to complications like hydrocephalus, which often requires treatment with external ventricular drains, neurosurgical interventions, and ventilatory support [8]. This case report presents the diagnostic and therapeutic challenges encountered in treating MDR Acinetobacter meningitis in a young infant, with implications for infection control and antibiotic stewardship.

## Case Presentation

A one-month-old boy presented to the emergency department with a two-week history of fever, respiratory distress, and increasing head circumference. The patient was born to a 32-year-old woman without any preterm or post-term complications. At one and five minutes after delivery, the infant had reassuring scores

for all components of the Appearance, Pulse, Grimace, Activity, and Respiration (APGAR) score, indicating a smooth transition to life outside the uterus [9]. He was initially taken to a private hospital with neonatal sepsis and was admitted to the neonatal intensive care unit (NICU), where he was diagnosed with pneumonia and meningitis. Appropriate treatment was started. Later, they noticed an increase in head circumference, and the patient was suspected to be developing hydrocephalus. He was discharged and referred to us. On presentation, the boy was lethargic and febrile and had a head circumference of 38 cm. The rest of the systemic examination showed normal findings.

The patient was subjected to an initial workup, including all baseline tests (complete blood count, renal function test, liver function test, and serum electrolytes), which showed a raised total leukocyte count (TLC), but the rest of the investigation was normal (Table *1*).

| Parameter | Unit | Patient value | Reference value |
|---|---|---|---|
| Hemoglobin | g/dL | 12.8 | 9.6–15.9 |
| Total WBC | $\times 10^9$/L | 20.5 ↑ | 5–19 |
| Neutrophils | $\times 10^9$/L | 13.0 ↑ | 1.5–5.4 |
| Lymphocytes | $\times 10^9$/L | 6.0 | 2.8–9.1 |
| Monocytes | $\times 10^9$/L | 1.0 | 0.1–1.7 |
| Eosinophils | $\times 10^9$/L | 0.3 | 0.1–0.9 |
| Platelets | $\times 10^9$/L | 320 | 200–500 |
| Total bilirubin | mg/dL | 2.0 | 0.2–5.0 |
| Direct bilirubin | mg/dL | 0.4 | <1.0 |
| ALT | U/L | 28 | 10–50 |
| AST | U/L | 45 | 20–70 |
| ALP | U/L | 300 | 150–500 |
| Albumin | g/dL | 3.7 | 2.8–4.5 |
| Blood urea | mg/dL | 22 | 10–40 |
| Serum creatinine | mg/dL | 0.4 | 0.2–0.5 |
| Sodium ($Na^+$) | mmol/L | 138 | 135–145 |
| Potassium ($K^+$) | mmol/L | 4.7 | 3.5–5.5 |
| Chloride ($Cl^-$) | mmol/L | 103 | 98–108 |

**TABLE 1: Hematological and biochemical profile of the patient at presentation**

$Na^+$: Sodium; $K^+$: Potassium; $Cl^-$: Chloride; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; ALP: Alkaline phosphatase; WBC: White blood cells; ↑: Increased.

A computed tomography (CT) brain scan was performed, which showed gross hydrocephalus. The scan showed gross ventriculomegaly with transependymal hypodensities suggestive of transependymal seepage. He then underwent a diagnostic and therapeutic ventricular tap; 30 mL of cerebrospinal fluid (CSF) was drained and sent for routine investigation as well as culture and antimicrobial susceptibility testing. Empirically, intravenous ceftriaxone at 100 mg/kg/day was administered in two divided doses. The culture came out to be infected with *A. baumannii* (Figure *1*).

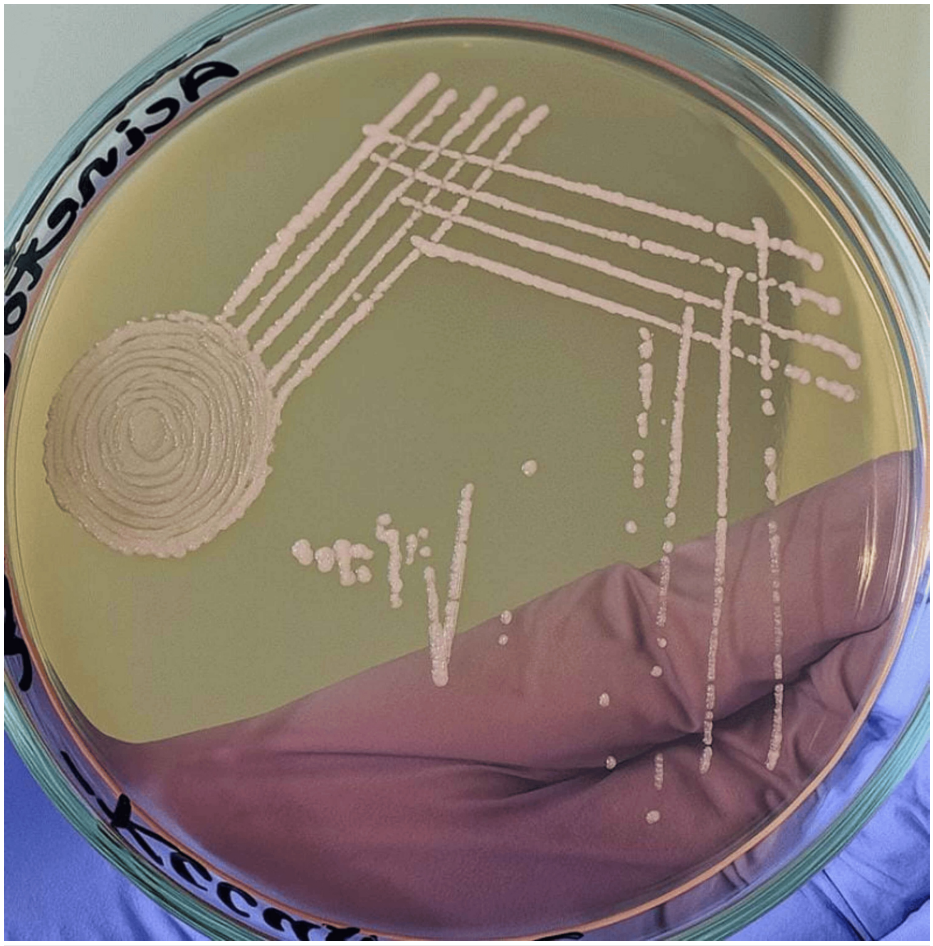


**FIGURE 1: Colonies of Acinetobacter baumannii on MacConkey agar**

Characteristics of *A. baumannii* on MacConkey agar and biochemical tests performed for *A. baumannii* are given in Tables 2, 3, respectively.

| Variables | Characteristic features |
|---|---|
| Size | Medium to large colonies |
| Shape | Circular and smooth |
| Surface | Convex and opaque |
| Texture | Slightly sticky |
| Color | Non-lactose fermenter (colorless) |
| Odor | Typically odorless |
| Consistency | Colonies appear mucoid in strains with a capsule |
| Growth | Good growth after 24 hours at 37°C |

**TABLE 2: Characteristics of Acinetobacter baumannii on MacConkey agar**

| Test | Reaction |
|---|---|
| Gram stain | Gram-negative coccobacilli |
| Oxidase | Negative |
| Catalase | Positive |
| Motility | Non-motile |
| Oxidation-fermentation (OF) test - (glucose) | Oxidative |
| Nitrate reduction | Variable (usually negative) |
| Urease | Usually negative |
| Citrate utilization | Positive |
| Indole | Negative |
| Triple sugar iron (TSI) test | Alkaline/alkaline (no fermentation) |

**TABLE 3: Biochemical tests performed to confirm Acinetobacter baumannii**

The isolate was resistant to multiple antibiotics, including cephalosporins, aminoglycosides, and carbapenems, and was only intermediately sensitive to colistin (Table 4). Based on culture and sensitivity, ceftriaxone was discontinued, and the patient was started on IV colistin 5 mg/kg/day for one day and then 2.5 mg/kg/day afterward for two weeks. His CSF samples were not clear, so intrathecal colistin was initiated at 10 mg/day. Intrathecal colistin was administered every time CSF was drained through a ventricular tap. This whole treatment was continued for almost 1.5 months, and he tolerated it well, without any renal complications.

| Antibiotics for sensitivity | | Result |
|---|---|---|
| Cephalosporins | Cefepime | Resistant |
| | Ceftazidime | Resistant |
| Carbapenems | Meropenem | Resistant |
| Aminoglycosides | Amikacin | Resistant |
| | Gentamicin | Resistant |
| Folate pathway inhibitors | Trimethoprim-sulfamethoxazole | Resistant |
| Miscellaneous | Colistin | Intermediate (MIC < 1.0 µg/mL) |

**TABLE 4: Antibiotic resistance shown by Acinetobacter baumannii on sensitivity testing**

MIC: Minimum inhibitory concentration.

His CSF became clear after treatment. Three clear CSF samples were collected, and ventriculoperitoneal (VP) shunting was deemed necessary because of continuous CSF buildup in the ventricles. A VP shunt (HP Bio, counter-flow medium pressure) was placed under general anesthesia at three months of age. The procedure was completed without intraoperative or postoperative complications.

Postoperatively, the patient remained stable, and the surgical incision was healthy. All baseline investigations were repeated and yielded normal results. He was discharged after one day. At the two-week follow-up, he was doing well, and the shunt chamber had adequate refills. He continued to attend follow-up visits, with the last visit occurring one year after surgery. At that visit, he was growing normally, with no complications related to the VP shunt.

## Discussion

In this case report, we discuss a one-month-old male infant with an uncommon case of *A. baumannii* meningitis. *A. baumannii*, an opportunistic Gram-negative coccobacillus, has become one of the most challenging pathogens in healthcare settings. In a seven-year review conducted in the United States, *A. baumannii* was found to account for 1.3% of all monomicrobial bloodstream infections, most of which were ICU-acquired. In the central nervous system, it causes meningitis, which is becoming exceedingly common among post-neurosurgical cases [2,3]. Meningitis cases were observed more frequently in males, with a male-to-female ratio of 2:1. The average patient age was 52.70 ± 48.14 months compared with 24 months in a review published in 2024 [1,4]. Factors such as invasive procedures, a history of previous neurosurgery, central venous catheters, ventricular drainage, bloodstream infections, community- or ventilator-acquired pneumonia, prior antibiotic use, and immunosuppressive therapy have been associated with these cases [4]. In the case presented above, the infant initially presented with signs and symptoms of pneumonia and neonatal sepsis and subsequently developed hydrocephalus as a complication of meningitis.

Multidrug resistance and extensive drug resistance (XDR) are a huge concern in the current era. More than 40% of pediatric isolates exhibit MDR/XDR profiles, limiting therapeutic contributions in China and other Asian countries [4]. Studies have identified OmpA as the major determinant in *A. baumannii* virulence, promoting biofilm formation, complement resistance, and epithelial adhesion. Multidrug resistance primarily stems from overexpression of chromosomal AmpC cephalosporinases (ADCs) and OXA-51-like oxacillinases. These enzymes confer resistance to extended-spectrum cephalosporins, penicillins, and, to a lesser extent, carbapenems by hydrolyzing them [10]. A case series confirmed that most isolates were pan-resistant and that mortality remained high despite intravenous and intrathecal polymyxin therapy [2].

Cephalosporins and carbapenems are ineffective against MDR polymyxin intravenously or intraventricularly. Administration of intrathecal polymyxin B or colistin has reported clinical success in various case reports. However, relapses and delayed CSF sterilization have been reported [1,11]. Sulbactam, tigecycline, and fosfomycin combination therapies have been undertaken with limited benefits [2,4]. Limited access to newer antimicrobial compounds is a major problem, highlighting the importance of tailored therapy and multidisciplinary management. Our case further emphasizes the problem of multidrug resistance by exhibiting resistance to major classes of antibiotics and justifying the rationale to switch from ceftriaxone to colistin after no clinical improvement was observed.

Acinetobacter meningitis carries a high risk of mortality, prolonged hospitalization, and, rarely, neurological sequelae. MDR *A. baumannii* is an evolving neonatal pathogen, which is evident by regional data, stressing the urgent need for strict infection control practices, rational antibiotic use, and early recognition of resistive patterns. Global awareness regarding *A. baumannii* is needed to improve patient management and treatment strategies [2,3]. The use of colistin has been studied variably, but good patient satisfaction was observed, as in this one-year-old infant.

Across reported series, clinical outcomes remained guarded. The literature showed two of eight children died, including one with pan-resistant isolates, despite intensive use of colistin [5]. In other case series, mortality ranged from 30% to 50%, with survivors often experiencing long-term neurological sequelae [5,12]. In an Iranian review of neonatal meningitis, nearly 40% of cases with *Acinetobacter* infection resulted in death, highlighting the vulnerability in resource-limited settings [2]. Even in centers with access to intraventricular therapy, outcomes often depended on timely diagnosis, appropriate dosing, and supportive care, with delays correlating directly with higher mortality [6,7]. In contrast to other studies, our case developed hydrocephalus as a complication of *A. baumannii* meningitis and was managed with a VP shunt following a successful 1.5-month course of intrathecal colistin. This sequela can be used to highlight the harmful impact of delayed diagnosis caused by unstandardized practices and limited resources.

Recurring challenges have been reinforced by our case: multidrug resistance, limited therapeutic options, and the need for extensive but toxic therapies. It also focuses on strictly preventing such infections, careful stewardship of existing antibiotics, and the requirement of multicenter studies to develop standardized treatment protocols. Future strategies should emphasize refining intraventricular dosing, investigating novel combination regimens, and enhancing infection-control measures in intensive care and neurosurgical units. The authors acknowledge that CT images of the brain were unavailable and molecular typing to identify resistance genes was not performed because of limited resources.

## Conclusions

Our case highlights the significant threat posed by MDR *A. baumannii* in infants, with limited therapeutic options and high mortality. Intrathecal colistin may be effective when intravenous therapy fails, emphasizing the importance of tailored treatment. The development of complications such as hydrocephalus emphasizes the need for early recognition and multidisciplinary management. Strengthening infection control, promoting rational antibiotic use, and conducting ongoing research into optimized therapies remain crucial to improving outcomes.

## 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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