

Case Report

A rare case of community acquired meningitis caused by multidrug resistant *Acinetobacter baumannii* in Bengaluru India

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ABSTRACT

Acinetobacter baumannii can be a potentially life-threatening cause of severe community-acquired meningitis outside healthcare facilities and as a nosocomial infection following neurosurgical procedures in intensive care units (ICUs). Despite the availability of potent newer antibiotics, mortality and complications remain high in India, ranging from 16% to 32%. Community-acquired meningitis caused by *A. baumannii* is relatively rare, and proper antibiotic usage and adherence to infection control protocols in hospitals should be prioritized. In this case study, we report a case of community-acquired meningitis caused by *A. baumannii* in Bangalore without any prior history of neurosurgical complications or trauma.

Keywords: *Acinetobacter baumannii*, Community acquired meningitis, Hospital acquired infection, Antibiotics

INTRODUCTION

Acinetobacter baumannii, a gram-negative, non-flagellar bacterium, is typically short, nearly round, and rod-shaped (coccobacillus).¹ Named after the bacteriologist Paul Baumann, it can be an opportunistic pathogen in humans, particularly affecting individuals with compromised immune systems. As a hospital-derived (nosocomial) infection, its significance is growing. While other *Acinetobacter* species are commonly found in soil samples, leading to the misconception that *A. baumannii* is a soil organism, it is almost exclusively isolated from hospital environments.² *A. baumannii* has also been identified as an ESKAPE (*Enterococcus faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *A. baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter* species) pathogen, a group of pathogens with a high rate of antibiotic resistance responsible for the majority of nosocomial infections.³ During the COVID-19 pandemic, coinfection with *A. baumannii* secondary to SARS-CoV-2 infections has been reported in multiple studies.⁴ *A.*

baumannii is a significant contributor to nosocomial infections, particularly due to the increasing prevalence of multi-drug and pan-resistant pathogens.^{5,6} Its presence in skin and soft tissue infections has been reported in traumatic injuries and surgical wounds. Complications arising from *A. baumannii* infections can hinder treatment, especially when burns become infected with this organism.⁷ In some cases, systemic infection can develop when the bacteria enter the bloodstream, leading to septicemia. Prolonged use of catheters and antibiotic therapy has also been associated with *A. baumannii* infections. *A. baumannii* possesses a remarkable array of resistance mechanisms, resulting in its ability to resist all or almost all commercially available antibiotics. This organism specifically targets moist tissues, such as the mucus membrane of exposed skin areas, either due to accidents or injuries. Initially, skin and soft tissue infected with *A. baumannii* may present with a “peau d’Orange” appearance, resembling the skin of an orange.⁸ Subsequently, a sandpaper-like texture develops, eventually leading to the formation of clear vesicles on the skin. *Acinetobacter* meningitis typically occurs

following neurosurgery. Patients who experience cerebrospinal fluid leakage, concomitant incision infections, prolonged surgical duration, surgical procedures that involve sinuses, prolonged external ventricular drain, and the need for repeat surgery are at heightened risk of developing post-neurosurgical meningitis.⁹ The attributable mortality rates in patients with *A. baumannii* healthcare-associated infections, particularly ventilator-associated pneumonia and bloodstream infections, exhibit substantial variability. In general hospital wards, the mortality rate typically ranges from 5% to 10%, whereas in the intensive care unit (ICU), it can attain up to 54%.¹⁰ Notably, the occurrence of community-acquired *Acinetobacter* meningitis of the central nervous system (CNS) is exceptionally uncommon.¹¹ In the present study, the prevalence of *Acinetobacter* spp. was observed to be 2.50%.¹² Furthermore, literature suggests that *Acinetobacter* species constitute a mere 0.2% of community-acquired meningitis cases in adults.¹³

CASE REPORT

A 23-year-old male was admitted to the intensive care unit (ICU) on August 3, 2025, with the primary complaints of fever over 20 days prior, severe headache, neck pain, difficulty to open right side of the eyes, and altered sensorium since the third day of having high grade fever. He had also been experiencing cough and expectoration for the past week, accompanied by nausea and vomiting. Initially, he was provisionally diagnosed with tuberculosis meningitis with vasculitis. However, subsequent Et culture and sensitivity (polymerase chain reaction) and MRI reports revealed his final diagnosis as *Acinetobacter* meningitis.

Clinical signs

Upon examination, the patient's body temperature was 102 degrees Fahrenheit, pulse rate was 72 beats per minute, blood pressure was 130/90 millimeters of mercury, and respiration rate was 24 breaths per minute. SpO₂ was 93%. Systemic examination revealed a positive chest X-ray (S1 S2) and a positive respiratory system chest X-ray (B/L NUBS). The pulmonary artery was soft and nontender. CNS examination revealed an E4VTM3 pattern with anisocoria (unequal pupil sizes). The right eye exhibited lateral rectus palsy (LR PALSY) with a 2-millimeter dilation. The left eye had ptosis (the dropping of the upper eyelid, which can partially or completely obstruct vision) and generalized edema.

Investigation

Upon the patient's arrival, pertinent investigations were conducted. Hematology results indicate a hemoglobin concentration of 12.8%, a red blood cell count of 4.89 million/cumm, a hematocrit of 41.8%, a platelet count of 1.42 lacks/cumm, and a total white blood cell count of 21390 cells/cum. Neutrophils comprise 88%,

lymphocytes are 0.2%, monocytes are 9%, eosinophils are 1%, and basophils are 0%. Liver function tests reveal a C-reactive protein level of 102.99 mg/l. Renal function tests indicate a serum sodium concentration of 125 mmol/l, a serum potassium concentration of 5.4 mmol/l, a serum chloride concentration of 91 mmol/l, and a serum urea concentration of 1.9 mg/dl. ABG analysis indicates a FiO₂ level of 40%, a pH level of 7.4, a PO₂ level of 26 mmHg, a PCO₂ level of 40 mmHg, and an HCO₃ level of 18.3 mEq/l. Blood culture analysis indicates the presence of *Acinetobacter baumannii*. MRI analysis reveals sub centimeter diffusion restrictions in the left posterior corona radiata, mild dilatation of both ventricles, and mild thickening of the medial aspect of the right tentorium approximately 2 mm. A hypoplastic right transverse sinus is also noted (Figure 1). In fluid cytology, the cerebrospinal fluid (CSF) cell count is 48 cells/cumm, the fluid's color is clear, and its volume is 1 ml. The cell count consists predominantly of lymphocytes, with a few neutrophils. A clear percutaneous background is observed on the smears. Based on the FRAT scale, the patient's fall risk assessment score is 9.

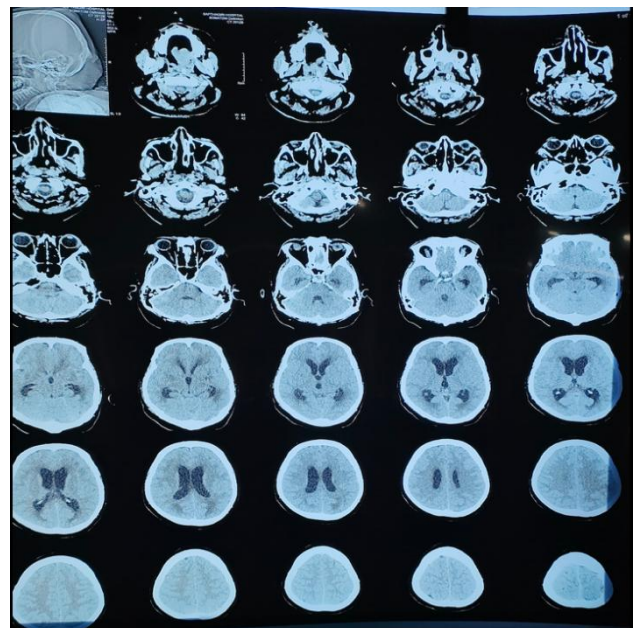


Figure 1: Presents with sub-centimeter diffusion restriction in the left posterior corona radiata with mild dilatation of bilateral lateral, 3rd, and 4th ventricles.

Therapeutic intervention

After the confirmation of the diagnosis by diagnostic tests and radiological investigations, the patient was prescribed NS @100 ml/hour, Tab AKT 4 × OD, Inj Dexa × TDS (16 mg-16 mg-8 mg), inj meropenem 1 gm IV × BD, inj levipil 1 gm IV × BD, inj 3% NS@10 ml/hour, inj pan 40 mg IV × OD (before food), syp glycerol 30 ml × TDS, tab pyridoxine 40 mg × OD and inj clexane 40 mg S/C × HS. Furthermore, it was also advised to monitor intake and

output chart daily, use of deep vein thrombosis (DVT) stocking, back care, endo tracheal (ET) care, and physiotherapy.

CULTURE			
ET CULTURE & SENSITIVITY			
Method: Aerobic method		Nature Of Sample: ET	
Organism Isolated: <i>Acinetobacter baumannii</i> .			
Antibiotic	Sensitivity	Antibiotic	Sensitivity
AMIKACIN	R	IMIPENEM	R
COTRIMOXAZOLE	R	MEROPENEM	R
GENTAMICIN	R	TIGECYCLINE	S
CEFOPERAZONE/SULBACTAM	R	CEFEPIME	R
CIPROFLOXACIN	R	PIPERACILLIN/TAZOBACTAM	R
		CEFTRIAXONE	R
Note:			
Antibiogram used are as per CLSI international guideline for the isolate.			
S : Sensitive	R: Resistant	I : Intermediate i.e. sensitive at higher doses only	RA: Restricted Antibiotics (Contact microbiology lab for assistance)
For query contact Microbiology Laboratory Hospital extension : Ph : 3800			
*** End of Report ***			

Figure 2: ET culture and sensitivity report.

DISCUSSION

A. baumannii has emerged as an opportunistic pathogen responsible for a wide range of severe nosocomial infections. A significant portion of *A. baumannii*'s success can be directly attributed to its genome plasticity, which exhibits rapid mutation under stress. This ability to resist most last-line antimicrobial agents poses a substantial challenge, particularly in critically ill patients.¹⁴ *A. baumannii* is a frequent cause of hospital-acquired pneumonia, primarily affecting the lungs. Resistance rates of this pathogen to cefoperazone/sulbactam and minocycline were 49.7% and 38.8%, respectively. However, its resistance rates to polymyxin B and tigecycline were relatively low (0.7% and 5.0%, respectively). Given that *A. baumannii* is relatively rare, the number of effective antibiotics against Multidrug-resistant *A. baumannii* (MDRAB) is limited. Consequently, polymyxins serve as the cornerstone of treatment. However, the use of polymyxins alone often fails to achieve satisfactory therapeutic outcomes.¹⁵⁻¹⁷ In our case, the patient was initially diagnosed with tuberculosis meningitis. However, subsequent PCR testing confirmed that the meningitis was caused by *A. baumannii*. In post-neurosurgical wards, the organism is highly prevalent among patients with ventricular shunts, making device removal a crucial measure to prevent infection. Intraventricular antibiotics play a significant role in treating *Acinetobacter* meningitis, as the minimum inhibitory concentration (MIC) of commonly used intravenously administered antibiotics for meningitis is typically high. Consequently, adequate cerebrospinal

fluid (CSF) concentrations may not be achieved when these antibiotics are administered intravenously. It is imperative that any agent administered intraventricularly be prepared in a preservative-free medium to avoid toxicity.^{18,19} *Acinetobacter* meningitis can occur during post-surgical procedures and prolonged hospital stays. However, in our case, the patient developed meningitis due to *A. baumannii* without any trauma or prior surgical history. To date, there have been only a few reported cases of *Acinetobacter* meningitis, most of which are nosocomial infections acquired in hospital intensive care unit (ICU) environments. These infections are highly resistant to most commercially available antibiotics, leaving limited treatment options. After being unconscious for a day and placed on ventilator support, the patient regained consciousness and responded to all neurophysiological parameters. Notably, there are very few reported cases of community-acquired *Acinetobacter* meningitis in India. This observed case of *A. baumannii* indicates that the threat has extended beyond hospitals in India, potentially developing into a significant community pathogen.²⁰ Therefore, prompt diagnosis is essential for early treatment initiation. Additionally, it is crucial for healthcare workers, hospital policy developers, and government stakeholders to implement measures to educate about the proper use of personal protective devices and adherence to hospital protocols for high-risk areas.

CONCLUSION

Community-acquired meningitis caused by *A. baumannii* can pose a severe life-threatening risk. The indiscriminate use of antibiotics should be strictly avoided to prevent the emergence of highly resistant strains of *A. baumannii*. Additionally, it is crucial to utilize narrow-spectrum antibiotics to specifically target these organisms. *A. baumannii* can significantly prolong the recovery of patients with meningitis. Consequently, it is imperative to adhere strictly to the hospital's infection control protocol.

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