Multidrug-resistant Acinetobacter meningitis in children

Ira Shah¹, Muznah Kapdi¹

¹Department of Pediatrics, B. J. Wadia Hospital for Children, Mumbai, Maharashtra, India

ABSTRACT

Acinetobacter species have emerged as one of the most troublesome pathogens for healthcare institutions globally. In more recent times, nosocomial infections involving the central nervous system, skin and soft tissue, and bone have emerged as highly problematic. Acinetobacter species infection is common in intensive care units; however, Acinetobacter baumannii meningitis is rarely reported. Here, we report two cases of Acinetobacter baumannii meningitis which was multidrug resistance and ultimately required the carbapenem group of drugs for the treatment.

Keywords: Coronary heart disease, functional foods, hypercholesterolemia, probiotics

Introduction

Acinetobacter spp are aerobic Gram-negative coccobacilli or rods. They are commonly found in environment and hospital. It is clearly pathogenic when recovered from blood and normally sterile body sites. Risk factors include hospitalization, Intensive Care Unit (ICU) stay, surgery, antibiotic exposure, and catheters.^[1] The organism commonly targets the most vulnerable hospitalized patients, those who are critically ill with breaches in skin integrity and airway protection.^[2] Nosocomial, postneurosurgical Acinetobacter baumannii meningitis is an increasingly important entity rarely reported in children.^[1-3] We present two children with Acinetobacter baumannii meningitis, where the first one had antibiotic exposure while the second one had many risk factors such as antibiotic exposure, prolonged ICU stay, and multiple procedures being performed.

Case Reports

Case 1

A 7-month-old male child presented with fever for 20 days and a generalized tonic convulsion in the morning. His sensorium

Address for correspondence: Dr. Ira Shah, 1/B, Saguna, 271/B, St. Francis Road, Vile Parle (W), Mumbai - 400 056, Maharashtra, India. E-mail: irashah@pediatriconcall.com

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was normal and was on cefpodoxime for the fever. There was no contact with the patient suffering from tuberculosis. His birth history and development was normal and was on breast feeds and cow's milk. On examination, his weight (5.4 kg) and height (64 cm) were below the fifth centile. He had pallor, bulging anterior fontanelle with hypertonia. Deep tendon reflexes were brisk, and planters were extensors. There was no focal neurological deficit. Other systems were normal. Investigations showed hemoglobin of 9.0 g/dl, white blood cell (WBC) count of 16,800 cells/cumm (77% polymorphs and 23% lymphocytes) with platelets of 506,000/cumm. His C-reactive protein (CRP) was 88 mg/dl. Cerebrospinal fluid (CSF) examination showed 101 mg/dl of proteins, 380 cells/cumm of which 15% were polymorphs and 85% were lymphocytes), 33 mg/dl of sugar with corresponding blood sugar of 110 mg/dl. He was started on ceftriaxone but did not show any improvement. After 4 days, repeat CSF showed 117 mg/dl of proteins, 330 cells/cumm (5% polymorphs, 95% lymphocytes), and sugar of 20 mg/dl. He was started on antituberculous therapy. A computed tomography of the brain showed moderate hydrocephalus with infarcts in the basal ganglia. His CSF culture grew Acinetobacter baumannii sensitive to imipenem, meropenem, and ciprofloxacin and resistant to cephalosporins. He was started on ciprofloxacin and meropenem. He subsequently took discharge against medical advice and was lost to follow-up.

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Case 2

A 4-month-old male child presented with fever for 2 days and multiple episodes of generalized tonic convulsion. There was no vomiting, altered sensorium in between convulsions, or ear discharge. He was born at full term without any postnatal complication, with birth weight of 3.1 kg. He was immunized till date and milestones were normal. He was on exclusive breast feeds. On examination, his heart rate was 136/min, and respiratory rate was 40/min. He had pallor and anterior fontanelle was at level. Head circumference was 40 cm, length was 57 cm, and weight was 5.7 kg. He was drowsy and had hypertonia with brisk deep tendon reflexes. There was no focal neurological deficit and other systems were normal. Investigations showed hemoglobin of 8.7 g/dl, WBC count of 14,200/cumm (48% polymorphs, 52% lymphocytes), platelets of 334,000/cumm. CRP was nonreactive. CSF showed proteins of 48 mg/dl, glucose of 73 mg/dl (corresponding blood sugar of 215 mg/dl), and 21 cells with 2 polymorphs and 19 lymphocytes. Renal functions were normal. Bilirubin was 0.8 mg/dl, serum glutamic oxaloacetic transaminase was 213 IU/L, and serum glutamic pyruvic transaminase was 167 IU/L. Prothrombin time (16 s) and partial thromboplastin time (51.3 s) were prolonged. Arterial blood gas showed metabolic acidosis (pH = 7.3, bicarbonate = 12.3) without hypoxia. HIV ELISA and CSF culture were negative. He subsequently developed shock and cardiorespiratory failure on day 3 of hospitalization and had to be intubated and put on positive pressure ventilation along with inotropic support. Blood culture grew Candida albicans, for which the child was started on amphotericin B which he received for 21 days. A repeat blood culture on day 6 of hospitalization grew methicillin-sensitive Staphylococcus aureus, for which he was started on linezolid which was given for 14 days. He improved and inotropes and ventilation were stopped on day 10 of hospitalization. On day 15, he again started having fever with drowsiness, for which lumbar puncture was done that showed 48 mg/dl proteins, 222 cells/cumm (65% polymorphs and 35% lymphocytes) with sugar of 57 mg/dl (corresponding blood sugar of 258 mg/dl). CSF culture grew multidrug-resistant (MDR) Acinetobacter baumannii sensitive only to meropenem, imipenem, and colistin. He was started on meropenem. His CRP now was 142 mg/dl, and ultrasound of the skull showed bilateral subdural empyema. His subdural empyema on the left side was drained on day 20 of hospitalization, and pus showed 1442 cells with 2 g% proteins and pus culture also grew Acinetobacter baumannii. He had partial response but again fever restarted. A repeat ultrasound of the skull on day 30 of hospitalization showed an increase in the right subdural empyema to 1.3 cm. He again underwent right-sided drainage of pus, following which he became afebrile. Meropenem was given for total 42 days, and CRP decreased to 8 mg/dl and ultrasound of the skull showed 5 mm collection.

Discussion

The treatment of *A. baumannii* is an issue of concern, specifically because of the increasing prevalence of multidrug resistance and pandrug resistance. Multidrug resistance is resistance to more than two of the following five drug classes: antipseudomonal cephalosporins (ceftazidime or cefepime),

antipseudomonal carbapenems (imipenem or meropenem), ampicillin-sulbactam, fluoroquinolones (ciprofloxacin or levofloxacin), and aminoglycosides (gentamicin, tobramycin, or amikacin), whereas pandrug resistance is defined as resistance to all antimicrobials that undergo first-line susceptibility testing that have therapeutic potential against *A. baumannii* which would include all lactams (including carbapenems and sulbactam), fluoroquinolones, and aminoglycosides. [4-6] Both our cases were MDR and sensitive to the carbapenems group of drugs. Numerous outbreaks of pandrug-resistant *A. baumannii* have been documented in Asian and Middle Eastern hospitals, and a variety of carbapenemases have been described to originate there. [4-6] In case with pandrug resistance, polymyxins and tigecycline come to the rescue.

Conclusion

Actinetobacter meningitis is a hazardous infection in hospital units. Actions aiming at reducing irrational antibiotic use and postprocedure or prolonged hospital stay infection must be evaluated routinely. Despite the majority of A. baumannii strains still being susceptible to carbapenems, many institutions globally are faced with the challenging issue of pandrug resistance. Given the current therapeutic environment, optimizing the use of existing antimicrobials is critical.

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Conflicts of interest

There are no conflicts of interest.

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