ACINETOBACTER JOHNSONII PERITONITIS IN A PATIENT ON PERITONEAL DIALYSIS: A CASE REPORT AND REVIEW OF THE LITERATURE

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ABSTRACT

Acinetobacter species have important role in modern medicine due to their increasing presence in health-care facilities and antibiotic resistance. They are non-fermentative, aerobic, Gram-negative coccobacilli and are important bacteria causing peritonitis in patients on peritoneal dialysis. *Acinetobacter johnsonii* is widely found in the aquatic environment and animals. Peritonitis caused by *A. johnsonii* is rarely encountered and reported. Here we report a case of *A. johnsonii* peritonitis in a patient on peritoneal dialysis. To the best of our knowledge, this is the first case of peritoneal dialysis-associated peritonitis by *A. johnsonii* reported from India.

KEYWORDS

Acinetobacter johnsonii, PDAP, peritonitis, peritoneal dialysis

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INTRODUCTION

Acinetobacter species are generally ubiquitous in nature and are frequently found in water, soil, and hospital environment; many of these species have been implicated in a variety of hospital-acquired infections.[1] They are Gram-negative, non-motile, non-fermentative coccobacilli belonging to family *Moraxellaceae.*[2] Currently, 22 species of *Acinetobacter* are there with valid names, and atleast 11 additional putative species have been recognized within genus, with as many as 25 of them have been found associated with human infections[3]. Important ones are *A. calcoaceticus-baumannii* complex, *A. lwoffii, A. junii, A. johnsonii* and *A. hemolyticus.*[2] They are non-fastidious and can utilize variety of substrates as their sole source of energy. *A. lwoffii* and *A. johnsonii* are predominantly found in foods such as chicken, eggs, fish, other meat and milk products.[1] According to some studies, *A. johnsonii* and *A. lwoffii* are also regarded as emerging opportunistic fish pathogens.[4] It indicates that fish may transmit these microbes to humans.

This is a case of peritonitis due to *A. johnsonii* in a patient on peritoneal dialysis. Continuous ambulatory peritoneal dialysis (CAPD) has been long purported over hemodialysis as a treatment modality for patients with end-stage renal disease because of its similar effects on patient outcomes and economical advantages.[5] Peritoneal dialysis-associated peritonitis (PDAP) is the most life-threatening condition. *Acinetobacter* species peritonitis is clinically more severe and is associated with prolonged hospitalization.[6]

CASE REPORT

A 6-year old boy admitted to the hospital with complaints of breathlessness, generalized swelling of body and altered level of consciousness. His medical history included congenital heart disease with chronic kidney disease and having recurrent infections since childhood. Child had complaints of dribbling of urine with poor urine stream, dull aching pain in left loin region. Echocardiogram showed dysplastic tricuspid valve with tricuspid stenosis, mild left ventricular dysfunction with ejection fraction 48.0%. On physical examination, heart rate was 160 per minute; blood pressure 118/70 mmHg; respiratory rate 28 per minute; temperature 98.4°F; SpO_2 99.0%; cold mottled skin, and chest wall was barrel shaped. His laboratory tests were as follows: hemoglobin: 9.2 g/dl; white blood cell count (WBC): 28,800/mm^3 (with 91% neutrophils); platelet: 60,000/mm^3; Urea: 311 mg/dl; creatinine: 6.7 mg/dl; GFR: 14 mL/min per 1.73 m^2; albumin: 2.7 g/dl; erythrocyte sedimentation rate: 102 mm/h; C-reactive protein (CRP): 170 mg/dl; Na^+: 135 mmol/l; K^+: 3.2 mmol/l; Ca^{++}: 1.05 mmol/l; Cl^-: 114 mmol/l, and ascitic fluid WBC: 1000 cells/mm^3 (with 90% neutrophils).

Child was intubated and central venous line was fixed. Then the patient was put on peritoneal dialysis (PD) under aseptic precautions with Stiff PD catheter. Intravenously, ceftriaxone 750 mg and vancomycin 1 gm once daily empiric treatment had been started because of septic condition of the patient. Blood, urine and peritoneal fluid specimens were sent for microbiological investigations. Blood and urine specimens were sterile for bacterial culture. On the fourth day of treatment, patient developed fever and abdominal tenderness. Gram stained smear of peritoneal fluid showed numerous polymorphonuclear cells and Gram-negative bacilli. Peritoneal fluid inoculated on 5% sheep blood agar, MacConkey agar, chocolate agar and incubated overnight aerobically at 37°C. Next day, on blood agar non-hemolytic, sparse growth appeared; no growth was observed on MacConkey agar but growth was enhanced on chocolate agar. The bacillus produced catalase and utilized citrate as the sole source of carbon. It did not produce oxidase, hydrolyze urea or reduce nitrate. It did not ferment glucose, sucrose or lactose and hydrogen sulphide was not formed in triple sugar iron test. This non-fermenter utilized sugars oxidatively and was non-motile in motility agar. On the basis of biochemical tests organism was identified as *Acinetobacter* spp. The same organism was isolated from a repeat sample on the next day.

Thereafter, species level identification and in-vitro antibiotic susceptibility tests were performed. The culture isolate was identified as *A. johnsonii* by Matrix-assisted laser desorption ionization/time-of-flight mass spectrometry (MALDI–TOF/MS) using bioMérieux VITEK MS system (IVD database version 2.0, USA). Antibiotic susceptibility testing was performed in Vitek 2 (bioMérieux SA, France) by using AST N281 card. According to the breakpoints recommended by CLSI standards, isolate was sensitive to amikacin, cefoperazone-sulbactam, imipenem, meropenem, ciprofloxacin, piperacillin-tazobactam and colistin but resistant to ceftazidime, tigecycline and cotrimoxazole.

DISCUSSION

Peritonitis is a most serious complication of CAPD, which results in 8-20% of infection related mortality.[7] Worldwide, 30-40% of peritonitis episodes are due to Gram-negative bacilli[7]. Among peritonitis due to Gram-negative bacilli, the acronym SPICE (*Serratia, Providencia, indole-positive Proteus/Morganella/Acinetobacter, Citrobacter* and *Enterobacter* spp.) denotes a spectrum of intrinsically antibiotic resistant organisms. These organisms have inducible beta-lactamase production during initial therapy, which potentially lead to empirical antibiotic treatment failure with prolonged peritoneal damage and worse outcomes. Of this SPICE group, *Acinetobacter* spp. is responsible for approximately 20.0% peritonitis episodes.[7] Among *Acinetobacter* spp., *A. baumannii* and *A. lwoffii* are the most commonly identified causative agent of PDAP.[7] In a study of *Acinetobacter* PDAP, the most common species were *A. baumannii* (54%) and *A. lwoffii* (35%), followed by *A. ursingii* (4%) and *A. junii* (4%).[7] Cases of *A. johnsonii* peritonitis are rarely encountered and reported. *A. johnsonii* species was identified to encode an extended spectrum beta-lactamase that conferred resistance against some of beta lactam antibiotics like penicillins, cephalosporins, and monobactams, has been scarcely reported to cause human or animal diseases.[8]

A. johnsonii is Gram-negative, strictly aerobic, non-motile, non-fastidious, non-fermenting coccobacilli. The optimum growth temperature for *A. johnsonii* is 25–30°C and no growth occurs at 37°C or above.[2] But growth on chocolate agar can occur at 37°C (reason not known). Conventionally it is difficult to identify till species level but with the help of automated bacterial identification system this becomes easy.

Acinetobacter spp. infection tends to occur in patients with chronic diseases as renal disease, diabetes mellitus and chronic obstructive pulmonary disease. Chronic glomerulonephritis and diabetes mellitus are the most common causes of renal disease in *Acinetobacter* peritonitis patients.[7] *Acinetobacter* peritonitis tends to occur in early phase of peritoneal dialysis and can be recurrent within first 1-2 months following another PD peritonitis episode.[9] There are several therapeutic options available for the treatment of antibiotic-sensitive *Acinetobacter* infections based on antibiotic susceptibility pattern. For the resistant isolates, therapeutic options are colistin, polymyxin and tigecycline.[10]

In this case, empirical antibiotic treatment had been changed to targeted therapy with cefoperazone-sulbactam 750 mg thrice daily intravenously based on antibiotic sensitivity pattern. According to our best knowledge from different literatures, there is no clue for the optimal antibiotic treatment period for *A. johnsonii* peritonitis. A clinical outcome depends on improvements in primary disease. Fever and abdominal tenderness had been improved in our case on continuation of targeted therapy.

In conclusion, *A. johnsonii* has been recognized as an important pathogen in PDAP, which can be treated successfully with appropriate antibiotic treatment based on antibiotic susceptibility pattern.

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