

# *Burkholderia* Septicemia: A Case Report

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## ABSTRACT

*Burkholderia cepacia* is a bacterial pathogen which usually causes infection in immunocompromised or hospitalized patients. It also causes associated infections in patients with underlying pulmonary diseases, such as cystic fibrosis and chronic granulomatous diseases. It is often resistant to multiple antibiotics and has been found to grow even in medium containing penicillin. Here, we report a case of *B. cepacia* bacteremia in a post-operative patient of rheumatic heart disease with bilateral pleural effusion who, on being timely diagnosed, was managed effectively with appropriate antibiotics.

**KEY WORDS:** *Burkholderia cepacia*, rheumatic heart disease, septicemia.

## Introduction

*Burkholderia cepacia* is a pathogen that causes infection usually to immunocompromised or hospitalized patients. It is also associated with infections in patients with underlying lung disease, such as cystic fibrosis and chronic granulomatous disease. It may cause “cepacia syndrome” which is a form of progressive necrotizing pneumonia. It is accompanied by an acute systemic infection and may often prove fatal. The organism is often resistant to multiple antibiotics and has been shown to grow in penicillin medium. Here we report a case of *B. cepacia* bacteremia in a post-operative patient of rheumatic heart disease with bilateral pleural effusion. The patient was accurately diagnosed on time and hence, managed effectively with appropriate antibiotics.

## Case Report

A 63-year-old male with the complaints of breathlessness and chest discomfort was referred to the emergency department of our hospital for medical stabilization. He was a known case of rheumatic heart disease with severe mitral stenosis and moderate atrial regurgitation with bilateral

pleural effusion. On examination, the patient was afebrile with a body temperature 98.4°F, pulse rate of 48/min, and blood pressure 100/60 mmHg. The patient was admitted and shifted to cardiac care unit (CCU) where he was put on oxygen support and later on the ventilator. Echocardiography showed severe calcified mitral stenosis, mild aortic and tricuspid regurgitation, normal left ventricular (LV) systolic function, and LV ejection fraction-60% with normal right ventricular function. Since there was no improvement, the patient was taken up for mitral valve replacement following which the patient was weaned off from ventilator. Two days later, the patient became febrile and the general condition also deteriorated, so he was put on ventilator again. CT scan of thorax revealed bilateral pleural effusion with basal lung collapse. The patient's laboratory investigations revealed leukocytosis with the TLC of 16,000/mm<sup>3</sup>. Serum creatinine and blood urea were 1.1 mg/dl and 92 mg/dl, respectively. Pleural fluid and urine were reported as sterile on culture. Blood samples were sent for culture on two separate occasions where both times, the culture showed pure growth of non-lactose fermenting, mucoid, smooth colonies with diameter of approximately 2 mm on MacConkey agar. On Gram staining, the colonies were found to be Gram-negative rods which were motile and tested positive for catalase and oxidase production and also for nitrate reduction. They also oxidized sugars which included glucose, lactose, mannitol, and maltose. They decarboxylated lysine but did not hydrolyze arginine. The antibiotic susceptibility test showed that the organism was

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susceptible to trimethoprim-sulfamethoxazole, imipenem, levofloxacin, and piperacillin-tazobactam but resistant to aztreonam, gentamicin, ampicillin, cephalosporins, and polymyxin B. The organism was subjected to identification by automated VITEK2 Compact system and was identified as *Burkholderia cepacia*. Piperacillin-tazobactam (4.5 g, 8 h) and ciprofloxacin (100 mL, 8 h) were started intravenously, following which the fever subsided. The general condition of patient also improved gradually and he was finally discharged from the hospital after complete stabilization.

## Conclusion

*B. cepacia* is a member of a group known as *B. cepacia* complex. It comprises nine different recognized genomovars, including *Burkholderia multivorans*, *Burkholderia cenocepacia*, *Burkholderia stabilis*, *Burkholderia vietnamiensis*, *Burkholderia dolosa*, *Burkholderia ambifaria*, *Burkholderia anthina*, and *Burkholderia pyrrocinia*.<sup>[1]</sup> It is innately resistant to various antimicrobial agents including polymyxins, aminoglycosides, chloramphenicol, and b-lactams. Mechanisms conferring resistance include efflux pumps, production of b-lactamases and other modifying enzymes, as well as modification of antibiotic targets.<sup>[2,3]</sup>

There are many reports of *B. cepacia* sepsis in patients with cystic fibrosis and some reports of catheter-induced *B. cepacia* bacteremia in hemodialysis patients.<sup>[4]</sup> A case of septicemia due to *B. cepacia* has been reported in a patient with COPD also.<sup>[5]</sup>

We report a case of *B. cepacia* septicemia in a patient of rheumatic heart disease with pleural effusion who had undergone mitral valve replacement. A case of endocarditis due to *B. cepacia* has also been reported in a neonate.<sup>[6]</sup>

*B. cepacia*, the emerging pathogen, is overlooked in many cases due to the low index of suspicion and awareness among microbiologists and clinicians. Therefore, due consideration should be given to this organism to know the true magnitude of its infection in our country. As *B. cepacia* is a non-fastidious organism without any exacting growth requirements, all non-fermenters should be subjected to speciation as a part of routine microbiological work-up.

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