Dual Infection by *Burkholderia Cepacia* and *Pseudomonas Putida* in an Infective Endocarditis Case

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**ABSTRACT**

Infective endocarditis is rarely caused by *Burkholderia cepacia*. *Pseudomonas putida* has not been reported to cause infective endocarditis so far. This is the first case of infective endocarditis being reported, that is caused by *Pseudomonas putida* and *Burkholderia cepacia* in an immunocompetent host with no predisposing factors. Aortic valve replacement surgery was carried out and antibiotics were given, to which the patient responded well and recovered.

**Key Words:** Infective endocarditis. *Burkholderia cepacia*. *Pseudomonas putida*.

**INTRODUCTION**

*Burkholderia cepacia*, a Gram-negative bacillus, is notorious for causing nosocomial infections, especially in patients suffering from cystic fibrosis and chronic granulomatous diseases.\(^1\) It has intrinsic resistance against many antibacterial agents. Infective endocarditis caused by *Burkholderia cepacia* is relatively uncommon in immunocompetent people; however, it may cause infective endocarditis in intravenous drug users and in patients with prosthetic valve replacement.\(^2\) Treatment of *Burkholderia cepacia* may either be conservative or surgical; however, mortality is high despite aggressive treatment.\(^3,4\)

*Pseudomonas putida*, member of the fluorescent group of *Pseudomonads*, can colonize moist and inanimate surfaces. It was previously thought to be of low pathogenicity; but now it has been implicated in nosocomial infections, especially in the immunocompromised and those with invasive medical devices.\(^5\) To date, a variety of infections caused by *Pseudomonas putida* have been reported, including wound infections, device related infections, acute cholecystitis and cholangitis, tonsillitis, thrombophlebitis and skin and soft tissue infections.\(^5,6\) There is higher antimicrobial susceptibility of *Pseudomonas putida* as compared to other *Pseudomonas* spp.\(^5,7\)

To our knowledge, we present the first case of infective endocarditis caused by *Pseudomonas putida* and *Burkholderia cepacia* in an immunocompetent host with no predisposing factors.

**CASE REPORT**

A 44-year known diabetic man was referred to the cardiology unit of the hospital from a local clinic with complaints of fever, chills, fatigue and shortness of breath for the past two months. He fell unconscious a day before admission. There was no history of any intravenous drug use. On examination, vital signs revealed a temperature of 39ºC, heart rate 90/minute, respiratory rate 17/minute, and blood pressure 125/80 mmHg. On auscultation, a diastolic murmur was revealed at left sternal border.

His laboratory investigations revealed, total leukocyte count of 5.1x10\(^9\)/L, red blood cell count 4.41x10\(^12\)/L, haemoglobin 13g/dL, and platelet count 152x10\(^9\)/L. Differential leukocyte count revealed neutrophils 62%, lymphocytes 29%, monocytes 5%, and eosinophils 4%; ESR was 14mm at 1st hour. Fasting blood sugar was 5.1 mmol/l. Paired blood cultures yielded no growth of organism at 37ºC after 7 days of incubation. There were no significant findings in electrocardiography.

Chest X-ray was insignificant. Echocardiography revealed 2.5 cm\(^2\) vegetation on the aortic valve. Transesophageal echocardiography revealed a large aortic vegetation on the anterior cusp of the aortic valve measuring 2.5 cm\(^2\) resulting in thickened calcific aortic valve with moderate aortic regurgitation. However, left ventricular function was normal. Aortic annulus measured 25 mm, and other valves appeared normal. There was no evidence of diastolic dysfunction and pericardial effusion. Aortic arch was normal. 2D echocardiography showed left ventricular ejection fraction of 60%. The patient was started empirically on an intravenous imipenem/ cilastatin, 1gm 8 hourly and vancomycin 15 mg/kg body weight 12 hourly. Despite treatment, intermittent low-grade fever was persisted. Aortic valve replacement surgery was carried out and the excised tissue was sent for culture and susceptibility testing to our laboratory.

The sample was processed in the microbiology department. It was put in Brain Heart Infusion broth. The
sample was inoculated on blood agar (Oxoid, UK) and MacConkey agar (Oxoid, UK). After 24 hours, two distinct types of small colonies were observed, which could be appreciated better after 48 hours of incubation. Gram staining of both the colonies revealed Gram negative rods. Both isolates were non-lactose fermenters, catalase positive but one was oxidase positive. Smooth and brownish colonies were similar to *Burkholderia cepacia*. Smooth, pigmented and shiny colonies were appreciated as *Pseudomonas putida*. The organisms were confirmed on API 20 NE (biomerieux, France) as *Burkholderia cepacia* and *Pseudomonas putida*. Antibiotic susceptibility testing was performed by agar dilution method for minimum inhibitory concentration (MIC) determination by multipoint inoculators system; interpretation was determined using Clinical and Laboratory Standard Institute (CLSI) guidelines. *Burkholderia cepacia* was found to be susceptible to ceftazidime (4 µg/ml), levofloxacain (1 µg/ml), and ceftriaxone (1/19 µg/ml). It showed intermediate susceptibility to minocycline (8 µg/ml) and was resistant to meropenem (32 µg/ml) and chloramphenicol (32 µg/ml). *Pseudomonas putida* was found to be susceptible to cefpime (4 µg/ml), ciprofloxacin (0.5 µg/ml), imipenem (1 µg/ml), levofloxacain (1 µg/ml), polymyxin B (0.5 µg/ml) and piperacillin/tazobactam (8/4 µg/ml). It was resistant to amikacin (64 µg/ml), aztreonam (32 µg/ml), ceftazidime (64 µg/ml), gentamicin (16 µg/ml), and meropenem (8 µg/ml). *Pseudomonas aeruginosa* ATCC27853 was used as control strain.

After susceptibility report, antimicrobials were changed to ciprofloxacin 400 mg IV, 8 hourly; piperacillin/tazobactam 4.5 gm, 6 hourly, and vancomycin 15 mg/kg body weight, 12 hourly. The patient responded well and with clinical improvement and fever started settling down. The patient was discharged one week after the surgical intervention, with oral ciprofloxacin 500 mg, 12 hourly.

**DISCUSSION**

Dual infection in a native valve is a rarity; this makes our case very unique. This has extreme clinical importance because of its implications and appropriate treatment. Symptomatically, it is not possible to differentiate that whether it is single or dual organism infection; however, the aggressive nature of certain causative microorganisms carries significance. *Burkholderia cepacia* is known to cause native valve endocarditis. In our patient, normal aortic valve was affected as the patient did not have any predisposing factors; and native valve endocarditis caused by this organism is rare.2,4 Therapeutic options for *Burkholderia cepacia* are unfortunately limited because many strains of this organism exhibit high levels of resistance to many antimicrobial agents in vitro, which may be intrinsic to certain cases. Studies report up to 50.4% resistance to every antibiotic tested, indicating that multidrug resistant isolates are common. The antimicrobial option, most commonly used in *Burkholderia cepacia* infection, is co-trimoxazole (trimethoprim/sulfamethoxazole). There is some hurdle to the use of co-trimoxazole, since allergic or hypersensitivity reactions, intolerance, and resistance may be observed in patients receiving co-trimoxazole.8

Outbreaks of bloodstream infection associated with contaminated fluids have also been reported.9 Strains of this species have occasionally been isolated from patients in hospitals in Japan, the United States, Italy and France. Infections by these microorganisms have been linked to insertion of catheters or drainage tubes.10 There has been a case of early prosthetic valve endocarditis with *Burkholderia cepacia* which was effectively treated with six weeks parenteral antibiotics (trimethoprim-sulfamethoxazole, levofloxacain, and ceftazidime) along with re-do mitral valve replacement.11 Another previously reported *Burkholderia cepacia* induced native valve endocarditis case with consequent cerebral involvement without any predisposing factors was treated successfully by antimicrobial agents only.4 A renal transplant patient has been reported in Brazil for late infective endocarditis with positive blood cultures for *Burkholderia cepacia* and was associated with an intracardiac fragment of a previously inserted catheter.12 *Burkholderia cepacia* complex can be transmitted among humans, both inside and outside the hospital environments; contaminated intravenous solutions and drugs are common sources of hospital infections.13

To our knowledge, this is the first case of infective endocarditis implicating two different pathogens – *Burkholderia cepacia* and *Pseudomonas putida* in an immunocompetent person. Furthermore, *Pseudomonas putida* has never been previously reported as a source of endocarditis. Another thing that makes our patient unique is that *Burkholderia cepacia* usually involves tricuspid valve, and mitral valve is less commonly involved.2,3 So far only one case of infective endocarditis affecting the aortic valve, caused by this pathogen, has been reported in a patient without any predisposing factors.9

Due to high antimicrobial resistance against *Burkholderia cepacia*, combination of drugs and surgery is usually required for successful treatment.2 Keeping in mind the susceptibility of both organisms, our case responded well to combination of piperacillin/tazobactam, ciprofloxacin, and vancomycin. Successful outcome with antimicrobial drugs only has been reported in a few cases, as well.4

Infective endocarditis caused dual infection with *Burkholderia cepacia* and *Pseudomonas putida* in a previously healthy individual is a very rare clinical entity. Our case responded well due to proper identification and
appropriate treatment, combination of antimicrobial therapy, and surgery. The hallmark was exact identification of the pathogen and proper treatment.

REFERENCES


