Case Report

Extensively drug-resistant *Pseudomonas putida* bacteremia that was resolved spontaneously

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Abstract

*Pseudomonas putida* (*P. putida*) is a rare pathogen that causes various infections in newborns, neutropenic and cancer patients, or in patients with risk factors leading to immunosuppression. Antibiotic resistance in *P. putida* is seen in growing numbers. Although it is less virulent compared to *Pseudomonas aeruginosa*, mortal infections are reported.

Here, a *P. putida* case after an invasive procedure in a patient with gastrointestinal malignancy is reported. Although it caused an antibiotic resistant bacteremia, it resolved spontaneously without any treatment. *P. Putida* might have lower virulence and a different antibiotic susceptibility when compared to *Pseudomonas aeruginosa* in different cases. More clinical information is needed for further evaluation.

Key words: *Pseudomonas putida*; extensive drug-resistance; low virulence.


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Introduction

*Pseudomonas putida*, is a non-fermenting Gram-negative bacillus from Pseudomonadaceae family. It is fluorescent, has variable metabolic features and exists in soil [1]. It usually presents as skin and soft tissue infection, pneumonia, urinary infection or bacteremia in immunocompromised or traumatized patients. It can colonize on moist and inanimate hospital surfaces and cause nosocomial infection. Also, bloodstream infections associated with contaminated intravenous fluids, intensive care units, specialized wards or war wounds have been reported [2-5]. It shows altered pathogenicity, which might result as a fatal infection. [6,7].

*P. putida* is usually susceptible to antipseudomonal carbapenem treatment. Here, a case report presenting with fever after a distal esophageal stent replacement for malignancy, that was diagnosed with *P. putida* bacteremia and found to be multidrug resistant including carbapenem and resolved spontaneously is presented.

Case Presentation

A 59- year-old male patient presented to our hospital with belly pain and dark stool. He had been diagnosed with distal esophageal adenocancer in an outpatient clinic 18 months ago and he received only chemotherapy as the cancer was inoperable. He had not been admitted to hospital before or given any other medications than chemotherapy. He lost 10% of his body weight due to malnutrition and has had poor oral intake for the past week. His body mass index was 20 kg/m² and he had decreased skin turgor and muscular tonus. His pathological laboratory results were: Hb; 8 gr/Lt (N: 13.5-18.1), Hct; 24% (N:40-53.7), Total protein; 6.2 gr/Lt (N:6.6-8.3), Albumin; 2.1 gr/Lt (N:3.5-5.2), Procalcitonin; 0.76 ng/mL (N 0-0.5) and hsCRP 159 mg/L (N 0-5).

Supportive treatment was given for his acute upper gastrointestinal bleeding. Upper gastrointestinal tract gastroscopy was performed. Esophagus was completely obstructed in distal region by tumoral lesion. The tumor was passed by guide and passage to stomach was succeeded with replacement of a 120*20 cm partially coated antireflux esophageal stent. Passage clearance was observed following an oral intake in 24 hours. The patient developed fever (38.3 °C) 48 hours after stent replacement. 2 blood cultures (from 2 different arms with a half-an-hour break) were taken and *P. putida* growth was detected in the cultures on the 5th day. Meantime, as the patient’s fever did not last and
A) The media showed no further symptoms of infection. Hospital, the fever reduced spontaneously and he subfebrile fever for 2 days after his discharge from developmental cycles. Fass et al. declared 100% susceptibility to carbapenem, ciprofloxacin, ceftazidime and colistin and intermediate-susceptibility to amikasin (Table 1). The pathogen was accepted as XDR (extensively drug resistant= non-susceptibility to at least one agent in all but two or fewer antimicrobial categories) [9]. The patient refused the treatment and left hospital although the possible outcomes were declared clearly. After 20 days, he presented to emergency room with recurrent upper gastrointestinal bleeding. He stated that he had subfebrile fever for 2 days after his discharge from hospital, the fever reduced spontaneously and he showed no further symptoms of infection.

**Discussion**

*P. putida* is a bacterium that is rarely isolated in infections. It exists in soil and is not a part of normal human bacterial flora [10,11]. It is a non-lactose fermenting, oxidase-positive Gram-negative bacilli and a member of the fluorescent group of pseudomonas. It produces pyoverdin, a yellow–green pigment that fluoresces under UV light [12]. It can cause pandemics as nosocomial infection after invasive procedures in hospitals [13-15]. It is isolated in sputum, skin lesions and urine. Presence of a solid tumor or a hematological malignancy increases tendency to develop infection caused by *P. putida* [16].

Yang et al. showed that 55 % of *P. putida* infection was nosocomial and its incidence was higher among patients who underwent invasive procedures like placement of intravascular devices, urinary catheterization and intubation [13].

Yoshino et al. isolated five *P. putida* bacteraemia in a 4-year-period and declared that four of them had medical devices as the primary infection site. The percentage of *P. putida* isolation was 0.22% in their hospital [6]. On the other hand, Anaissie et al. identified *P. putida* on 3 of 15 cancer patients’ cultures and they were all catheter-related and one of them resolved spontaneously after catheter removal [17].

*P. putida* is a bacterium that can promote formation of a biofilm [18]. In medical device-related infections, microorganisms adhere to materials and biofilm development is accelerated. This feature is an important part of its pathogenicity [19]. *P. putida* can colonize in the normal oropharyngeal flora and also in the intestine to cause cholecystitis [20,21]. Our case is also probably a medical device-related infection as bacteremia developed after a stent implantation to esophagus.

Prognosis of *P. putida* bacteraemia is usually good and the treatment should be planned according to antimicrobial susceptibility as it is in other infections. Removal of infected medical implants and debridement of the wound if present are also important. After removal of the source, the blood culture result should be repeated with clinical stable time.

In previous reports, clinical isolates of *P. putida* showed high susceptibility to various antibiotic regimens. Fass et al. declared high susceptibility to various antibiotic regimens. Fass et al. declared 100% susceptibility to

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**Table 1. Antibiotic resistance patterns of isolated bacteria.**

<table>
<thead>
<tr>
<th>Antibiotic name</th>
<th>Zone diameter</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piperacillin zone diameter</td>
<td>6mm</td>
<td>Resistant</td>
</tr>
<tr>
<td>Piperacillin/tazobactam</td>
<td>10mm</td>
<td>Resistant</td>
</tr>
<tr>
<td>Cefazidime</td>
<td>12mm</td>
<td>Resistant</td>
</tr>
<tr>
<td>Cefepime</td>
<td>10mm</td>
<td>Resistant</td>
</tr>
<tr>
<td>Imipenem</td>
<td>10mm</td>
<td>Resistant</td>
</tr>
<tr>
<td>Meropenem (6mm (MIC = 32))</td>
<td>Gradient E-Test</td>
<td>Resistant</td>
</tr>
<tr>
<td>Amikacin</td>
<td>16 mm</td>
<td>Intermediate-susceptible</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>13mm</td>
<td>Resistant</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>6mm</td>
<td>Resistant</td>
</tr>
<tr>
<td>Colistin</td>
<td>10 mm</td>
<td>Resistant</td>
</tr>
</tbody>
</table>

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ciprofloxacin and tobramycin and 87% susceptibility to imipenem and piperacillin / tazobactam [22]. But still carbapenem resistant *P. putida* infections in sputum, blood and urine cultures were reported [23,24]. Kim et al. reported that 4 of their 18 *P. putida* isolates were resistant to imipenem (22%) and 5 (28%) were resistant to meropenem [25]. Our isolate showed resistance to imipenem, meropenem, ceftazidime and colistin and intermediate-susceptibility to amikacin.

Several mechanisms are suggested for carbapenem resistance; derepression of chromosomal AmpC cephalosporinase; production of plasmid or integron-mediated beta-lactamases from different molecular classes (carbenicillinases and extended-spectrum beta-lactamases belonging to class A, class D oxacillinases and class B carbapenem-hydrolysing enzymes); diminished outer membrane permeability, overexpression of active efflux systems with wide substrate profiles [26-29]. In our country, most common carbapenem resistance mechanisms in *P. aeruginosa* infection are the ones mediated by metallo-β-lactamase (MBL) and OXA-23 carbapenemase enzymes but also oprD downregulation and mexB, mexY, and mexD overexpression were demonstrated [30-34].

Generally, *P. putida* is considered less virulent and clinically less important than *Pseudomonas aeruginosa*. [35]. In his mini review, Yoshino et al. declared that in his *P. putida* -case-series, *P. putida* recovery rate was 93 % [6]. Kim et al. declared their recovery rate as 40%. The difference could be due to severity of their cases as they had pneumonia which worsened the prognosis [25]. Yank et al. [13] reported that in one of their two cases in which the patient died, inappropriate antibiotic therapy for co-pathogens could have been a contributing factor. Our case had only presented fever and although the infection was resistant to carbapenem, it resolved spontaneously which could be interpreted as the pathogen had low virulence.

Although we did not have the chance to use them, there are novel treatment options for serious infections that are difficult to treat. Ceftazidime-avibactam and ceftolozane-tazobactam are promising agents for such complicated infections [36,37].

In conclusion; *P. putida* produces an infection with higher prevalence in patients with co-morbidities and medical device implants. It might have lower virulence and a different antibiotic susceptibility profile when compared to *P. aeruginosa* in different occasions. Thus, more clinical information is needed to understand its nature and possible outcomes.

### References


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