Pseudomonas putida is an aerobic non-lactose fermenting gram-negative and oxidase positive bacillus that is commonly found in the environment including soil. Because P. putida can colonize moist and inanimate hospital surfaces, it causes nosocomial infections, especially in immunocompromised patients, gaining access to sterile body compartments through invasive medical devices or traumatic mucocutaneous defects. We report a case of nosocomial carbapenem-resistant P. putida meningitis following a neurosurgical procedure in a 26-year-old male patient. The objective of presenting this case is to increase doctors’ awareness of this infection, and review previous cases of P. putida meningitis to provide a concise review of the risk factors, clinical picture, management, and outcome of infections due to this organism.

**CASE REPORT**

A 26-year-old Nepalese man presented to the emergency department with a two-week history of progressive right sided weakness, headache, and repeated vomiting. His past medical history was unremarkable. Clinical examination showed right sided weakness of 3/5 on the medical research council scale. Tendon reflexes were exaggerated in the right side with up-going planter reflex.

Brain computed tomography (CT) and magnetic resonance imaging (MRI) showed right cystic lesion rising from the quadrigeminal cistern on the left side and extending supratentorially, indenting the third ventricle causing obstructive hydrocephalus. The most probable diagnosis was intracranial epidermoid cyst. Ommaya reservoir was placed into the cyst for drainage and external ventricular drainage (EVD) was performed. After four days, the patient was transferred to the ward in stable condition. His weakness resolved partially and headache severity decreased. After three days, the patient developed fever and headache severity increased with deterioration of consciousness level. Cerebrospinal fluid (CSF) through EVD showed 2,200 leucocytes/µL, protein level of 295 mg/dL, and glucose level of < 1.8 mg/dL. Meropenem was started on the patient. Aspirate from Ommaya reservoir and CSF showed gram-negative rods and cultures yielded P. putida sensitive to cefepime, gentamycin, ciprofloxacin, and amikacin, but resistant to meropenem and piperacillin-tazobactam. EVD was replaced and the patient received cefepime and ciprofloxacin for 21 days after which he improved and was discharged with right sided residual weakness.
<table>
<thead>
<tr>
<th>Reference</th>
<th>Age/sex</th>
<th>Infection pattern</th>
<th>Underlying conditions</th>
<th>Clinical picture</th>
<th>Antibiotic treatment</th>
<th>Antibiotics susceptibility</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ghosh et al</td>
<td>19/F</td>
<td>CA</td>
<td>β-thalassemia major, S/P splenectomy</td>
<td>Fever, altered consciousness, neck rigidity</td>
<td>Cefuroxime, Piperacillin</td>
<td>S, S, ND, ND, ND, ND, ND, ND, S</td>
<td>Cured</td>
</tr>
<tr>
<td>Yang et al</td>
<td>71/F</td>
<td>NOCL</td>
<td>Hg stroke, S/P ventriculoperitoneal shunt</td>
<td>Fever, altered consciousness</td>
<td>Cefotaxime, Gentamicin, Cefazidime, Amikacin</td>
<td>ND, ND, S, S, R, ND</td>
<td>Cured</td>
</tr>
<tr>
<td>Yang et al</td>
<td>77/F</td>
<td>NOCL</td>
<td>HTN, Hg stroke, S/P ventriculostomy and EVD insertion</td>
<td>Fever, altered consciousness</td>
<td>Ceftriaxone, Chloramphenicol, Cefazidime, Amikacin</td>
<td>ND, ND, S, S, R, ND</td>
<td>Cured</td>
</tr>
<tr>
<td>Tsuru et al</td>
<td>79/F</td>
<td>CA</td>
<td>None</td>
<td>Fever, vomiting, altered consciousness</td>
<td>Ceftriaxone, Vancomycin, MEM</td>
<td>ND, S, ND, ND, S, S, ND, ND, ND</td>
<td>Died</td>
</tr>
<tr>
<td>Khan et al (Present case)</td>
<td>26/M</td>
<td>NOCL</td>
<td>S/P Ommaya reservoir and EVD insertion</td>
<td>Fever, headache, altered consciousness</td>
<td>MEM, Cefepime, Ciprofloxacin</td>
<td>ND, S, R, S, S, R, ND, ND, ND</td>
<td>Cured</td>
</tr>
</tbody>
</table>

M: male; F: female; NOCL: nosocomial; CA: community acquired; S/P: status post; EVD: external ventricular drain; HTN: hypertension; Hg: hemorrhagic; CTR: ceftriaxone; FEP: cefepime; TAZ: Piperacillin/tazobactam; Pip: Piperacillin; CIP: ciprofloxacin; MEM: meropenem; GEN: gentamicin; Ctz: Cefazidime; Amk: amikacin; Cf: Cefotaxime; R: resistance; S: sensitive; ND: not done.
**DISCUSSION**

*P. putida* has been recognized as a rare pathogen of meningitis in adult patients. To our knowledge, this is the first case reported from Qatar and only four cases have been reported in the literature.1–4 Table 1 describes the demographic and clinical data of reported cases of *P. putida* meningitis.

Data in this table demonstrated that the vast majority of *P. putida* meningitis cases were females with the age ranged between 19 and 79 years. The infection was nosocomial in three cases and community acquired in two cases. Most cases occurred in patients who underwent invasive neurological procedures (e.g., craniotomy, placement of internal catheters, or EVDs), and in patients with conditions that weaken their immune system, such as those who undergone splenectomy.2,3 However, *P. putida* meningitis occurred in healthy individuals with no known predisposing condition,4 challenging the clinical suspicion to consider it as a differential diagnosis, especially in the setting of the non-specific clinical features of this form of disease, such as fever, altered level of consciousness, headache, seizures, and signs of meningeal irritation, which are indistinguishable from other forms of bacterial meningitis. Therefore, a high index of suspicion is very important for the diagnosis. Similarly, the CSF (biochemistry and cell count) findings in this series were indistinguishable from other forms of bacterial meningitis. These findings made CSF gram staining and culturing crucial in all patients.

The susceptibility profile of *P. putida* isolates to various antimicrobials in Table 1 is variable. The β-lactam resistance pattern was unusual for *P. putida*.6 In particular, resistance to carbapenems suggests the possibility of carbapenemase production or another acquired resistance mechanism. The emergence of carbapenemases, such as acquired metallo-β-lactamases and other β-lactamases affecting carbapenems, is becoming a therapeutic challenge because these enzymes confer a high level of resistance to most β-lactams, including carbapenems with the exception of aztreonam.3 Our patient showed resistance to carbapenems and piperacillin-tazobactam. This finding suggests the emergence of carbapenem-resistant *P. putida*, and gives the alarming signal for the future, making the therapeutic options more difficult especially in empirically treating post-neurosurgical meningitis. The outcome of the treatment of *P. putida* meningitis is unclear due to paucity of cases, but as described in Table 1, it seems good since all patients except one had clinical improvement.

**CONCLUSION**

*P. putida* is a rare pathogen of meningitis as evident by the paucity of cases in the literature. As the clinical picture and CSF (biochemistry and cell count) findings were indistinguishable from other forms of bacterial meningitis, there needs to be a high index of suspicion for this diagnosis. Thus, *P. putida* should be suspected in patients with a new onset fever or an unexplained change in their neurological status especially in the presence of predisposing conditions, and confirmed by CSF gram staining and culturing. We suggest performing regular CSF studies on high risk patients for early diagnosis and effective treatment of these infections. Moreover, strict infection control measures are to be followed to contain the *P. putida*.

**Disclosure**
The authors declared no conflicts of interest.

**References**