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Ralstonia mannitolilytica and COPD: a case report

To the Editors:

Ralstonia mannitolilytica is a recently established species of clinical significance and was previously known as *Pseudomonas thomasii* or *Ralstonia pickettii* biovar 3/*thomasii* [1]. It has been recovered from the respiratory tract of patients with cystic fibrosis and has also been associated with catheter-associated bacteraemia, recurrent meningitis, infection of a haemoperitoneum, urinary tract infection and post-renal transplant infection. Hospital outbreaks of *R. mannitolilytica* due to contamination of water [2], saline solutions [3] or oxygen-delivery devices [4] have also been reported. However, this bacterium has not been reported in patients with respiratory illnesses other than cystic fibrosis.

Isolate G100 was recovered from a sputum sample from a male, 78-yr-old patient in April, 2010. This patient was presented with cough and gradually worsening dyspnoea for 1 month, but without fever. He received no antimicrobial agents prior to admission. This patient had a 20-yr history of intermittent cough, and chronic obstructive pulmonary disease (COPD) was diagnosed 10 yrs previously. He had also had type II diabetes mellitus for 5 yrs and had been a cigarette smoker for >20 yrs, but had stopped smoking 10 yrs previously. Physical examination revealed a ''barrel-shaped'' chest, reduced breath sounds and crackles. On admission, a full blood count revealed haemoglobin 15.1 $g \cdot dL^{-1}$, white cell count 5.93×10^9 $cells \cdot L^{-1}$ (neutrophils 4.49×10^9 $cells \cdot L^{-1}$ and lymphocytes 1.25×10^9 $cells \cdot L^{-1}$) and platelets 187×10^9 $cells \cdot L^{-1}$. Routine serum chemistry was normal. Blood-gas analysis revealed pH 7.28, oxygen tension 64 mmHg, carbon dioxide tension 80 mmHg, HCO_3^- 37.6 $mmol \cdot L^{-1}$ and arterial oxygen saturation 89%, suggesting type II respiratory failure and respiratory acidosis. High-resolution chest computed tomography on admission revealed barrel-shaped chest and increased lung markings, but no infiltrations. He was diagnosed with an acute exacerbation of COPD and a sputum sample was sent on admission, from which G100 was isolated. The sputum sample was of good quality when examined by microscopy and Gram-negative rods were detected.

G100 was identified as a *Ralstonia* sp. of Centers for Disease Control group II using a MicroScan Walkaway 96 SI automated system (Siemens Healthcare Diagnostics, Deerfield, IL, USA). Species identification was performed by partially sequencing the 16S ribosomal RNA (rRNA) gene amplified with universal primers 27F and 1492R [5]. The 1,405-bp partial 16S rRNA sequence of G100 was identical to that of *R. mannitolilytica* strain AU0428 from a cystic fibrosis patient in the USA (GenBank accession number AY043378) [6], strains LMG 19090 (LBV407; AJ270257) and LMG 19091 (LBV371; AJ270256) from cases of recurrent meningitis and haemoperitoneum infection in Belgium [1], and many uncultured clones (*e.g.* GQ417788) from environmental samples in France.

Random amplification of polymorphic DNA (RAPD) typing has been used previously to determine the clonal relatedness of *R. mannitolilytica* isolates. Using the *R. pickettii* RAPD primers P3 (5′-AGACGTCCAC-3′) and P15 (5′-AATGGCGCAG-3′) [7], 30 *R. mannitolilytica* clinical isolates from Austria have been classified four distinct genotypes [8]. RAPD using P3 and P15 was performed as described previously [7], revealing that G100 belonged to a genotype different from those seen in Austria (data not shown).

G100 was resistant to amikacin, gentamicin, tobramycin, ampicillin, ampicillin/sulbactam, amoxicillin/clavulanate, piperacillin, ticarcillin/clavulanate, cefazolin, ceftazidime, cefoxitin and aztreonam, intermediate to cefotaxime and cefepime, and sensitive to ceftriaxone, piperacillin/tazobactam, imipenem, ciprofloxacin, levofloxacin and trimethoprim/sulphamethoxazole, as determined with the Walkaway system using Negative Combo panel type 31.

Piperacillin/tazobactam (Wyeth, Beijing, China), 4.5 g every 8 h, was initiated as empirical therapy and continued after the culture result was available, since G100 was sensitive to this compound. This patient also received noninvasive ventilation. However, The patient's condition gradually deteriorated. Therefore, after 9 days of therapy, piperacillin/tazobactam was replaced by panipenem/betamipron (Daiichi Sankyo, Beijing, China) but patient's condition did not improve. After 1 month of hospitalisation, the patient received endotracheal intubation and was transferred to the intensive care unit (ICU) due to deteriorating respiratory failure. Various microorganisms, including *Aspergillus* spp., *Candida albicans*, *Klebsiella oxytoca*, *Klebsiella planticola*, *Acinetobacter baumannii* and *Pseudomonas aeruginosa*, were recovered from the sputum samples collected at different time-points before or after invasive ventilation.

Although antibacterial (*e.g.* cefepime, cefoperazone/sulbactam and moxifloxacin) and antifungal agents (itraconazole) were administered intravenously according to the microbiological results, the patient had no response and finally died of respiratory failure after a 28-day ICU stay.

The isolation of G100 was unlikely to have been due to contamination, based on the following observations. First, the isolation of *Ralstonia* spp. from clinical samples is extremely rare at our centre and no other cultures in West China Hospital grew *Ralstonia* spp. in this year. Secondly, no saline solutions or water were used to collect the sputum sample, which was collected prior to noninvasive ventilation. Thirdly, Gram-negative rods were detected in the sputum by microscopy. As G100 was recovered from a sample collected on admission and this patient had no recent history of hospitalisation, this isolate probably had a community origin. After administration of piperacillin/tazobactam, no *R. mannitolilytica* was isolated from sputum samples collected from this patient afterwards, but the patient's condition did not improve. This suggests that G100 was probably not the causative agent, or at least, not the sole causative agent of the acute exacerbation of COPD in this patient. It is more likely that *R. mannitolilytica* had colonised in the respiratory tract in this case.

R. mannitolilytica has been recovered from the respiratory tract of patients with cystic fibrosis [6]. However, cystic fibrosis is not common in China and this patient did not have this disease. As microbial colonisation of airways could lead to exacerbations of COPD, the isolation of *R. mannitolilytica* in the respiratory tract of the COPD patient could be of clinical importance. As misidentification is common, *R. mannitolilytica* might be an overlooked member of the bacterial flora in the respiratory tract of COPD patients. *Ralstonia* spp. (identified as *R. pickettii*, but without describing the method for identification) have previously been found to cause COPD exacerbation [9]. Nonetheless, our report describes a rare case of *R. mannitolilytica* associated with a COPD patient and, to our knowledge, this case is the first identification of *R. mannitolilytica* from clinical samples in mainland China.

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Can transmissible strains of *Pseudomonas aeruginosa* be successfully eradicated?

To the Editors:

Recent microbiological surveillance using molecular typing (genotyping) has provided compelling evidence for *Pseudomonas aeruginosa* cross-infection at many European, Australian and Canadian cystic fibrosis (CF) centres [1–4]. The transmissible strains responsible for this cross-infection pose an increased risk for acquisition of infection for patients currently free of *P. aeruginosa*. As transmissible strains are often resistant to multiple antibiotics, they may also be more difficult to eradicate. At present, there is a paucity of evidence regarding this. We therefore report the efficacy of eradication therapy in a cohort of six patients who acquired a transmissible strain as their first isolate of *P. aeruginosa*.