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Letter to the Editor

Ocular infection associated with *Delftia lacustris*: first report



Dear Editor,

Delftia is an aerobic, Gram-negative, oxidase-positive, non-glucose-fermenting bacillus. *Delftia* species are ubiquitous in water and soil.¹ However, they are rarely associated with human infections. To date, four *Delftia* species (*D. acidovorans*, *D. tsuruhatensis*, *D. lacustris*, and *D. litopenaei*) have been described. *D. acidovorans* (formerly known as *Comamonas acidovorans*) and *D. tsuruhatensis* have been reported as causes of human infections such as catheter-related bacteremia, pneumonia, empyema, peritonitis in a patient receiving peritoneal dialysis, urinary tract infections, and ocular infections.² However, there has been no report of ocular infection by *D. lacustris*. Herein, we present a patient with keratitis and probable endophthalmitis caused by *D. lacustris*.

A 70-year-old male farmer visited the hospital with a complaint of painful foreign body sensation and epiphora in his left eye. His symptoms developed following non-penetrating eye trauma from a tree branch while picking red peppers two weeks earlier. He had been taking anti-hypertensive and anti-diabetic medications for over 30 years, but was otherwise in good health. On examination, visual acuity without correction was 20/25 in his right eye and he could count fingers at 30 cm using his left eye. The left eye demonstrated a corneal infiltrate and an approximately 1 mm corneal epithelial defect. The right eye was clear. Cultures of corneal scrapings and eye discharge grew Gram-negative bacilli. Isolates were identified as *D. acidovorans* by Vitek 2 system (bioMérieux Inc., Durham, NC, USA). Because isolation of *D. acidovorans* from the eye is rare, we sent the isolate to the Infectious Disease Research Institute (IDRI) at the Asia Pacific Foundation for Infectious Diseases (APFID) for further testing. The 16S rRNA gene sequence (1276 bp) that was identified was compared using BLAST searches of the GenBank and EzTaxon servers (<http://www.ezbiocloud.net/eztaxon>). The sequence was 99.92% identical to that of *D. lacustris* (GenBank accession number EU888308). The second and the third closest matches were *D. tsuruhatensis* and *D. acidovorans* with 99.84% and 98.51% homology, respectively (accession numbers AB075017

and AF078774). Since our strain could utilize D-mannitol and D-malic acid (API 20 NE, bioMérieux Inc.) for growth, it was determined to be *D. lacustris*. The isolates were susceptible to aztreonam, cefepime, ceftazidime, piperacillin/tazobactam, and carbapenems, but resistant to aminoglycosides according to the Vitek system (bioMérieux Inc.) using 2011 Clinical Laboratory Standards Institute criteria for *Pseudomonas* (Table 1). The patient was initially treated with fortified topical ofloxacin, voriconazole, and gentamicin. Antibiotics were switched to topical ciprofloxacin and systemic ceftazidime after isolation of *Delftia*. However, the patient did not respond to therapy, and two months later underwent evisceration.

D. lacustris was first described in 2009 in freshwater in Denmark.³ *D. lacustris* and *D. tsuruhatensis* have 99.9% nucleotide similarity in the 16S rRNA gene sequence, as shown in this report. They can be differentiated based on the use of certain carbon sources for growth, such as D-mannitol and D-malic acid, as well as chitinase activity.³ In this study, the isolate was determined to be *D. lacustris* as D-mannitol and D-malate were utilized for growth in the API 20 NE system (bioMérieux). Shin et al. reported four possible human infections caused by *D. lacustris*. However, all of these were considered to be contaminants because only one bottle out of two sets of blood cultures grew *Delftia*, and some patients recovered without antibiotic therapy.⁴ Very recently, we described a true bloodstream infection by *D. lacustris*,² which was initially identified as *D. acidovorans* by Vitek 2 system, as in this report. All four *D. lacustris* infections reported by Shin et al. were also originally identified as *D. acidovorans* by Vitek 2.⁴ This indicates that infections due to *D. lacustris* may be more common than previously thought due to misidentification by commercial systems.

According to species-independent clinical breakpoints provided by the European Committee on Antimicrobial Susceptibility Testing (EUCAST, 2014), our isolate was susceptible to aztreonam, ceftazidime, cefotaxime, piperacillin-tazobactam, ticarcillin-clavulanate, and carbapenems, but resistant to all aminoglycosides tested and ciprofloxacin. *Delftia* is generally considered resistant to aminoglycosides.⁵ Ceftriaxone

Table 1 – Antimicrobial susceptibility profiles for *Delftia lacustris*.

| Antibiotics | MIC ($\mu\text{g/mL}$) | Susceptibility ^a |
|-----------------------------|--------------------------|-----------------------------|
| Amikacin | ≥ 64 | R |
| Aztreonam | 4 | S |
| Cefepime | 8 | S |
| Ceftazidime | ≤ 1 | S |
| Ciprofloxacin | 2 | I |
| Gentamicin | ≥ 16 | R |
| Imipenem | 1 | S |
| Meropenem | 0.5 | S |
| Piperacillin/tazobactam | ≤ 4 | S |
| Ticarcillin/clavulanic acid | ≤ 8 | S |

MIC, minimum inhibitory concentration; S, susceptible; R, resistant.

^a Because breakpoints for *Delftia* have not been established, this result refers to established criteria for *Pseudomonas* (2011 CLSI).

and cefotaxime may be effective for the treatment of *Delftia* infections because endocarditis caused by *D. acidovorans* has been successfully controlled using ceftriaxone alone.¹ Although the minimum inhibitory concentrations (MICs) of trimethoprim–sulfamethoxazole, minocycline, and tigecycline were very low, we do not know the clinical implications of this because there are no antibiotic susceptibility criteria or guidelines for *Delftia* species.

This is the first report of ocular infection with *D. lacustris* accompanied by significant complications. Because commercial systems can misidentify *Delftia* species, molecular methods such as 16S rRNA gene sequencing may be required. Further clinical investigation of *D. lacustris* is necessary to determine optimal therapy for this unusual pathogen.

Conflicts of interest

The authors declare no conflicts of interest.

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