Escherichia hermannii as the sole pathogen in urosepsis: case report

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A 43-year-old man presented with sudden onset fever and chills. His medical history included insulin-dependent diabetes mellitus, kidney transplant, complicated pancreas transplantation that which had required transplantectomy and an aneurysm of the artery iliaca communis that had required endovascular aneurysm repair.

Physical examination revealed signs of a slightly enlarged liver. Vital signs were a respiratory rate of 16 breaths per minute, saturation 100% (without O2), heart rate 120 beats per minute and blood pressure 118/65 mmHg. Laboratory testing showed C-reactive protein levels of 46 mg/L, haemoglobin 6.3 mmol/L, white blood cell count 9.3 × 10⁹/L with 75% neutrophils and blood platelets 236 × 10⁹/L. Urinalysis revealed a leukocyte esterase with >50 white blood count and 10 to 20 red blood cells per high-power field. His serum creatinine level was 205 μmol/L, and Modification of Diet in Renal Disease glomerular filtration rate 31 mL/min/1.73 m². Chest radiograph revealed nothing remarkable. A bacterial infection of the urinary tract was considered, and antibiotic therapy was initiated with oral cotrimoxazole (two divided doses of 480 mg, based on glomerular filtration rate).

The next day, blood and urine cultures yielded Escherichia hermannii (Fig. 1), identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (BioTyper; Bruker Daltonics, Bremen, Germany). We sequenced an 850 bp long fragment of the 16S rDNA gene, which revealed a 99% (849/850 bp) match with the E. hermannii CIP 103176 type strain (GenBank accession no. NR_104940.1). This strain was susceptible to amoxicillin/clavulanic acid and resistant to amoxicillin and ciprofloxacin in the Phoenix system (BD Bioscience, Erembodegem, Belgium), applying European Committee on Antimicrobial Susceptibility Testing clinical breakpoints. Cotrimoxazole was switched to oral amoxicillin/clavulanic acid (3 divided doses of 625 mg) for a total treatment duration of 2 weeks. Defervescence occurred in 48 hours, and the patient made a full, uneventful recovery while receiving treatment. No relapse occurred.

Urinary tract infections can be restricted to the bladder (cystitis) or can spread to the kidney (pyelonephritis) and even to the bloodstream (urosepsis). Escherichia coli is involved in >80% of cases [1]. The association of E. hermannii with urinary tract infections has rarely been reported. Tong et al. [2] described a patient with pyelonephritis but without bacteremia, where E. hermannii was the sole pathogen, indicating the uropathogenic potential of this species.

E. hermannii—a Gram-negative, rod-shaped bacterium—was first described in 1982 and is a member of the family Enterobacteriaceae. On the basis of phenotypic data and the DNA hybridization technique, E. hermannii forms a distinct species within the Escherichia genus; it produces yellow pigment and showed only 35% to 45% DNA relatedness to E. coli [3]. E. hermannii is mainly isolated from environmental sources and has been sporadically identified from wound, respiratory and stool specimens [3–5]. Much is still unclear about the pathogenicity of E. hermannii because in most cases it has been isolated with other coexisting bacteria that were more pathogenic. A possible mechanism for pathogenicity of E. hermannii is the feature of biofilm formation, which was suspected in one case of catheter-related sepsis in a haemodialysis patient [2,6–8]. Furthermore, E. hermannii is inherently resistant to penicillin, ampicillin and carbenicillin because of its β-lactamase production [9]. For antibiotic treatment, β-lactams and quinolones with in vivo susceptibility have been used [6].

In conclusion, the present case demonstrates that E. hermannii may cause urosepsis as a sole pathogen, which confirms the uropathogenic potential of E. hermannii.
Conflict of interest

None declared.

References