Escherichia hermannii as the sole pathogen in urosepsis: case report

R. A. Haasdijk and J. van Ingen
Department of Medical Microbiology, Radboudumc, Nijmegen, The Netherlands

Original Submission: 8 February 2017; Revised Submission: 5 November 2017; Accepted: 16 November 2017

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 63 mmol/L, white blood cell count 9.3 × 10^9/L with 75% neutrophils and blood platelets 236 × 10^11/L. Urinalysis 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 one week treatment). 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 vitro 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


FIG. 1. Microbiological culture of Escherichia hermannii. (A) Columbia III agar with 5% sheep’s blood (BD Bioscience). (B) MacConkey agar without salt (BD Bioscience). (C) Bankit2056671 Escherichia_hermannii_B16064920 MG256497.