A Rare Case of Urinary Tract Infection Caused by Enterococcus hirae in an Elderly Man with Benign Prostate Hyperplasia

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Enterococcus hirae (E. hirae) is a zoonotic pathogen, which accounts for 1-3% of enterococcal species isolated in clinical samples of humans.¹ Heretofore, there had been no report of urinary tract infection (UTI) with acute kidney injury (AKI) caused by E. hirae infection. Here however, we describe a patient with benign prostate hyperplasia (BPH) and duodenal ulcer who experienced acute pyelonephritis (APN) and acute kidney injury (AKI) due to E. hirae infection. To our knowledge, this is the first case report of AKI due to E. hirae in Korea.

A 71-year-old Korean man was admitted to our hospital for evaluation of fever (39.3°C) and abdominal pain. He had a 1-year history of BPH. Laboratory studies produced the following results: serum creatinine at 1.6 mg/dL, C-reactive protein at 19.6 mg/dL, fractional excretion of sodium at 1.3%, and prostate specific antigen level, 6.49 ng/mL (reference: 0-4 ng/mL). His previous serum creatinine concentration was 0.9 mg/dL. Urinalysis showed pyuria. Non-contrast enhanced computed tomography revealed urothelial thickening of the left renal pelvis. Cefotaxime was administered for a presumed diagnosis of APN. The urine cultures of Nelaton-catch urine obtained on admission showed 1 and 2 mm in diameter, smooth, cream or white colonies on 5% sheep blood agar after growth for 24 hours under the aerobic 5% CO2 incubation at 37°C (Fig. 1). The identification of the strain by MALDI-TOF (matrix-assissated laser desorption/ionization time-of-flight mass spectometry, VITEK® MS system V3.0) revealed the E. hirae, with confidence value of 99.9%.

Cefotaxime was substituted with ampicillin/sulbactam based on the results of the susceptibility testing (Table 1). Although inflammatory markers improved, he complained of epigastric pain. Therefore, he underwent gastroscopy, which revealed duodenal ulceration. After a more detailed epidemiological interview, it was found that the patient was a farmer who had contact with farm animals. The patient’s condition improved with the restoration of normal renal function, and he was discharged on day 16.

![Colonial morphology of Enterococcus hirae](image)

**FIG. 1.** Colonial morphology of Enterococcus hirae. Smooth, cream or white colonies were grown for 24 hours on 5% sheep blood agar plate in 5% CO2 incubation at 37°C. The strain was identified as Enterococcus hirae by MALDI-TOF (matrix-assissated laser desorption/ionization time-of-flight mass spectometry, VITEK® MS system V3.0).

| Antibiotics                              | Susceptibility | MIC (µg/mL) |
|------------------------------------------|----------------|-------------|
| Penicillin                               | S              | 0.25        |
| Ampicillin                               | S              | ≤2          |
| Gentamycin (high level resistance)      | SYN           | S           |
| Streptomycin (high level resistance)    | SYN           | ≤0.25       |
| Erythromycin                            | S              | 1           |
| Quinupristin/Dafopristin                | S              | 2           |
| Linezolid                                | S              | ≤0.5        |
| Teicoplanin                              | S              | ≤0.5        |
| Vancomycin                               | S              | ≤0.5        |

S: susceptible, MIC: minimum inhibitory concentration, SYN: synergy.

**TABLE 1.** Antimicrobial susceptibility of Enterococcus hirae isolated from urine

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APN usually develops secondary to an ascending infection of gram-negative bacteria in women and is relatively uncommon in men. However, the prostate gland is considered a major culprit in the development of urinary tract infection in men due to urethral obstruction and incomplete voiding.² Bourafa et al.³ reported a case of UTI caused by *E. hirae* in a male farmer with BPH, which was also observed in our case. Therefore, we suggest that men with BPH who frequently handle animals should be cautious of APN caused by *E. hirae*.

Although several cases of UTI in humans caused by *E. hirae* have been reported,⁴ AKI did not develop in those cases. In this case, the patient's fractional sodium excretion was 1.3% and renal function normalized 10 days after treatment, which was suggestive of intrinsic AKI. Further, the patient had not eaten for several days due to epigastric pain, which was associated with the duodenal ulcer. Old age is regarded as a risk factor for AKI due to impairment of the vasodilatory response in the afferent arteriole,⁵ therefore, we also believe that old age might also contribute the development of AKI in the present case. Thus, various mechanisms might have contributed to the development of AKI. In summary, we report a case of APN associated with AKI caused by *E. hirae* in a patient with BPH, in which the AKI was successfully treated via appropriate antibiotic therapy and supportive care.

**CONFLICT OF INTEREST STATEMENT**

None declared.