

## Case Report

# ***Enterococcus hirae* as a cause of bacteremic urinary tract infection: first case report from Turkey**

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### Abstract

**Introduction:** *Enterococcus hirae* (*E. hirae*) constitutes less than 1% of the enterococci strains in human clinical specimens. In this article, we report the first case of urinary tract infection-related bacteremia due to *E. hirae* from Turkey.

**Case Presentation:** A 74-year-old male patient with a history of coronary artery disease, hypertension, and chronic renal failure was admitted to the emergency department with abdominal pain, dysuria, and fever. The urine sample collected from the urinary catheter resulted as ampicillin-sensitive *E. hirae*. On the 4th day of hospitalization, *E. hirae* growth with the same sensitivity pattern was also reported in blood culture. Intravenous ampicillin 4×2 g/day treatment was initiated. There was no growth in subsequent blood and urine cultures. Fever resolved and general condition improved. The patient was discharged on the thirteenth day with clinical improvement after moxifloxacin treatment for four days and ampicillin treatment for nine days.

**Discussion:** The patient's medical history included risk factors for enterococcal bacteremia. There are a limited number of reports in the literature describing human infections caused by *E. hirae*. The reason for the rare isolation of *E. hirae* from clinical specimens may be the difficulty of identifying with standard diagnostic approaches.

**Conclusions:** For diagnostic purposes, as in our case, rapid and high sensitive diagnostic methods such as Matrix-assisted Laser Desorption/Ionization Time of Flight (MALDI-TOF) and molecular techniques may be useful to guide the selection of the least toxic and optimal duration of antibiotic treatment.

**Keywords:** Bacteremia; *Enterococcus hirae*; urinary tract infections.

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### Introduction

Enterococci are part of the human intestinal flora and can be found in small amounts on the skin, oropharyngeal and vaginal secretions. These bacteria can cause a wide variety of infections, including urinary tract, intra-abdominal infections, endocarditis, and bacteremia. In the past two decades, enterococcus species have also been reported as opportunistic pathogens in the clinic [1]. Enterococci show partial or complete resistance to many antibiotics used in the treatment of Gram-positive bacterial infections. Since the antibiotic choices are limited for enterococcal infections, the correct identification of the agents and determination of their sensitivity in the treatment of infections is very important [2]. *Enterococcus faecalis* (80%) and *Enterococcus faecium* (10%) are the most common strains isolated from human clinical specimens. Other enterococcus species are rarely isolated and *Enterococcus hirae* (*E. hirae*) accounts for

less than 1% of strains in human clinical specimens [3]. In this article, we report the first case of urinary tract infection-related bacteremia due to *E. hirae* from Turkey.

### Case Presentation

A 74-year-old male patient with a history of coronary artery disease, hypertension, and chronic renal failure was admitted to the emergency department with abdominal pain, dysuria, and fever. On admission, he had a temperature of 38.2°C, his blood pressure was 100/80 mmHg, and heart rate was 100 beats per minute. In the neurological and respiratory system examinations, no pathological findings were detected, and there was no feature other than an increase in the heart rate in the cardiovascular system examination. The patient, who described mild pain in the epigastric region, had no sensitivity in the costovertebral and suprapubic regions. First laboratory results in the

emergency department revealed: White blood cell count (WBC):  $28.6 \times 10^3/\mu\text{L}$  (85% neutrophil), hemoglobin (HB): 12.8 g/dL, platelet (PLT):  $278 \times 10^3/\mu\text{L}$ , creatinine: 2.7 mg/dL, urine microscopy and stained examination findings: 32 erythrocytes / $\mu\text{L}$ , 48 leukocytes / $\mu\text{L}$  and Gram-positive cocci were observed. The patient's blood and urine cultures were obtained. Blood cultures were incubated on a fully automated BacT/ALERT 3D® (bioMérieux, France) system. To investigate the etiology of fever, abdominal and thoracic computed tomography (CT) scans were performed and the patient was hospitalized in the infectious diseases service. During hospitalization, a urinary catheter was inserted to monitor urine output, and also a urine sample was taken from the catheter for urine microscopy and culture examinations. Unlike urine microscopy results in the emergency department, there was no erythrocyte and leukocyte in the microscopic examination of urine taken from the urinary catheter. On abdominal CT, there were no pathological findings in the liver, biliary tract, pancreas, and spleen, the right kidney was measured as 130 mm, left kidney as 93 mm, and pelvic structures and ureters were of normal width. Thoracic CT was reported as suspicious alveolar density increase which may indicate pneumonia. Although the clinical and radiological findings related to pneumonia were not typical, empirical moxifloxacin 400 mg/day was initiated because no alternative focus could be detected. The laboratory examinations performed one day after the start of treatment revealed; WBC:  $22.6 \times 10^3/\mu\text{L}$  (87.3% neutrophils), C-reactive protein (CRP): 356 mg/L, creatinine: 3.02 mg/dL, procalcitonin (PCT): 3.45 ng/mL. Urine culture results from the emergency department were reported as contamination at 48th hour, but the urine sample collected from the urinary

catheter resulted as 10,000 colony-forming units (CFU) of ampicillin-sensitive *E. hirae*. The reported agent was not considered as the etiological agent and current treatment was continued. In the follow-up of the patient, the fever continued to occur less frequently. On the 4th day of hospitalization, *E. hirae* growth with the same sensitivity pattern was also reported in blood culture. Antibiotic resistance analysis and identification were performed with automated VITEK® 2 Systems 8.01 (bioMérieux, Inc., Marcy l'Etoile, France). Identification of *E. hirae* is also verified with automated Matrix-Assisted Laser Desorption Ionization- Time of Flight (MALDI-TOF) Mass Spectrometry system (Vitek MS® systems, bioMérieux, Inc., Marcy l'Etoile, France) and the polymerase chain reaction (PCR) technique using specific primers (HiF TTA TGT CCC AGT ATT GAA AAA TCA A and HiR TTT TGT TAG ACC TCT TCC GGA). Considering the bacteremia originated from the urinary system, moxifloxacin was discontinued and intravenous ampicillin 4x2 g/day treatment was initiated. There was no growth in subsequent blood and urine cultures. Fever resolved and general condition improved. In the last laboratory examinations of the patient before discharge were; WBC:  $10.5 \times 10^3/\mu\text{L}$  (75% neutrophils), creatinine: 2.03 mg/dL, CRP: 31 mg/L, PCT: 0.12 ng/mL. The patient was discharged on the thirteenth day with clinical improvement after moxifloxacin treatment for four days and ampicillin treatment for nine days.

## Discussion

Male gender, advanced age, liver disease, kidney failure, diabetes, hematological transplantation, malignancy, and previous antibiotic treatments have been identified as risk factors for enterococcal bacteremia [4]. The medical history of a 74-year-old

**Table 1.** *Enterococcus hirae* case reports in the literature.

Year	Reference	Sample	Age/gender	Diagnosis
1998	Gilad J <i>et al.</i> [6]	Blood	48/M	Septicemia
2000	Park J <i>et al.</i> [7]	Blood, urine	21/F	Acute pyelonephritis
2002	Poyart C <i>et al.</i> [8]	Blood	72/M	Native valve endocarditis
2008	Canalejo E <i>et al.</i> [9]	Blood	55/M	Spondylodiscitis
2009	Kim HI <i>et al.</i> [10]	Blood, urine	57/F	Acute pyelonephritis
2011	Talarmin JP <i>et al.</i> [11]	Blood	78/F	Infective endocarditis
2012	Chan TS <i>et al.</i> [12]	Blood, urine	62/F	Acute pyelonephritis
2012	Chan TS <i>et al.</i> [12]	Blood	83/F	Acute cholangitis
2012	Sim JS <i>et al.</i> [13]	Blood, acid fluid	61/M	Bacterial peritonitis
2013	Anghinah R <i>et al.</i> [14]	Blood	56/F	Infective endocarditis
2014	Alfouzan W <i>et al.</i> [15]	Blood, pus	48/M	Spleen abscess
2015	Bourafa N <i>et al.</i> [16]	Urine	50/M	Urinary tract infection
2016	Pãosinho A <i>et al.</i> [17]	Blood, urine	78/F	Acute pyelonephritis
2017	Lee GH <i>et al.</i> [18]	Blood, urine	78 M/74 F	Acute pyelonephritis

M: male; F: female.

male patient with a history of coronary artery disease, hypertension, and chronic renal failure also included more than one of these risk factors. There are a limited number of reports in the literature reporting human infections caused by *E. hirae* (Table 1).

This is due to the difficulties in the diagnosis of *E. hirae*, because it is difficult to define and may be misdiagnosed with the standard diagnostic approach [5].

## Conclusions

For accurate identification of rare infection agents like *E. hirae*, fast and highly sensitive methods such as MALDI-TOF or molecular diagnostic techniques like PCR, may guide the selection of the least toxic antibiotics and optimal treatment duration.

## References

- Kayser FH (2003) Safety aspects of enterococci from the medical point of view. *Int J Food Microbiol* 88: 255–262.
- Conceição N, da Cunha Hueb Barata de Oliveira C, Silva PR, Avila BG, de Oliveira AG (2011) Trends in antimicrobial resistance among clinical isolates of enterococci in a Brazilian tertiary hospital: a 4-year study. *Rev Soc Bras Med Trop* 44: 177–181.
- Vandamme P, Vercauteren E, Lammens C, Pensart N, Ieven M, Pot B, Leclercq R, Goossens H (1996) Survey of enterococcal susceptibility patterns in Belgium. *J Clin Microbiol* 34: 2572–2576.
- Billington EO, Phang SH, Gregson DB, Pitout JD, Ross T, Church DL, Laupland KB, Parkins MD (2014) Incidence, risk factors, and outcomes for *Enterococcus* spp. blood stream infections: A population-based study. *Int J Infect Dis* 26: 76–82.
- Benagli C, Rossi V, Dolina M, Tonolla M, Petrini O (2011) Matrix-assisted laser desorption ionization-time of flight mass spectrometry for the identification of clinically relevant bacteria. *PLoS One* 6: e16424.
- Gilad J, Borer A, Riesenberk K, Peled N, Shnaider A, Schlaeffer F (1998) *Enterococcus hirae* septicemia in a patient with end-stage renal disease undergoing hemodialysis. *Eur J Clin Microbiol Infect Dis* 17: 576e7.
- Park J, Uh Y, Jang IH, Yoon KJ, Kim SJ (2000) A case of *Enterococcus hirae* septicemia in a patient with acute pyelonephritis. *Korean J Clin Pathol* 20: 501–503.
- Poyart C, Lambert T, Morand P, Abassade P, Quesne G, Baudouy Y, Trieu-Cuot P (2002) Native valve endocarditis due to *Enterococcus hirae*. *J Clin Microbiol* 40: 2689–90.
- Canalejo E, Ballesteros R, Cabezudo J, Garcia-Arata MI, Moreno J (2008) Bacteraemic spondylodiscitis caused by *Enterococcus hirae*. *Eur J Clin Microbiol Infect Dis* 27: 613–615.
- Kim HI, Lim DS, Seo JY, Choi SH (2009) A case of pyelonephritis accompanied by *Enterococcus hirae* bacteremia. *Infect Chemother* 41: 359–361.
- Talarmin JP, Pineau S, Guillouzoic A, Boutoille D, Giraudeau C, Reynaud A, Lepelletier D, Corvec S (2011) Relapse of *Enterococcus hirae* prosthetic valve endocarditis. *J Clin Microbiol* 49: 1182–1184.
- Chan TS, Wu MS, Suk FM, Chen CN, Chen YF, Hou YH, Lien GS (2012) *Enterococcus hirae* related acute pyelonephritis and cholangitis with bacteremia: An unusual infection in humans. *Kaohsiung J Med Sci* 28: 111–114.
- Sim JS, Kim HS, Oh KJ, Park MS, Jung EJ, Jung YJ, Kang DG, Seo SI, Kim WJ, Jang MK (2012) Spontaneous bacterial peritonitis with sepsis caused by *Enterococcus hirae*. *J Korean Med Sci* 27: 1598–1600.
- Anghinah R, Watanabe RG, Simabukuro MM, Guariglia C, Pinto LF, Gonçalves DC (2013) Native valve endocarditis due to *Enterococcus hirae* presenting as a neurological deficit. *Case Rep Neurol Med* 2013: 636070..
- Alfouzian W, Al-Sheridah S, Al-Jabban A, Dhar R, Al-Mutairi A, Udo E (2014) A case of multiple splenic abscesses due to *Enterococcus hirae*. *JMM Case Reports* 1; 1–4.
- Bourafa N, Loucif L, Boutefnouchet N, Rolain JM (2015) *Enterococcus hirae*, an unusual pathogen in humans causing urinary tract infection in a patient with benign prostatic hyperplasia: first case report in Algeria. *New Microbes New Infect* 17: 7–9.
- Pãozinho A, Azevedo T, Alves JV, Costa IA, Carvalho G, Peres SR, Baptista T, Borges F, Mansinho K (2016) Acute pyelonephritis with bacteremia caused by *Enterococcus hirae*: A rare infection in humans. *Case Rep Infect Dis* 2016: 4698462.
- Lee GH, Lee HW, Lee YJ, Park BS, Kim YW, Park S (2017) Acute pyelonephritis with *Enterococcus hirae* and literature review. *Urogenit Tract Infect* 12: 49–53.

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