

Urinary tract infection caused by *Enterococcus gallinarum* in a 65-year-old female patient: Case report and review of literature

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ABSTRACT

Urinary tract infection (UTI) is very common in adult females. *Enterococcus* spp. is an uncommon cause of UTI in females. They are sometimes resistant to vancomycin and make treatment difficult and prolonged. *Enterococcus gallinarum* is exceedingly rare as a uropathogen and can often be missed if not meticulously looked for. We here report a case of recurrent UTI due to *E. gallinarum* in a female diabetic patient aged 65 years, with confirmation of the isolate done by conventional as well as automated methods.

Key words: Adult patient, Diabetic, *Enterococcus gallinarum*, Urinary tract infection

Urinary tract infections (UTIs) are among the most common bacterial infections, affecting millions of people annually. While *Escherichia coli* is the predominant uropathogen, other bacteria, including *Enterococcus* species, are increasingly recognized as causative agents, particularly in health-care-associated infections, in male subjects, or individuals with underlying comorbidities. *Enterococcus gallinarum* is a relatively uncommon species within the *Enterococcus* genus, often associated with vancomycin resistance and previously considered of less clinical significance [1]. However, its emergence as a pathogen in various infections, including UTIs, warrants attention. *E. gallinarum* shows significant pathogenicity in immunocompromised patients. It is reported from UTIs and bacteremia [2]. *E. gallinarum* is an opportunistic pathogen and has also rarely been reported as a predominant pathogen responsible for biliary tract infections in man [3].

This case report describes a UTI in a 65-year-old female outpatient caused by *E. gallinarum*, highlighting the importance of accurate microbiological identification and appropriate management of such infections. *E. gallinarum* is an uncommon cause of UTI in adult female diabetic subjects, and hence, this case deserves special mention.

CASE REPORT

A 65-year-old female patient, resident of South Kolkata, India, presented to the outpatient department with a 3-month history of off-and-on and progressively worsening urinary symptoms such as lower abdominal pain and dysuria. She was a divorcee and had been living all alone. There was no history of fever. Her chief complaints included dysuria (burning sensation during urination), increased urinary frequency (urination every 1–2 h during daytime), and nocturia (waking up many times at night to urinate). She had off-and-on moderate-to-severe burning pain in the lower abdomen, which was aggravated while urinating and got relieved or diminished after micturition. There were no features such as gross hematuria, fever, chills, flank pain, or suprapubic tenderness. Her medical history was significant for recently developed diabetes mellitus, osteoarthritis of the right knee joint, and a history of recurrent UTIs, though the causative organisms of previous infections were not readily available in her records. She was non-hypertensive. On two occasions, she collected urine in an improper manner, which yielded gross contamination on culture, and on the 3rd time, the collection was proper and colonies grew. She had no known allergies to medications. The patient reported no recent hospitalizations, indwelling catheters, or instrumental procedures of the urinary

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tract. She lived independently and had no apparent cognitive impairment.

Upon physical examination, the patient appeared comfortable and afebrile with a temperature of 36.8°C. Abdominal examination revealed no tenderness, masses, or organomegaly.

Given her presenting symptoms, a presumptive diagnosis of UTI was made. A clean-catch midstream urine sample was collected for routine urinalysis and urine culture. The urine sample was yellow and of cloudy consistency. Microscopic examination revealed white blood cells: 1–2 per high-power field, indicating significant pyuria. Red blood cells were not found. Bacteria were numerous, consistent with bacteriuria. No casts were observed. The urine sample was inoculated onto appropriate culture medium (cystine-lactose-electrolyte-deficient [CLED] with Bromothymol blue) and incubated aerobically at 37°C. After 24 h, the culture revealed a significant growth ($>10^5$ colony-forming units/mL) of opaque, small, yellowish-white colonies on CLED. On Gram stains, these microorganisms appeared as oval Gram-positive cocci arranged in pairs and short chains, characteristic of *Enterococcus* species. It was indole negative, non-motile, did not break down mannitol, and grew in the presence of 6.5% NaCl (Fig. 1). Growth was very slow and scanty on Muller–Hinton Agar (MHA) and poor at 40°C. Confirmation of the isolate was carried out using the VITEK-2 compact automated microbiology system (bioMérieux, Marcy l’Etoile, France). The VITEK-2 identified the isolate as *E. gallinarum* with a probability of 94% (Fig. 2). This automated system used a panel of biochemical tests to provide rapid and accurate species identification. D-Ribose and D-amydalin were fermented, and Leucine arylamidase and Alanine arylamidase tests were positive. Antimicrobial susceptibility testing could not be done since the isolate did not grow on MHA, but the patient was advised treatment with ciprofloxacin.

Based on the diagnosis of UTI caused by susceptible *E. gallinarum*, the patient was initially prescribed oral ciprofloxacin 250 mg twice daily for 7 days, considering its excellent urinary penetration and her history of

recurrent UTI. She was advised to drink plenty of water. However, the patient was lost to follow-up but was traced again later.

Presently, she is having no symptoms such as dysuria and is maintaining adequate hydration. She also took a 5-day course of oral cefixime a month back. The patient reported significant improvement in her symptoms, with resolution of dysuria and a marked decrease in urinary frequency. A follow-up urine culture was not performed given the patient’s symptomatic improvement, resolution of inflammatory markers, and also unwillingness on the part of the patient.

DISCUSSION

This case highlights UTI caused by *E. gallinarum* in an outpatient setting, a pathogen less frequently reported in community-acquired UTIs compared to other *Enterococcus* species such as *Enterococcus faecalis* or *Enterococcus faecium*. *E. gallinarum* is a member of the *Enterococcus* genus, which possesses intrinsic low-level resistance to vancomycin due to the presence of the *vanC* gene [4]. As an opportunistic pathogen, *E. gallinarum* mainly leads to nosocomial infections, and its multidrug resistance has gained more and more attention. It has been reported in various infections. Central nervous system infections caused by *E. gallinarum* are rare, but have been reported more often in recent years [5]. A study found an association between *E. gallinarum* gut translocation and autoantibody production, as evident in inflammatory bowel disease [6].

In recent times, with the increasing usage of broad-spectrum antibiotics and invasive medical devices, infections caused by *E. gallinarum* have gradually increased, and multidrug resistance has gained more and more attention. In 2010, among the isolated strains of *Enterococcus* in several Chinese hospitals, *E. gallinarum* accounted for 1.9% of isolates, and was second only to *E. faecalis* and *E. faecium* [7]. This underscores the importance of phenotypic susceptibility testing, as genotypic presence of *vanC* does not always translate to clinical vancomycin resistance, especially at standard therapeutic doses. While often considered commensal or of low virulence, *E. gallinarum* has been implicated in various infections, including bacteremia, endocarditis, and UTIs, particularly in immunocompromised individuals or those with underlying urinary tract abnormalities.

E. gallinarum and *Enterococcus casseliflavus/flavescens* are enterococci intrinsically resistant to vancomycin, belonging to the *E. gallinarum* group. They are responsible mainly for health-care-associated infections, in particular bloodstream, urinary tract, and surgical wound infections. *E. gallinarum* is typically seen in the gastrointestinal tracts of birds and mammals. Although it is rarely isolated from clinical specimens, *E. gallinarum* can lead to septicemia in immunocompromised individuals [8]. Diseases due to these bacteria are significantly increasing

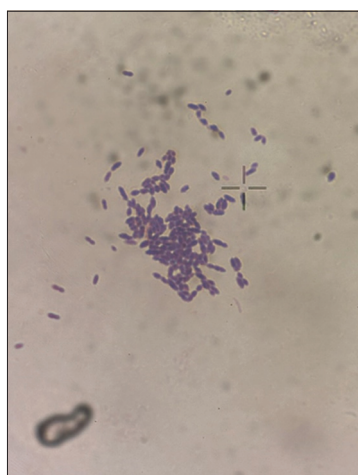


Figure 1: Gram stain showing oval Gram-positive cocci in pairs and short chains

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Patient Name: ISOLATE, BECT. Patient ID: VT9676
 Location: Physician:
 Lab ID: VT9676 Isolate Number: 1

Organism Quantity:
 Selected Organism: *Enterococcus gallinarum*

Source: OTHERS Collected:

Comments:

Identification Information Analysis Time: 4.82 hours Status: Final
 Selected Organism 94% Probability *Enterococcus gallinarum*
 Bionumber: 516002765777731

ID Analysis Messages

Biochemical Details																	
2	AMY	+	4	PIPLC	-	5	dXYL	+	8	ADHI	+	9	BGAL	-	11	AGLU	-
13	APPA	-	14	CDEX	+	15	AspA	+	16	BGAR	-	17	AMAN	-	19	PHOS	-
20	LeuA	-	23	ProA	-	24	BGURr	-	25	AGAL	-	26	PyrA	+	27	BGUR	-
28	AlaA	+	29	TyrA	+	30	dSOR	+	31	URE	-	32	POLYB	+	37	dGAL	+
38	dRIB	+	39	ILATr	-	42	LAC	+	44	NAG	+	45	dMAL	+	46	BACI	+
47	NOVO	+	50	NC6.5	+	52	dMAN	+	53	dMNE	+	54	MB6G	+	56	PUL	+
57	dRAF	+	58	O129R	+	59	SAL	+	60	SAC	+	62	dTRE	+	63	ADH2s	-
64	OPTO	+															

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Figure 2: VITEK-2 identification report

worldwide, since they tend to cause infection in patients with concurrent hepatobiliary or oncohematological disorders [9]. In this patient, the absence of obvious risk factors for severe infection or health-care association makes this presentation noteworthy, except for recently developed diabetes mellitus. Furthermore, sometimes this bacterium grows very slowly, and this might explain its poor growth on MHA. *E. gallinarum*'s ability to acquire antibiotic resistance is linked to its genetic makeup and the strategies it follows to adapt to environmental pressure. One of the primary processes this bacterium enhances its resistance is through horizontal gene transfer, a process that allows the exchange of genetic material between different bacterial species. This exchange can take place via transformation, transduction, or conjugation, each providing a unique pathway for genetic material to be shared and integrated [10].

The rapid and accurate identification of *E. gallinarum* by the VITEK-2 system was crucial for guiding appropriate

antimicrobial therapy. Traditional biochemical methods can sometimes misidentify *Enterococcus* species, leading to suboptimal treatment. Automated systems such as VITEK-2 offer a more precise and timely diagnosis, which is particularly important for less common or resistant pathogens. The susceptibility profile of *E. gallinarum* to ciprofloxacin, ampicillin, vancomycin (sometimes), nitrofurantoin, and linezolid provided several viable treatment options. Ciprofloxacin was chosen due to its broad spectrum against common uropathogens and good urinary excretion, which aligns with empirical treatment guidelines for complicated UTIs (though this case was initially managed as an uncomplicated UTI, the history of recurrence warranted a broader coverage initially). The resistance to trimethoprim-sulfamethoxazole is also notable, as this is a common first-line agent for UTIs, stressing the need for culture-guided therapy.

The patient's history of recurrent UTIs warrants further investigation to identify potential

underlying causes, such as anatomical abnormalities, postmenopausal changes, or other risk factors. Strategies to prevent future recurrences may include behavioral modifications, vaginal estrogen therapy (if applicable), or long-term low-dose prophylactic antibiotics, depending on the identification of specific risk factors. The patient responding well to cefixime is a bit unnatural. It may be possible that she may have consumed other antibiotics too, but not informing. It may be that the infection may recur again, due to possible suboptimal or inappropriate antibiotic intake. Goel *et al.* in 2016 have reported *E. gallinarum* to cause only about 1.7% of all community-acquired UTIs in North India [11]. As far as we know, this is the first report of isolation of this pathogen from a diabetic old female patient, especially from this part of the country, and warrants the need for further research into the pathogenic potential of this pathogen.

CONCLUSION

This case report describes a community-acquired UTI in a 63-year-old female caused by *E. gallinarum*, successfully treated with oral cefixime. This is rare given the known low susceptibility profile of enterococci to cephalosporins. The identification of this less common *Enterococcus* species highlights the evolving epidemiology of UTIs and the importance of accurate microbiological diagnostics, such as the VITEK-2 system, for effective patient management. As the prevalence of antimicrobial resistance continues to rise, thorough identification and susceptibility testing are vital to ensure appropriate antimicrobial stewardship and optimize patient outcomes, even in routine outpatient presentations. VITEK-2 is also a viable option for accurate identification in such cases. Further research is needed to better understand the clinical significance and epidemiological patterns of *E. gallinarum* in various infection settings.

AUTHOR STATEMENT

Dr. Sayan Bhattacharyya collected data and prepared the article, while Ashima Nayek helped trace the case.

Others helped in drafting the manuscript and providing clinical details.

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