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CASE REPORT

Relapsing Enterococcus Faecalis Endocarditis in a Patient with Severe Aortic Stenosis and Heart Failure: Clinical Course and Management

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Abstract

Background: Infective endocarditis caused by Enterococcus faecalis remains a challenging clinical entity, particularly in patients with pre-existing valvular heart disease and reduced left ventricular ejection fraction. Relapse after apparently adequate antimicrobial therapy poses significant diagnostic and therapeutic dilemmas.

Case Summary: A 51-year-old male with severe aortic valve stenosis and regurgitation, atrial fibrillation, and heart failure with reduced ejection fraction (LVEF 25%) presented with dyspnoea and night sweats. Following coronary angiography, the patient developed sepsis. Blood cultures revealed Enterococcus faecalis, and echocardiography demonstrated an aortic root abscess. Initial treatment with vancomycin and ceftriaxone was modified to teicoplanin due to nephrotoxicity. After six weeks of therapy, clinical improvement and recovery of LVEF to 45% were observed. Two months later, the patient experienced relapse with recurrent E. faecalis bacteraemia and worsening renal function. Given the life-threatening septic state, vancomycin-based therapy was reintroduced despite nephrotoxicity. After successful infection control, transcatheter aortic valve implantation (TAVI) was performed with favourable outcome.

Discussion: This case highlights the risk of relapsing enterococcal endocarditis after invasive procedures in patients with structural heart disease. It underscores the importance of prolonged combination antimicrobial therapy, careful renal monitoring, and timely valve intervention after infection eradication.

Keywords: Infective endocarditis; Enterococcus faecalis; Aortic valve disease; Relapse; Vancomisin, TAVI

Introduction

Infective endocarditis (IE) remains associated with high morbidity and mortality, particularly in patients with underlying valvular heart disease and heart failure. Enterococcus faecalis is increasingly recognized as a cause of healthcare-associated IE, often related to invasive diagnostic or therapeutic procedures. Relapsing infection presents a significant challenge, necessitating individualized multidisciplinary management.

Case Presentation

A 51-year-old male presented to the outpatient clinic of the Scientific Research Institute of Cardiology on 10 June 2025 with dyspnoea and night sweats. The left ventricle demonstrates dilatation in both systolic and diastolic dimensions with reduced systolic function (ejection fraction approximately 25%). Left ventricular myocardial hypertrophy is present. Diastolic function is impaired, consistent with a relaxation-type diastolic dysfunction. The left atrium is enlarged. The right cardiac chambers are not dilated. The aortic valve shows fibrotic changes with reduced leaflet mobility. Moderate aortic stenosis is noted. Segmental wall motion abnormalities are observed, characterized by diffuse hypokinesia. A pericardial effusion is detected. Laboratory testing showed elevated high-sensitivity troponin (90 ng/mL) and NT-proBNP (2762 pg/mL).

The patient was admitted for further evaluation and underwent coronary angiography, which demonstrated three-vessel coronary anatomy without acute occlusion.

On 26 July 2025, the patient re-presented with high-grade fever (38.8 °C), chills, and worsening dyspnoea. Laboratory tests showed markedly elevated inflammatory markers (CRP 47.96 mg/L, ESR 106 mm/h, procalcitonin 12 ng/mL) (fig 1A,B). Blood cultures grew Enterococcus faecalis. Transesophageal echocardiography was performed with the patient in the left lateral decubitus position. Examination revealed a dilated left ventricle with a globally reduced ejection fraction of 30–35%, while right heart chamber dimensions were within normal limits. The interatrial septum was intact, and the left atrial appendage was free of thrombus. Notably, a mobile vegetative mass was visualized on the aortic valve. Additionally, at the level of the aortic sinotubular junction, a 15 × 20 mm irregular, hyperechogenic mass consistent with an aortic abscess was identified, confirming structural complications associated with severe aortic stenosis.

Initial antimicrobial therapy with vancomycin and ceftriaxone was initiated. Due to rising serum creatinine (1.6 mg/dL)(fig 1D), therapy was switched to teicoplanin plus ceftriaxone for six weeks, resulting in clinical improvement and recovery of LVEF to 45%(fig 1E).

Two months later, the patient developed recurrent fever, fatigue, and dyspnoea. Blood cultures again yielded E. faecalis, confirming relapsing infective endocarditis. Despite worsening renal function (creatinine 2.67 mg/dL), vancomycin-based therapy was reintroduced due to the lifethreatening septic condition. Transthoracic and transesophageal echocardiography revealed a markedly dilated left ventricle with severely reduced systolic function. The left ventricular ejection fraction (LVEF) was approximately 25%, consistent with severe systolic dysfunction, and findings were compatible with restrictive-type diastolic dysfunction. Spontaneous echo contrast was observed within the left ventricular cavity, and the left atrium was enlarged (LA diameter ≈ 45 mm). Right-sided cardiac chambers were within normal limits, and both the interatrial and interventricular septa were intact and of normal thickness. Severe global hypokinesis of the left ventricular myocardium was noted.The aortic valve cusps were thickened and calcified. A 14 × 7 mm mass was visualized on the non-coronary cusp, suspicious for vegetation. Aortic valve opening was restricted, consistent with a low-flow, low-gradient aortic stenosis pattern (peak gradient 36.2 mmHg, mean gradient 19.6 mmHg). Mild eccentric aortic regurgitation was present. The mitral valve leaflets were fibrotic, with trace to mild regurgitation, and mild tricuspid regurgitation was also observed; pulmonary regurgitation was minimal.The ascending aorta, aortic arch, and pulmonary artery were within normal limits, with normal pulmonary artery systolic pressure. No evidence of aortic coarctation was detected, and the pericardial space was free of effusion.

After six weeks of treatment (December 2025), inflammatory markers normalized, and transcatheter aortic valve implantation(TAVİ) was successfully performed.The patient remains clinically stable on follow-up.

Discussion

Relapsing Enterococcus faecalis infective endocarditis represents a well-recognized clinical challenge, particularly in patients with advanced structural heart disease and perivalvular complications. In the present case, despite six weeks of guideline-concordant combination therapy with teicoplanin and ceftriaxone, complete eradication of infection was not achieved, resulting in relapse two months after apparent clinical recovery [1].

Several pathophysiological and pharmacological factors likely contributed to treatment failure. Enterococcus faecalis is known for its ability to form biofilms on damaged valvular tissue, significantly reducing antibiotic penetration and rendering bacteria metabolically less active. Glycopeptide antibiotics, including teicoplanin, demonstrate limited bactericidal activity against enterococci within biofilm environments, exerting predominantly bacteriostatic effects.

Consequently, bacterial suppression rather than definitive eradication may occur, predisposing to late relapse [2].

Furthermore, the presence of an aortic root abscess constituted a critical determinant of antimicrobial failure. Perivalvular abscesses are characterized by impaired local perfusion, resulting in subtherapeutic antibiotic concentrations at the site of infection [3]. In such settings, medical therapy alone is often insufficient, and delayed surgical or transcatheter intervention following infection control is required to achieve durable cure [4].

According to current ESC and IDSA guidelines, the preferred first-line regimen for Enterococcus faecalis infective endocarditis is a β-lactam–based combination, most commonly ampicillin plus ceftriaxone, due to its proven bactericidal synergy and favorable safety profile. However, in the present case, the use of β-lactam antibiotics was contraindicated because of a documented β-lactam allergy, precluding the administration of ampicillin-containing regimens. Consequently, alternative glycopeptide-based therapy was required [1].

In patients with β-lactam intolerance, vancomycin-based regimens are recommended as guidelinesupported alternatives, particularly in the setting of severe sepsis. Teicoplanin may be considered in selected cases due to its improved tolerability and lower nephrotoxicity; however, it is not regarded as a first-line agent in high-inoculum

infections, such as infective endocarditis complicated by perivalvular abscess formation. Its relatively limited bactericidal activity and suboptimal penetration into abscess cavities and biofilm structures may contribute to incomplete microbial eradication.

In our patient, initial treatment with teicoplanin combined with ceftriaxone resulted in temporary clinical and echocardiographic improvement, including partial recovery of left ventricular systolic function. Nevertheless, the subsequent relapse of E. faecalis bacteremia suggests persistent infection, likely related to the presence of an aortic root abscess and the intrinsic limitations of teicoplanin in achieving sustained sterilization in this context. During the relapse, given the lifethreatening septic state, vancomycin-based therapy was prioritized despite the risk of nephrotoxicity, aligning with guideline recommendations that emphasize survival over renal safety in critical infections.

Pharmacokinetic considerations also merit attention. Effective treatment of enterococcal endocarditis with teicoplanin requires sustained high serum trough concentrations, typically ≥20– 25 mg/L [5]. In routine clinical practice, therapeutic drug monitoring of teicoplanin is not universally performed, increasing the risk of underdosing and inadequate tissue exposure. In contrast, vancomycin therapy benefits from standardized monitoring strategies and more predictable bactericidal activity against enterococci.

Importantly, host-related factors further increased the risk of relapse in this patient. Severe left ventricular systolic dysfunction, atrial fibrillation, advanced aortic valve disease, and recent invasive coronary angiography collectively created a high-risk substrate for persistent infection. The recurrence of E. faecalis bacteremia with an identical clinical phenotype strongly supports relapse due to persistent intracardiac infection rather than reinfection.

Taken together, this case illustrates that while teicoplanin-based therapy may achieve temporary clinical stabilization in enterococcal infective endocarditis, it may be insufficient for definitive eradication in the presence of biofilm formation and perivalvular abscess. In life-threatening septic relapse, prioritization of bactericidal efficacy over nephrotoxicity is justified, and aggressive antimicrobial therapy combined with timely valve intervention is essential for favorable outcomes.

Conclusion

This case illustrates the complexity of managing relapsing Enterococcus faecalis infective endocarditis in patients with advanced valvular heart disease. Aggressive antimicrobial therapy, even in the presence of nephrotoxicity, and appropriately timed transcatheter valve intervention can result in successful outcomes. These findings are consistent with current ESC guidelines and prior studies demonstrating that enterococcal infective endocarditis complicated by perivalvular abscess and biofilm formation often requires highly bactericidal regimens and timely valve intervention to prevent relapse.

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