Polymicrobial Endocarditis with *Enterococcus faecalis* and *Proteus mirabilis:* A Case Report

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Polymicrobial endocarditis is a rare clinical entity associated with high rate of morbidity and mortality. We present a case of prosthetic valve endocarditis caused by *Enterococcus faecalis* and *Proteus mirabilis*, emphasizing the clinical course, microbiological findings, and potential microbial synergy contributing to disease persistence.

E. faecalis is a gram-positive, facultatively anaerobic bacterium that can cause significant morbidity and mortality. It commonly presents as bacteremia and endocarditis and is a major contributor to nosocomial infections. This bacterium produces biofilms on native and artificial tissues, allowing it to establish resistance to antimicrobial agents. Many infections caused by *E. faecalis* are polymicrobial, as its biofilm creates an environment that supports the colonization of other microorganisms, leading to mixed-species biofilms on endogenous tissues or medical devices.

P. mirabilis, a gram-negative bacterium in the Enterobacteriaceae family, is a common cause of urinary tract infections (UTIs), particularly in patients with urinary tract abnormalities or indwelling urinary catheters. It is frequently isolated in cases of gram-negative bacteremia.

Polymicrobial bacteremia is defined as the presence of two or more distinct microorganisms detected in blood cultures. The incidence of polymicrobial bacteremia is estimated at 2–20% of all reported bloodstream infections [1]. Gram-negative bacteria are commonly found in these cases, present in up to 70% of samples [2]. In comparison, gram-positive organisms are less frequently detected, appearing in 42% to 52% of cases [2]. The combination of both gram-negative and gram-positive bacteria is most prevalent in polymicrobial bacteremia. Enterococcus species are often cited as the most common culprits, accounting for approximately 16% of cases [1]. However, some studies have identified *Escherichia coli* as the most frequently found microorganism in polymicrobial bloodstream infections [3]. The most common sources of polymicrobial bacteremia include intra-abdominal infections, primary bacteremia, and UTIs [1].

Polymicrobial endocarditis is relatively uncommon, occurring in approximately 1–6% of cases according to medical literature [4]. This disease can be caused by various combinations of viruses, bacteria, fungi, and parasites. *E. faecalis* has been implicated in cases of polymicrobial endocarditis in combination with Q Fever [4].

PATIENT DESCRIPTION

A 73-year-old male presented with fever one day prior to admission. His family noted confusion and dyspnea over the past weeks. Three days earlier, he was discharged from another hospital where he was hospitalized with iron deficiency anemia and received a blood transfusion. Notably, the patient had no history of urinary catheter insertion.

His past medical history was significant for coronary artery bypass grafting with aortic valve replacement (AVR) in 2021, followed by a repeat AVR in 2022 due to *Staphylococcus capitis* endocarditis and a subsequent cerebrovascular accident, which resulted in residual right hemiparesis. He also underwent the implantation of a cardiac pacemaker due to complete atrioventricular block.

At admission, the patient exhibited a fever of 39.6°C. The physical examination revealed fine crackles in the right lower lung lobe and bilateral leg edema. Laboratory tests indicated elevated inflammatory markers, leukocytosis, neutrophilia, thrombocytopenia, elevated creatinine, elevated total bilirubin levels, and elevated C-reactive protein levels. Blood cultures were obtained, and antibiotic treatment was initiated. One day later blood cultures revealed *E. faecalis* and *P. mirabilis*, which were confirmed in 10 subsequent blood cul-

tures collected within the first 2 days of his hospital stay.

Urine culture was positive for *E. faecalis*. An abdominal CT scan was performed to investigate the source of polymicrobial bacteremia. It showed gallbladder thickening; however, the physical exam showed no tenderness, and repeated ultrasounds revealed no signs of gallbladder infection. The patient also had a recent colonoscopy before hospitalization that did not display any pathology, and no biopsies or interventions were noted. A ^{18}F-fluorodeoxyglucose positron-emission tomography/CT scan was conducted, which revealed no hypermetabolic findings suggesting the source of the fever or infection. The scan also demonstrated that the gallbladder had returned to its original size and shape. A transesophageal echocardiogram showed no signs of vegetation or valve destruction, while a cardiac CT scan demonstrated slight thickening of the aortomitral continuity and diffuse thickening of the right aortic leaflet with evidence of hypoattenuation affecting motion, which could indicate a thrombus.

The patient was classified as a high-risk case of bacteremia and was therefore treated with 2 weeks of ampicillin and high-dose ceftriaxone, followed by 2 weeks of high-dose amoxicillin and ciprofloxacin after discharge. During this treatment period, repeated blood cultures showed no growth. However, at the end of the antibiotic course, the patient was readmitted with renewed fever, and blood cultures once again revealed both *E. faecalis* and *P. mirabilis* in 4/8 subsequent blood cultures.

As the patient's respiratory condition deteriorated, and a repeat transthoracic echocardiogram was performed, which showed rocking of the prosthetic aortic valve and new severe mitral regurgitation. The patient subsequently experienced severe acute pulmonary edema and was rushed to emergency surgery. During surgery, following the removal of the prosthetic aortic valve, an area with an abscess containing pus was found in the commissure between the right and left coronary cusps. Cultures taken from the affected valve grew both *E. faecalis* and *P. mirabilis*.

COMMENT

E. faecalis, often implicated in polymicrobial infections, continuously secretes L-ornithine in lab settings, facilitating metabolic cross-feeding that boosts the growth and pathogenicity of *Clostridioides difficile* during gastrointestinal infections and supports the growth of *Escherichia coli* under iron-limited conditions during wound infections. In clinical settings, *E. faecalis* was found to cause polymicrobial endocarditis with Q fever [4].

We present a case of a 73-year-old male diagnosed with polymicrobial endocarditis of *E. faecalis* and *P. mirabilis*, confirmed by blood cultures and cultures from infected valves after surgical removal. No other source of infection was found, although a thorough investigation was conducted.

Prior studies have shown that *E. faecalis* and *P. mirabilis* share a symbiotic relationship within bacterial biofilms. These bacteria frequently co-colonize catheterized patients, leading to unique biofilm communities characterized by increased biomass, persistence, and antibiotic resistance. The proposed mechanism for this symbiosis involves an arginine/ornithine antiport system in *E. faecalis*, enhancing L-arginine biosynthesis in *P. mirabilis*. This metabolic interaction results in greater protein content and biofilm biomass, likely due to the coordinated activity of adhesins. This process promotes biofilm formation in vitro and affects the severity of disease in a mouse model of polymicrobial catheter-associated urinary tract infections (CAUTI) [5]. The established symbiosis observed in laboratory models for CAUTI suggests a similar relationship occurring on the artificial surface of a prosthetic valve, creating a biofilm consisting of *E. faecalis* and *P. mirabilis*. This biofilm, due to the metabolic interplay between the two organisms, exhibits increased biomass, persistence, and antibiotic resistance.

CONCLUSIONS

Polymicrobial endocarditis is uncommon. We present a case of polymicrobial endocarditis caused by *E. faecalis* and *P. mirabilis* and review the literature for possible mechanisms. Polymicrobial endocarditis should be considered as a possibility in patients with predisposition and combined *E. faecalis* and *P. mirabilis* bacteremia.

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