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Heart on Fire: An Incidentally Found Case of Native Valve Infective Endocarditis Due to *Escherichia coli*

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Abstract

Escherichia coli (*E. coli*) is a common bacterium in the intestinal flora. It can cause severe extraintestinal infections, including infective endocarditis (IE), a bacterial infection of the heart's endocardium and valves. Historically associated with rheumatic heart disease, IE now affects a broader range of patients, including those with prosthetic valves, congenital heart disease, and implantable electronic cardiac devices. While Staphylococcus and Streptococcus species are the leading causes of IE, *E. coli* IE is rare and presents unique clinical challenges. Here, we report a rare case of *E. coli* IE, highlighting its unusual presentation and management considerations.

Keywords: *Escherichia coli*; Infective Endocarditis; Urinary Tract Infection; Bacteremia; Endocardium; Antibiotics

Introduction

IE is a serious and potentially life-threatening infection of the endocardium, typically involving heart valves. This condition poses significant clinical challenges because of its diverse etiology, varied clinical presentations, and the potential for serious complications. While historically associated with rheumatic heart disease, the landscape of IE has shifted, with non-rheumatic heart disease now accounting for a significant portion of cases. This change is largely attributed to the decrease in the prevalence of rheumatic heart disease and the ever increasing prevalence of other predisposing risk factors such as intravenous drug use, prosthetic heart valves, implantable electronic cardiac devices, and healthcare-associated procedures.

The incidence of IE varies between 3 to 10 cases per 100,000 individuals annually, fluctuating over time and across different populations. Staphylococcus and Streptococcus species are the most common causative agents of IE, but rare pathogens like *E. coli* present unique clinical challenges. These challenges include atypical presentation and difficulties in diagnosis and treatment compared to more commonly implicated organisms. Early recognition and prompt initiation of treatment are paramount in managing IE to prevent complications and improve outcomes.

Case Presentation

A 75-year-old female with a history of type 2 diabetes mellitus, hypertension, and breast cancer post bilateral mastectomy on anastrozole presented to our facility as a transfer from an outlying facility for acute pyelonephritis, pneumonia, and new-onset atrial fibrillation with a rapid ventricular response. She originally presented to the outlying facility with worsening shortness of breath, productive cough, and nausea for one week. Her EKG revealed atrial

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fibrillation with a rapid ventricular response. Her pertinent laboratory workup was a white blood cell count of 17,000/µL and a serum sodium level of 126 mEq/L. Her urinalysis was consistent with a urinary tract infection (UTI). Radiography of the chest at the outlying facility revealed bilateral lower lobe infiltrates. Her computerized tomography (CT) abdomen showed cholelithiasis without cholecystitis and perinephric stranding on the right without hydronephrosis.

Upon arrival at our facility, her BP was 160/60 mm Hg, HR 109/minute, RR 22/minute, Temperature 36.8 °C, Spo2 93% on 4L via nasal cannula. She was diagnosed with sepsis because of UTI and pneumonia. She received fluid boluses and was started on intravenous (IV) vancomycin and cefepime based on the sepsis management guidelines. Her heart rate was initially controlled with IV amiodarone, but she was later switched to oral metoprolol based on her response. The blood and urine cultures grew *E. coli* sensitive to third and fourth-generation cephalosporins, ciprofloxacin, and carbapenem with intermediate sensitivity to levofloxacin and resistance to ampicillin.

Transthoracic echocardiogram to evaluate the new onset of atrial fibrillation revealed an ejection fraction of 65%, thickened mitral valve with mild regurgitation, and a 1.2 × 0.9 cm mobile vegetation on the anterior leaflet of the mitral valve (Figure 1), normal aortic and tricuspid valves, enlarged left and right atria with a pulmonary artery systolic pressure of 60 mm Hg and a right atrial pressure of 15 mm Hg. Her antibiotic regimen was de-escalated to IV ceftriaxone based on the sensitivity report. She responded well to medical management. She was discharged to an inpatient rehabilitation center, where she completed her six weeks of IV antibiotics. She was doing well on a telephonic follow-up six months after hospital discharge.

Discussion

In native valves, turbulent blood flow, mechanical damage, and debris in the blood (e.g., from IV drug use) cause tissue injury to the affected valves, setting off a cascade of injury, inflammation, and repair. This results in the deposition of plugs rich in fibrin, RBC debris, inflammatory cells, and platelets at the injury site. A circulating pathogen then colonizes this tissue plug, setting off an infection. In the early stages, infection is limited to the valvular structures. Failure to address the infection at this early stage will increase the risk of progression with invasion of the surrounding structures, resulting in the development of paravalvular abscesses, heart block, septic embolization, and eventually cardiac failure and death.

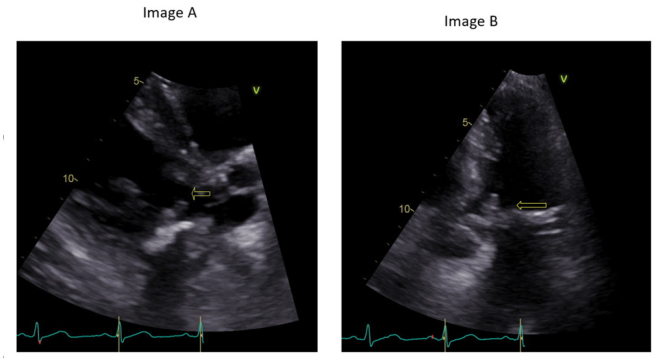

Figure 1: Image A Parasternal long view of the vegetation on the mitral valve, Image B Apical four chamber view of the vegetation on the mitral valve.

Staphylococcus, Streptococcus, and Enterococcus species account for approximately 80-90% of cases of infective endocarditis. Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, and Kingella, commonly known as HACEK organisms, account for between 6 and 14% of cases. Fungal endocarditis because of Candida and Aspergillus is rare and estimated to occur in only up to 1% of cases, especially in immunocompromised patients and those with mechanical valves. Other bacteria, including *E. coli*, cause approximately 6% of infective endocarditis.

E. coli is a normal inhabitant of the gastrointestinal tract and aids with metabolism. It is an opportunistic bacterium that can cause many infections, from travelers' diarrhea and simple cystitis to more severe infections like pneumonia, intra-abdominal and pelvic infections, meningitis, and bacteremia. It is commonly implicated in hospital-acquired infections, including catheter-associated urinary tract infections and ventilator-associated pneumonia with bacteremia. IE secondary to *E. coli* is rare, accounting for only 0.5% of the reported cases. In a literature review from 1909 to 2002, there were only thirty-six cases of *E. coli* IE involving the native valve. Most of these were because of UTIs, with 72% of them occurring in elderly females over 70 years with diabetes. Persistent bacteremia or widespread organ involvement with *E. coli* should prompt further diagnostic evaluations to rule out IE.

The low incidence of *E. coli* infective endocarditis may be due to naturally occurring antibodies against *E. coli* in normal blood and the lack of surface proteins decreasing the ability of the bacterium to adhere to the host matrix molecules and prosthesis. In the study by Morpeth and colleagues, *E. coli* was the most common non-HACEK gram-negative bacteria, followed by Pseudomonas causing IE, accounting for 29% of the cases. Most of these patients had indwelling endovascular devices and were nosocomial infections. Despite aggressive medical and surgical interventions, *E. coli* endocarditis is associated with significantly higher mortality than the HACEK organisms, 21% and 4%, respectively.

Most cases of *E. coli* endocarditis have no known valvular disease, contrary to those caused by Staphylococci,

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Streptococci, Enterococci, and HACEK organisms. Many patients with native valve *E. coli* infective endocarditis have positive blood and urine cultures. This could represent that the development of *E. coli* IE may be closely linked to the virulence factors of extraintestinal pathogenic *E. coli* in the urinary tract. Like in our patient, most of the time, it is unclear if the UTI with subsequent bacteremia causes infective endocarditis or vice versa, much akin to the UTI caused by Staphylococcal IE. At this time, it remains to be determined why *E. coli* infective endocarditis typically involves patients without preexisting valve disease. *E. coli* UTI complicated by bacteremia is not uncommon. However, the treatment for these two conditions differs and poses diagnostic and therapeutic dilemmas to clinicians. Treatment for IE involves intravenous antibiotics for six weeks based on the sensitivity, contrary to just ten days for complicated UTIs with bacteremia. This case should remind us of the complexity and challenges associated with managing *E. coli* infective endocarditis, especially in the setting of multiple comorbidities and concurrent infections. The prompt recognition and aggressive treatment strategy with appropriate antibiotic selection played a crucial role in our patient's successful outcome.

Conclusion

E. coli IE poses a multifaceted clinical challenge, necessitating a comprehensive understanding of its epidemiology, clinical presentations, and diagnostic and management strategies. Advances in diagnostic techniques and therapeutic modalities continue to refine our approach to this complex and potentially devastating condition. A high index of suspicion with appropriate therapeutic interventions is a must for successful management of the unusual manifestations of *E. coli*, including IE. This report should add to the growing literature on infective endocarditis caused by *E. coli*.

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