

A Case of Fever with Diagnostic Dilemma

Introduction

Infective endocarditis (IE) is a potentially life-threatening microbial infection involving the endocardial surface of the heart, most commonly affecting the cardiac valves, but occasionally involving the mural endocardium or implanted intracardiac devices (1). Despite significant advances in diagnostic techniques, antimicrobial therapy, and surgical management, IE continues to be associated with high morbidity and mortality worldwide, with reported in-hospital mortality ranging from 15–30% (1). The disease represents a complex interaction between host factors, microbial virulence, and hemodynamic conditions within the heart.

The pathogenesis of IE involves transient or sustained bacteremia or fungemia, allowing microorganisms to adhere to damaged endocardial surfaces or prosthetic material (1,2). Endothelial injury leads to deposition of platelets and fibrin, forming sterile thrombotic vegetation that provides a nidus for microbial colonization and needs prolonged duration antibiotic therapy. Subsequent proliferation of organisms within these vegetations results in persistent infection, embolization, and immune-mediated phenomena (1). A wide variety of microorganisms can cause IE, including bacteria and fungi; however, streptococci, staphylococci, and enterococci remain the most common causative agents (2,3).

Clinically, infective endocarditis exhibits a highly variable spectrum, ranging from an indolent subacute illness characterized by low-grade fever, malaise, and weight loss, to a rapidly progressive acute disease with severe sepsis and cardiovascular collapse (1,3). Common clinical manifestations include fever, cardiac murmurs, signs of heart failure, embolic events, and immunologic phenomena such as glomerulonephritis and vasculitis (1). The diagnosis of IE is often challenging due to its protean manifestations and relies on an integrated approach incorporating clinical findings, blood culture results, and echocardiographic evidence, as outlined in the modified Duke's criteria (4).

The epidemiology of infective endocarditis varies significantly between developed and developing countries. In high-income nations, IE is increasingly associated with degenerative valvular disease, prosthetic valves, intracardiac devices, and healthcare-

associated infections (3,5). In contrast, developing countries like India continue to report a high burden of IE related to rheumatic heart disease (RHD), younger patient populations, delayed presentation, and limited access to advanced healthcare facilities (5,6). Additionally, widespread empirical antibiotic use before hospital admission contributes to a high incidence of culture-negative endocarditis, complicating diagnosis and management (6). Given the changing epidemiological patterns, microbiological profile, and persistent high complication rates, infective endocarditis remains a major clinical challenge in the Indian setting. Case reports continue to play an important role in highlighting unusual presentations, diagnostic difficulties, and management strategies, thereby contributing to improved understanding and outcomes of this complex disease.

Case Presentation

Patient Information

A 70-year-old female patient presented to ER with complaints of Dyspnea and palpitation associated with generalized weakness for the last 4 days. It was also associated with a history of one week intermittent high-grade fever associated with chills and malaise. The reported dyspnea was progressive on exertion (NYHA II). It was insidious in onset and gradually progressive and had no relieving factor. There was no associated chest pain and any history of syncopal attacks. It was not associated with any seasonal or diurnal variation. There was no history of chest pain, palpitations, PND.

She was a known case of CKD Stage 5D on MHD (3/week), hypertension and hypothyroidism and on regular medication. She underwent a dental extraction four weeks prior to symptoms without antibiotic prophylaxis. There was no history of intravenous drug use.

Clinical Findings on Examination:

On Examination:

Patient was alert, conscious, cooperative well oriented to time, place and person. Temperature: 102 degree F
Pulse: 108/min, regular. BP: 106/70 mmHg.

In General Survey, there was conjunctival congestion present without itching or watering of eyes.

Respiratory examination revealed tachypnoea and cardiovascular examination revealed a high pitched pansystolic murmur best heard at the apex, radiating to the axilla with bibasal crepitations denoting heart failure. There was no focal neurological deficit. Examination of other systems were unremarkable.

Later, by the ophthalmoscopic examination, Roth's Spot with conjunctival haemorrhages were confirmed by the Ophthalmologist.

Treatment was started immediately after admission of the patient, initially with moist O₂ and Nebulization with Salbutamol. Foley's catheterization and i.v. cannulation was done. Inj Ceftriaxone - 1 gm in twice daily (APST) was administered with intravenous injection paracetamol, i.v fluid 0.9%, Normal Saline, Injection PPI and ondansetron. ABG was done. Blood Cultures sent with three sets of sample and as per IE protocol.

Other blood investigations were sent for:

Complete Blood Count, Random Blood Sugar, urea, creatinine, Sodium, Potassium, LFT, CRP, Procalcitonin. Fever profile

was also sent with MP Slide and dual Antigen, NS1 antigen, Typhi Dot M, Urine for RE/CS.

In Radiological investigation, Chest-x-ray done in PA view revealing cardiomegaly with hilar congestion, Echocardiogram-2D done, ECG was done. Laboratory Investigation showed:

Haemoglobin

-9.0g/dl TLC

- 18600/mm³

CRP-Elevated markedly.

Blood cultures were negative.

Patient was previously admitted in another hospital for two days before coming to this hospital.

In echocardiography, there was 'A large movable mass (26x14mm) attached to posterior mitral annulus in LA side'. with moderate MR, mild TR and Pulmonary arterial hypertension with normal ejection fraction. (**Fig 1**)

Transoesophageal Echocardiography (TEE) could not be performed due to some limitations and financial issues.

Diagnostic criteria:

Major criteria: Echo cardiograph's evidence of vegetation. Minor criteria: Fever, vascular and immunological phenomenon.

Therapeutic Intervention:

The patient was diagnosed as IE and started with Injection Ceftriaxone(1g) iv twice daily (APST), Injection Gentamicin(80 mg) once in every 48 hours(APST). She was also given Injection vancomycin (500mg.) post-Haemodialysis.(All antibiotics were given after renal dose adjustment) Supportive care included antipyretics and close monitoring. A multidisciplinary Endocarditis team, including cardiologist and cardiothoracic surgeon evaluated the patient. Surgical intervention was not indicated due to clinical improvement, controlled infection and lack of embolic events. Patient started to respond with diuretics, oxygen and antibiotics with gradual normalization of Total leukocyte count and Procalcitonin. The patient was referred to higher Centre for TEE after three days in a clinically and hemodynamically stable condition. All the reports of fever profile were negative, including blood culture due to prior antibiotics use. USG whole abdomen revealed no abnormality.

Discussion:

The clinical profile of infective endocarditis in India differs significantly from that observed in Western countries. Several Indian studies have demonstrated that native valve endocarditis accounts for the majority of cases, with rheumatic heart disease being the most common underlying cardiac abnormality (5–7). The mitral valve is most frequently involved, followed by the aortic valve, reflecting the high prevalence of rheumatic valvular lesions (5,7). Prolonged fever remains the most common presenting symptom, while heart failure is the most frequent and serious complication, often determining prognosis (7,8). Embolic complications involving the central nervous system, spleen, and kidneys are also commonly reported, particularly in patients with large vegetations and delayed diagnosis (6,8). These findings underscore the importance of early recognition and intervention. Blood culture remains the cornerstone of etiological diagnosis; however, Indian studies report culture positivity rates of only 50–60%, with culture-negative endocarditis occurring in up to 40% of cases (6,8). Prior antibiotic

exposure is the most important factor contributing to culture negativity. Recent Indian data suggest a shift in microbiological trends, with *Staphylococcus aureus* emerging as the most common causative organism, replacing viridans group streptococci, and reflecting an increase in healthcare-associated infections (9). Echocardiography is indispensable in the diagnosis and management of IE. While transthoracic echocardiography is widely available and serves as the initial imaging modality, transesophageal echocardiography offers superior sensitivity for detecting vegetations, abscesses, and prosthetic valve involvement (10). Management requires prolonged intravenous antimicrobial therapy tailored to microbiological findings whenever possible. Early surgical intervention in patients with heart failure, uncontrolled infection, or high embolic risk has been shown to significantly improve outcomes in Indian studies (7,8).

Conclusion:

Infective endocarditis remains a serious and potentially fatal disease in India, characterized by native valve involvement, a high prevalence of rheumatic heart disease, frequent culture-negative cases, and significant complication rates. Early diagnosis using modified Duke's criteria, prompt echocardiographic evaluation, appropriate antimicrobial therapy, and timely surgical intervention are crucial in reducing morbidity and mortality. Awareness of evolving microbiological trends is essential for guiding empirical therapy and improving patient outcomes.

References:

- 1) Sexton DJ, Chu VH. Infective endocarditis. In: Goldman L, Schafer A, editors. Cecil Textbook of Medicine. 26th ed. Philadelphia: Elsevier; 2020. p. 1898–1912.
- 2) Bennett JE, Dolin R, Blaser MJ, editors. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 9th ed. Philadelphia: Elsevier; 2020. p. 1025–1059.
- 3) Fauci AS, Braunwald E, Kasper DL, et al., editors. Harrison's Principles of Internal Medicine. 21st ed. New York: McGraw-Hill; 2022. p. 945–956.
- 4) Li JS, Sexton DJ, Mick N, et al. Proposed modification to the Duke criteria for the diagnosis of infective endocarditis. Clin Infect Dis. 2000;30(4):633–638.
- 5) Garg N, Kandpal B, Tewari S, et al. Characteristics of infective endocarditis in a developing country. Indian Heart J. 2017;69(3):301–306.
- 6) Senthilkumar S, Menon T, Subramanian G. Epidemiology of infective endoca

rditis in India. Indian J Med Res. 2010;132:124–130.

7) Math

RS, Sharma G, Kothari SS, et al. Prospective study of infective endocarditis from a developing country. J Assoc Physicians India. 2011;59:3–8.

8) Balakrishnan KG, Tharakan J, Titus T, et al. Infective endocarditis in India: a changing profile. Indian Heart J. 1995;47(2):121–126.

9) Khalid IA, Khan KA, Shahid M, et al. Changing trends in the microbiological profile of infective endocarditis. Indian Heart J. 2018;70(Suppl 3):S353–S357.

10) Bansal M, Kasliwal RR. Echocardiography in infective endocarditis. Indian Heart J. 2013;65(3):299–307.

Conflicts of interest-None

Source of Funding-Nil

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Fig 1: Mitral valve vegetation on Echocardiography

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