

Infective Endocarditis Complicated by Severe Mitral Regurgitation and Markedly Elevated Troponin Levels as A Prognostic Marker: A Case Report

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Background: Infective endocarditis (IE) is a potentially life-threatening infection of the heart valves, often caused by bacterial pathogens. It can lead to serious complications such as heart failure, embolic events, and myocardial injury. Troponins, cardiac biomarkers released from damaged myocardial cells, are frequently elevated in IE, particularly in cases with myocardial injury. Elevated troponin levels in IE may serve as a prognostic marker of adverse outcomes, including mortality.

Case Presentation: A 20-year-old male with no significant medical history presented to the emergency department with chest pain that had progressively worsened over the past week. On examination, the patient was stable, with a pan-systolic murmur at the apex, indicative of severe mitral regurgitation (MR). Laboratory tests revealed elevated troponin levels (5.553 ng/mL), and blood cultures returned positive for *Staphylococcus aureus*. A transthoracic echocardiogram and transesophageal echocardiogram (TEE) confirmed severe MR with large vegetation on the mitral valve. Despite starting a two-week course of intravenous antibiotics, the patient's condition persisted, with elevated troponin levels (8.6 ng/mL) and ongoing vegetation. Mitral valve surgery was performed due to the failure of medical therapy and the risk of further myocardial injury.

Discussion: This case underscores the critical role of elevated troponin levels as a prognostic marker for myocardial injury in infective endocarditis. Persistent troponin elevation, despite appropriate antibiotic treatment, indicates ongoing myocardial injury and suggests the need for urgent surgical intervention. Severe MR in IE contributes to hemodynamic instability and increases the risk of heart failure, necessitating prompt surgical correction. The collaboration between cardiologists, infectious disease specialists, and cardiac surgeons is vital in managing such complex cases.

Conclusion: Elevated troponin levels in infective endocarditis, particularly with severe mitral regurgitation, are indicative of significant myocardial injury and may predict adverse outcomes. Timely surgical intervention is crucial to improving patient survival and mitigating further complications.

Keywords: infective endocarditis, mitral regurgitation, troponin levels and prognostic marker

Introduction

Infectious endocarditis is the inflammation of the endocardium, the heart's inner lining, and the valves that divide the four cardiac chambers. It is mostly a bacterial disease with a broad range of symptoms and after effects. Numerous intracardiac and extensive extracardiac problems may arise if the condition is not detected and treated promptly. Consequently, meticulous assessment, encompassing a comprehensive history and physical examination, can facilitate diagnosis and inform management, hence reducing mortality and morbidity.¹

Cardiac biomarkers, particularly troponins, are essential tools in the diagnosis and management of myocardial injury in various cardiovascular conditions. Troponins, specifically troponin I and T, are proteins released from damaged myocardial cells and are widely used to diagnose acute myocardial infarction (MI). However, elevated troponin levels are not exclusive to MI and can also be seen in non-coronary conditions, including infective endocarditis, particularly when there is myocardial

injury resulting from embolic events, septic shock, or direct inflammation of the heart tissue.² Elevated troponin levels have been shown to correlate with worse outcomes in patients with infective endocarditis, indicating the severity of myocardial damage and suggesting a potential role as a prognostic marker in this population.³

The presence of various sequelae, such as heart failure, embolic events, abscess development, conductive abnormalities, and substantial vegetation size, is correlated with heightened in-hospital mortality in patients with infective endocarditis (IE).^{4–6} Troponins are cardiac-specific proteins produced during cardiomyocyte damage. In numerous disease states, including coronary artery disease, pulmonary embolism, heart failure, and various cardiac and non-cardiac disorders, elevated troponin levels may inadequately predict patient outcomes.⁷ Several studies with limited sample sizes have reported on the use of troponin as a clinical predictor of mortality in patients with infective endocarditis.^{7,8}

Prolonged intravenous drug use (IVDU) is a recognized risk factor for infective endocarditis (IE).⁹ Over the previous five decades, the prevalence of IE-related hospitalizations in North America has continued to rise with growing IVDU. *Staphylococcus aureus* is the predominant pathogenic bacterium.^{10,11}

In this case report, we discuss a patient with infective endocarditis complicated by severe mitral regurgitation and markedly elevated troponin levels. The significant elevation of troponin levels in this case serves as a crucial marker of myocardial injury, potentially due to embolic events or inflammation related to the infection. The role of elevated troponins in predicting outcomes in IE is becoming increasingly recognized, and our case underscores the need for careful monitoring and more aggressive treatment approaches in patients presenting with this combination of complications. Troponin elevation in IE may be an important factor to consider when assessing the severity of the disease and the need for urgent intervention.¹²

Case Presentation

A 20-year-old male presented to the emergency department with a complaint of chest pain that had started two months ago but had worsened significantly over the past week. He described the pain as sharp, located on the left side of his chest, occasionally radiating to his back. The patient denied associated symptoms such as fever, chills, cough, or shortness of breath. His medical history was unremarkable, and he did not smoke. Additionally, his family history of chronic diseases was non-contributory.

Upon assessment, the patient was stable with normal vital signs. A thorough cardiovascular examination revealed a pan-systolic murmur at the apex of the heart radiating to the left axilla, indicative of mitral regurgitation (MR). The remainder of the physical examination was unremarkable, with no signs of systemic embolization such as petechiae, Janeway lesions, or Osler nodes. Laboratory tests showed a hemoglobin level of 13.4 g/dL (hematocrit of 39.6%) and a white blood cell count of $13.75 \times 10^3/\text{mm}^3$, predominantly neutrophils.

The erythrocyte sedimentation rate (ESR) was mildly elevated at 5 mm, while C-reactive protein (CRP) was significantly high at 95 mg/L, indicating inflammation. The patient tested negative for HIV, HBsAg, and HCV. Hepatic and renal function tests, as well as urine analysis, were normal. However, troponin levels were markedly elevated at 5.553 ng/mL, indicating myocardial injury.

Based on the clinical presentation and laboratory findings, the patient was admitted for further evaluation. An electrocardiogram revealed an incomplete right bundle branch block (RBBB) with sinus tachycardia (Figure 1). A transthoracic echocardiogram (TTE) was performed to assess the cardiac structure and function. The TTE showed severe mitral regurgitation with extensive vegetation on the mitral valve leaflets, consistent with infective endocarditis (Figure 2). The left ventricular function appeared compromised due to volume overload. Subsequently, a transesophageal echocardiography (TEE) was conducted to obtain more detailed images of the mitral valve. It confirmed the presence of significant vegetation measuring 13×10 mm (Figure 3).

In summary, the patient presented with chest pain, significant mitral regurgitation, and elevated inflammatory markers. He was admitted to the hospital, and four blood cultures were taken, followed by the initiation of empirical antibiotic therapy. After three days, the blood cultures returned positive for *Staphylococcus aureus* (two positive cultures). Based on the Duke criteria for infective endocarditis (2023ESC guidelines), the patient met two major criteria, confirming the diagnosis of infective endocarditis.

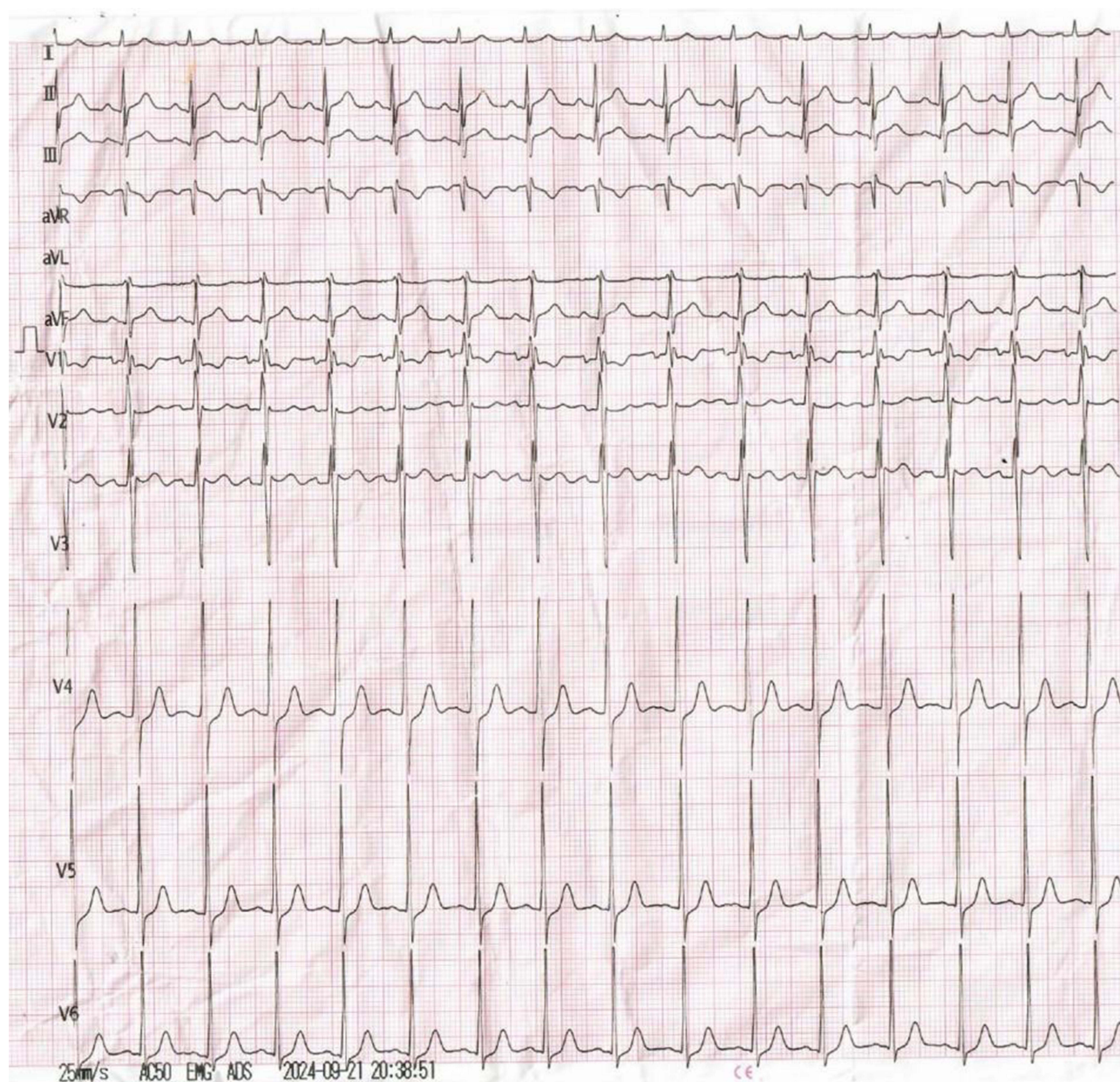


Figure 1 Incomplete RBBB with sinus tachycardia.

The patient was started on a two-week course of intravenous antibiotics. Despite completing the antibiotic course, he continued to have symptomatic severe mitral regurgitation and persistent vegetation. Follow-up blood cultures remained positive for *Staphylococcus aureus*, and a repeat troponin test still showed a high level of 8.6 ng/mL. Given the persistent positive blood cultures and elevated troponin levels indicative of ongoing myocardial injury, mitral valve surgery was performed. Three weeks later, the patient was stable, and his troponin level decreased to 1.5 ng/mL. One week later, the troponin level further decreased to 0.8 ng/mL, showing a gradual decline.

Discussion

Infective endocarditis (IE) remains a significant clinical challenge, particularly when complicated by severe mitral regurgitation (MR) and elevated troponin levels. This case report sheds light on the critical implications of these factors for patient management and outcomes. Severe MR in the context of IE can lead to rapid hemodynamic instability and

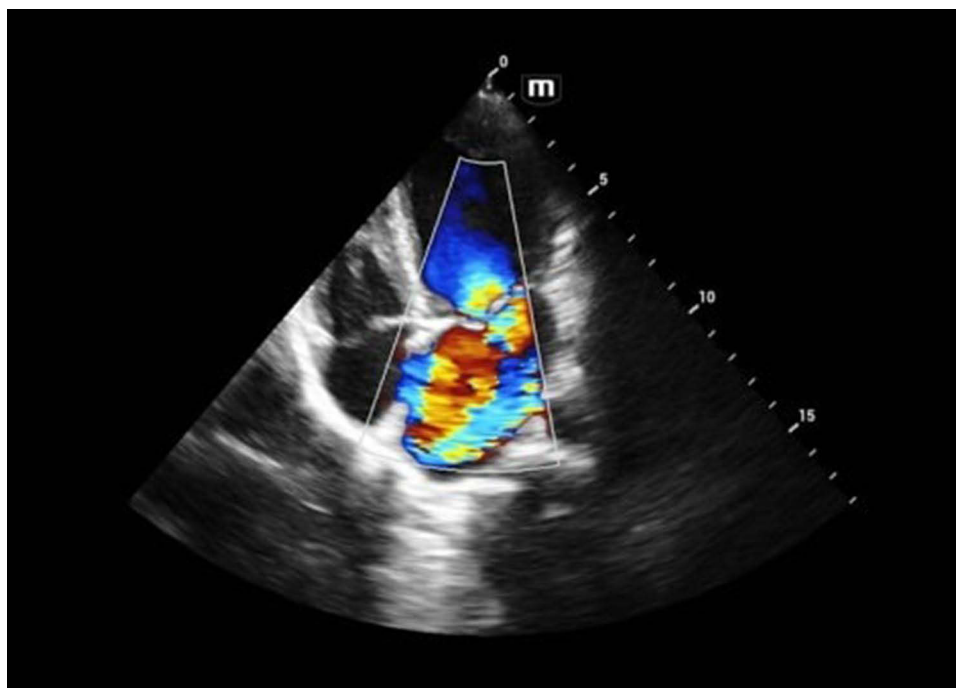


Figure 2 A Transthoracic echocardiography image showing the severe Mitral regurgitation.

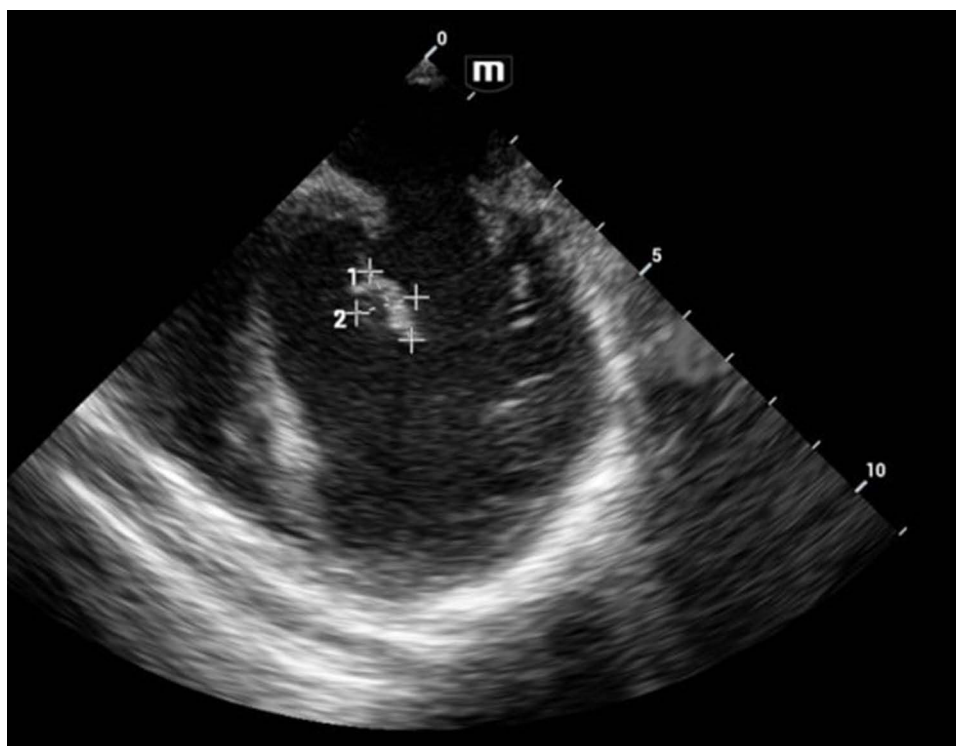


Figure 3 A Transesophageal Echocardiography image shows a large vegetation measuring 13x10mm attached to the posterior mitral leaflet.

heart failure due to the resultant volume overload and increased left atrial pressure. Studies have shown that the development of severe MR can precipitate acute pulmonary edema, shock, and multi-organ failure, necessitating urgent surgical intervention.¹³

In our case, the patient's deteriorating condition prompted immediate evaluation for surgical options, highlighting the importance of recognizing the urgency of such situations. Current guidelines recommend surgical intervention for patients with IE who exhibit signs of heart failure or significant valve dysfunction.¹⁴ Failure to act swiftly can lead to devastating outcomes, including increased mortality.¹⁵

Elevated troponin levels serve as a critical prognostic marker in patients with IE, reflecting myocardial injury and increasing the likelihood of adverse outcomes. Recent data indicates that elevated troponin levels, particularly in the context of infective endocarditis, correlate with an increased risk of death and sequelae, including myocardial infarction.¹⁶ Troponin elevation may arise from embolic events affecting coronary arteries, direct myocardial injury is also a possibility from the infective pathogen.

Surgeons, cardiologists, and infectious disease specialists must collaborate closely to assess the urgency of surgery, taking into account both the mechanical complications of the valve disease and the underlying myocardial condition.¹⁷ The timing of surgical intervention is paramount; delays can lead to irreversible myocardial damage, making early intervention critical for improving survival rates.

Moreover, the presence of severe MR in the context of infective endocarditis complicates the clinical picture significantly. Severe MR often leads to increased left ventricular volume overload, which can exacerbate heart failure symptoms and further strain the heart muscle. In this case, the severe MR likely contributed to myocardial injury, which was manifested by the elevated troponin levels. According to the 2024 ISCVI guidelines, severe valvular damage due to infective endocarditis requires prompt surgical intervention, particularly when there is evidence of heart failure or significant valvular dysfunction, as this can prevent further myocardial damage and improve overall prognosis.^{18,19}

In conclusion, this case highlights the complex relationship between infective endocarditis, severe mitral regurgitation, and markedly elevated troponin levels. The markedly high troponin levels observed in this patient are concerning, as they suggest significant myocardial injury and a poor prognosis. The integration of the Duke criteria for diagnosis, alongside the monitoring of biomarkers such as troponin, is essential for managing IE effectively and guiding therapeutic interventions. This case reinforces the value of a comprehensive approach to diagnosing and managing infective endocarditis, especially when complicated by severe valvular dysfunction and myocardial injury.

Ethical Approval

This case report is included in the publication; thus, further clearance from our Ethics Committee was not required.

Consent

Informed written consent was obtained from the patient, who was fully informed that details regarding his condition and any accompanying images would be published without disclosing his identity. The patient acknowledged and agreed to this arrangement.

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Disclosure

The authors declare no conflicts of interest pertaining to this report.

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