

## Tricuspid Valve Infective Endocarditis in an Intravenous Drug User with Methicillin Sensitive Staphylococcus Aureus Bacteremia

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### ABSTRACT

Infective endocarditis continues to cause significant mortality and morbidity across the globe. The infection is commonly seen in intravenous drug users, patients with prosthetics devices (pacemaker, prosthetic heart valve, knee or hip prosthetics) and in patients who are immunocompromised. The infective endocarditis is a systemic infection with varied presentations such as fatigue, tiredness, night sweats, fevers and weight loss and a wide spectrum of complications such as septic embolic, septic pulmonary infarct, infective ischemic stroke, splenic infarct, epidural abscess, osteomyelitis and discitis. The infection seeds to other organs via dissemination through bloodstream.

**Keywords:** Infective endocarditis; Septic embolic; Splenic infarct

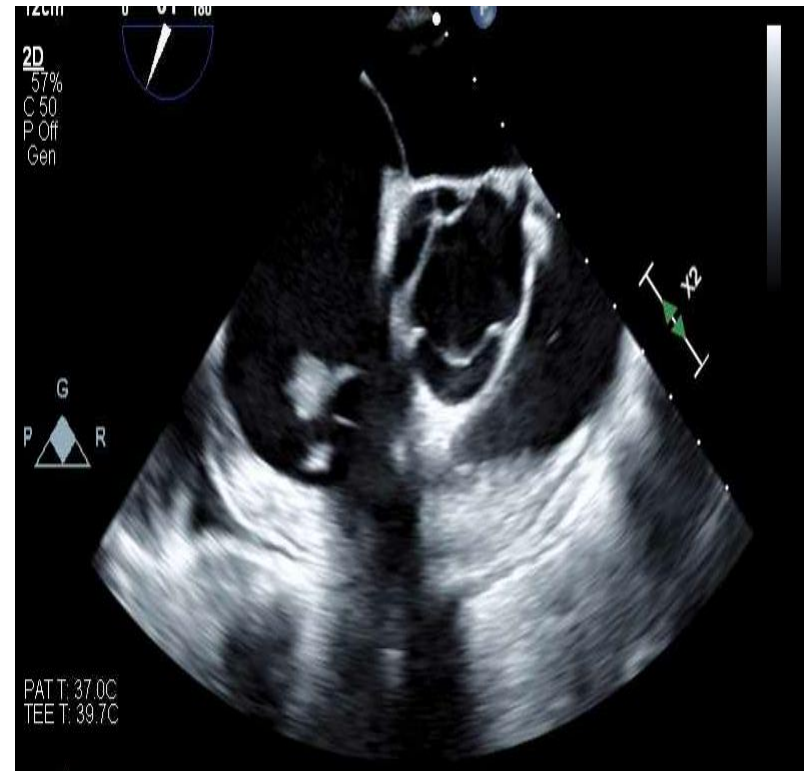
## INTRODUCTION

Infectious endocarditis is an inflammation of the endocardium. It is essentially a bacterial infection with numerous signs and consequences. Without prompt diagnosis and treatment, a slew of intracardiac and extracardiac problems might arise. Consequently, meticulous evaluation, including a comprehensive history and physical examination, can aid in the diagnosis of cases and guide treatment, therefore reducing mortality and morbidity.

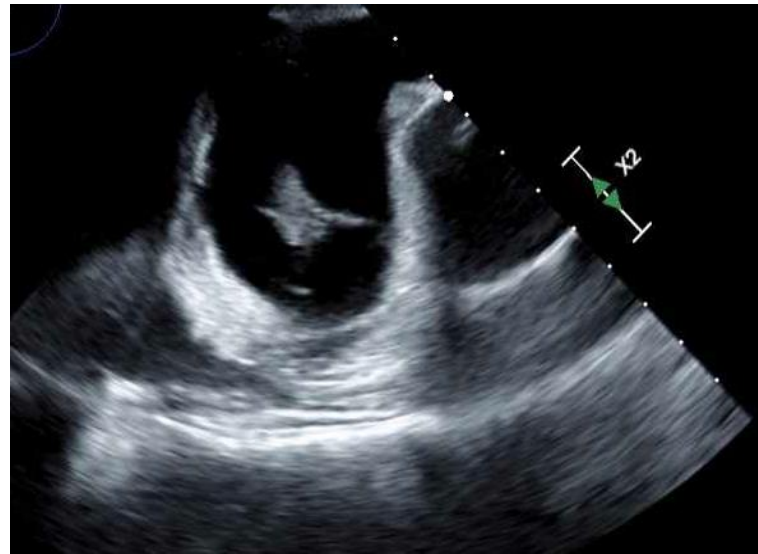
## CASE REPORT

46 years old gentlemen with history of intravenous (IV) methamphetamine, morphine, and heroine drug use presented to emergency department with fatigue and generalized weakness of 3 weeks duration. Patient further reported fevers but denied chills or night sweats. He had associated sharp chest pain and exertional shortness of breath of 1 week duration. The chest pain was left sided, sharp, non-radiating, non-exertional and 8-10/10 in intensity. He denied any significant past medical, surgical or family history. He did admit regular use of IV drug use. Patient was febrile with a Tmax of 102.F, tachycardic with a heart rate of 112 beats per minute and tachypneic with a respiratory rate of 22/minutes, although sating well on room air with pulse oximeter saturation of 98% on room air. Physical examination revealed poor dentition, respiratory rales, systolic murmur (3/6) over the left sternal border and needle stick marks. The physical examination was otherwise unremarkable. Laboratory evaluation on admission showed white cell count of 27000 /mL (4500-10500/mL), hemoglobin level of 14.5 g/dL (14-16 g/dL), platelet count of 325000/mL (145000-400000/mL), C reactive protein level of 325 mg/dL (8-10 mg/L), serum sodium of 142 mEq/L (135-145 mEq/L), chloride of 108 mEq/L (95-105 mEq/L), potassium of 3.7 mEq/L (3.5-4.5 mmol/L), creatinine of 1.2 mg/dL (0.8-1.3 mg/dL), alanine transaminotransferase of 38 U/L (10-40 U/L), aspartate aminotransferase 32 U/L (10-60 U/L), albumin- 3.8 g/L (3.5-5.0 g/L) and total bilirubin 0.8 (0.2-1.3 mg/dL). Blood cultures collection and patient was resuscitated with IV fluids for sepsis concerns, received total 3.5L of normal saline boluses and was empirically started on IV vancomycin and IV Zosyn for sepsis. A computed tomography with contrast was done given history of chest pain and dyspnea and it showed left sided segmental pulmonary infarct. The patient was started on IV heparin infusion for pulmonary embolism concerns. An echocardiogram was ordered given new murmur in the setting of sepsis. Echocardiogram revealed an ejection fraction of 50%, normal left ventricular size and tricuspid valve mobile vegetation (Figures 1& 2). Cardiology and infectious disease were consulted to assist with patient's management. Patient became afebrile within 24 hours of IV antibiotics treatment. IV vancomycin was continued while IV Zosyn was switched to IV ceftriaxone 1 gram every 24 hours. Blood cultures grew Methicillin sensitive Staphylococcus Aureus (MSSA). IV vancomycin was narrowed to IV cefazolin, infectious disease recommended a total of 4-6 weeks of IV antibiotics. Repeat blood cultures were negative. Patient did not have health insurance which complicated his ability to afford treatment or having home health services for IV antibiotics administration. IV cefazolin was switched to outpatient IV dalbavancin 1500 mg every 2 weeks for a

total of 4 weeks. IV heparin was switched to oral Eliquis for pulmonary embolism, although it was considered to be septic in nature and secondary to bacteremia, pulmonology recommended at least a short course (1-2 months) of anticoagulation with outpatient pulmonology follow up.



**Figure 1: Transesophageal echocardiogram with Tricuspid valve mobile vegetation**



**Figure 2: Transesophageal echocardiogram revealed mobile vegetation attached to the Tricuspid valve**

Follow up- Patient was doing well and had no complains during his 3 month follow up.

## DISCUSSION

Infectious endocarditis (IE) can be acquired in the community or by exposure in a healthcare setting. Community-associated infectious endocarditis refers to IE that develops in the absence of recent contact with a health care setting, whereas health care-associated infectious endocarditis refers to IE that develops in the context of recent contact with a health care setting, such as onset of symptoms 48 hours after hospitalization<sup>[1]</sup>.

In the majority of case series with IE, men predominate; male-to-female ratios range from 3:1 to 9:1<sup>[2]</sup>. While women are more susceptible to certain cardiac conditions<sup>[3-6]</sup>, IE is more prevalent in men. Bloodstream seeding with skin flora, mouth flora, and/or organisms infecting the substance or equipment used for injection are risk factors associated with injectable drug usage<sup>[7]</sup>. In addition, certain illegal medications may cause valvular endothelial damage, hence increasing the risk of

infection. Due to oral flora, poor dentition and/or tooth illness are likely risk factors for IE<sup>[8]</sup>. Dental procedures involving manipulation of gingival tissue or the periapical region of the teeth or perforation of the oral mucosa can enhance the risk for IE<sup>[9]</sup>.

Nearly three-quarters of individuals with IE have a pre-existing structural cardiac defect at the onset of endocarditis<sup>[10]</sup>. Rheumatic heart disease, mitral valve prolapses (typically with coexisting mitral regurgitation), aortic valve disease<sup>[4,5,11-16]</sup>, and other valvular anomalies are all examples of valvular disease. Staphylococci, streptococci, and enterococci are the three most common causes of IE worldwide. In the United States and the majority of developed nations, Staphylococcus aureus is the most prevalent cause of infectious endocarditis<sup>[17]</sup>. The underlying medical conditions, such as immunosuppression (malignancy)<sup>[18,19,12,14,20-27]</sup>, inflammatory disorders<sup>[12,16,21,28-30,30-40,41]</sup>, IV medication usage, and the presence of prosthetic devices, tend to increase the risk of IE. Patients with IE should receive multidisciplinary care including infectious disease, cardiology, and cardiac surgery specialists to optimize clinical evaluation and antibiotic and surgical treatment. The prompt diagnosis and administration of effective antimicrobial therapy to limit the risk of complications and the development of surgical indications are of paramount importance. The administration of antithrombotic treatment to lower the risk of thromboembolic consequences in IE, is not recommended. Evaluation of the need to remove infected implanted devices, arteriovenous fistulas, or grafts is highly encouraged and is mandatory to source control. The patient with cardiac abscess, valve perforation or valve leak may warrant evaluation and interventions by cardiothoracic surgery.

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