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Disseminated Infective Endocarditis Presenting as Hypotension

Rinusha Rachel*

Pulmonologist, Osmania University, Hyderabad, Telangana, India

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***Corresponding author:** Rinusha Rachel, Pulmonologist, Osmania University, Hyderabad, Telangana, India

ABSTRACT

Infective endocarditis is a systemic infection with an enigmatic clinical presentation, which can delay diagnosis and treatment, resulting in a wide range of consequences. Despite advancements in treatment choices and antibiotics, appropriate management of infective endocarditis remains a formidable challenge. Male gender, advanced age, the existence of a prosthetic heart valve, and recreational intravenous drug use remain the primary risk factors for community-acquired infective endocarditis. As the infection is systemically pervasive, resulting in a variety of cardiac and extracardiac consequences, a multidisciplinary and multispecialty approach is essential for accomplishing optimal treatment outcomes.

Keywords: Infective endocarditis; Hypotension; Prosthetic heart valve

INTRODUCTION

Despite treatment, infectious endocarditis (IE) is a leading cause of mortality and morbidity across the globe. Owing to its rather ambiguous presentation, it can be difficult for clinicians to diagnose. We describe a dubious presentation of infective endocarditis as hypotension in a young man patient who is otherwise healthy.

CASE DESCRIPTION

A 45-year-old male with history of alcohol use disorder was directed to present as Emergency department (ED) by the outpatient radiology department due to hypotension. The patient initially presented at outpatient radiology department for the X ray of his low back due to low back pain. He was noted to have a low blood pressure readings of about 90/56 mmHg prompting further evaluation in the ED. The hypotension was reaffirmed in the ED (92/62 mmHg), patient denied being on anti-hypertensive medications. On further questioning, reported feeling dizzy and lightheaded for one month prior to presentation. He also added that he had a recent tooth infection but otherwise denied any other recent illness/infection, sick contacts or travel. The patient reported that back pain also started about a month prior to presentation. He denied fevers, chills, night sweats but endorsed weight loss (15 pouds), fatigue and lethargy of 4-6 weeks duration. The patient reported drinking heavily in the past but had a recent inpatient acute alcohol rehabilitation and had been sober for at least one month prior to presentation. He denied history of liver cirrhosis, or hepatitis infection. The patient also denied other substance abuse including IV drug use. The patient reported going through a divorce and child custody legal battle and thought that was causing fatigue. Vitals on presentation were only remarkable for low blood pressure as mentioned above, but no fevers, tachypnea or tachycardia. The physical exam revealed a loud 3-4/6 systolic murmur over the aortic region. Initial blood work up showed leukocytosis with white cell count of 13.8 10^3/cmm (reference range 4.30-10.80 10^3/cmm), hemoglobin 8.6 g/dl (reference range 14.0-18.0 mg/dL), platelet count 524 10^3/cmm (reference range 150-400 10^3/cmm), creatinine was 1.0 mg/dL (reference range 0.9-1.30 mg/dL), C-reactive protein 198 mg/dL (reference range 0.0-1.0 mg/dL). Blood cultures were drawn, and he was empirically started on intravenous (IV) ceftriaxone and IV vancomycin. Due to the murmur and hypotension, IE was high in the differential, hence a transthoracic echocardiogram was obtained, which showed an ejection fraction of 60-65%, an echo density in the right coronary cusp of the aortic valve (AV) concerning for a vegetation (Figure 1) and moderate to severe AV insufficiency (Figure 2) along with AV perforation. Cardiology, cardiothoracic surgery and infectious disease were immediately consulted. The transthoracic echocardiogram findings were confirmed with a transesophageal echocardiogram (TEE) which also revealed 0.7 x 1.0 cm mass echo density on the right coronary cusp of AV with mild to moderate eccentric aortic regurgitation (AR/AI), AI maximum velocity (MV) 122.0 cm/sec and AI maximum peak gradient (MPG): 50.5 mmHg. A MRI of the lumbar spine was obtained due to low back pain concerning epidural abscess/discitis/osteomyelitis which did show a 2x2.2 cms L4 lumbar spine epidural abscess. The neurosurgery was consulted, and patient underwent prompt laminectomy and debridement of the abscess with local bone grafting, and a L5-S1 lumbar interbody fusion. Blood cultures grew pan sensitive *Streptococcus mitis.* Antibiotics were narrowed to IV ceftriaxone, repeat blood cultures were negative. The source of infection was infected tooth, and patient underwent immediate tooth extraction by oral maxillofacial surgery for source control. The patient also underwent AV repair for the severe aortic insufficiency and aortic valve small perforation by cardiothoracic surgery team. He had a left heart catheterization prior to the procedure which revealed normal coronaries.The patient received IV antibiotics for a total of 6 weeks duration and was doing well during his 3 months follow up.

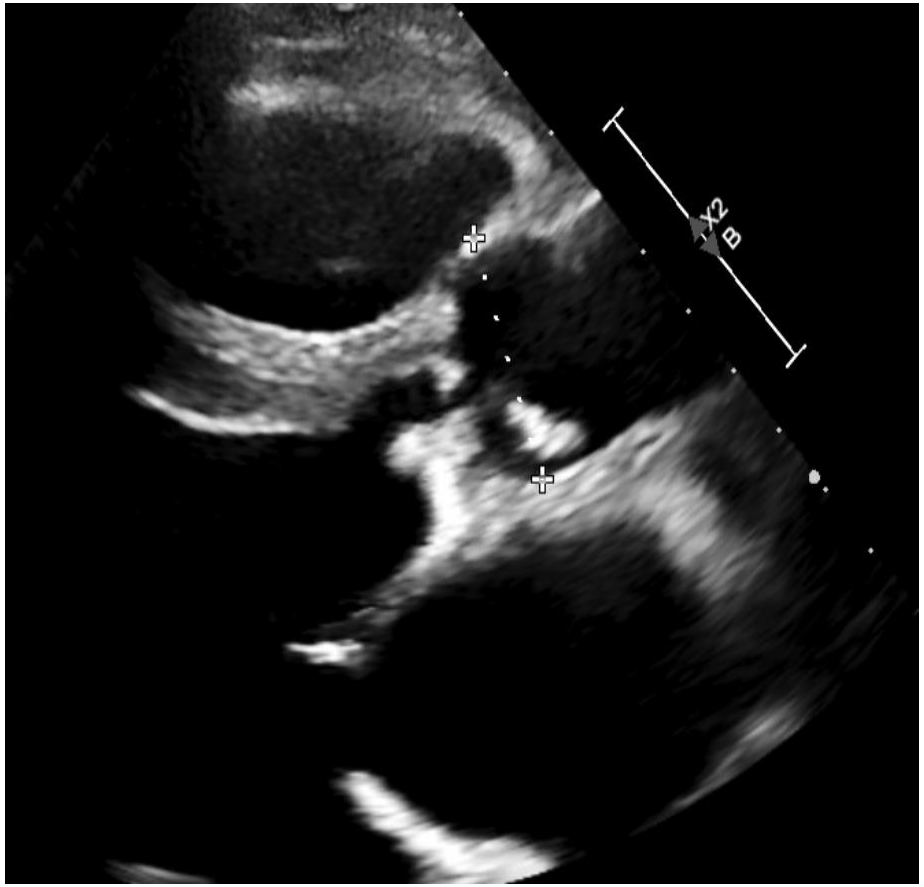

Figure 1: Transthoracic Echocardiogram showed mobile vegetation attached to the Aortic valve.

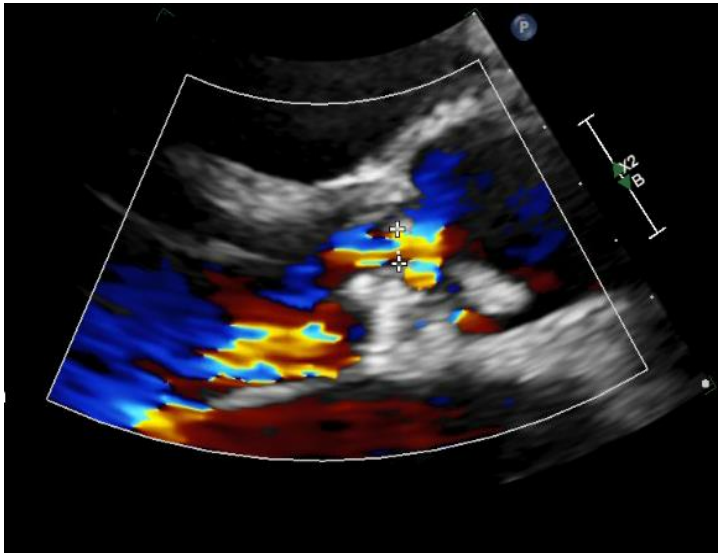

Figure 2: TTE Image revealed moderate to severe aortic regurgitation.

DISCUSSION

Infective endocarditis is a prominent cause of morbidity and mortality that has a substantial effect on healthcare. Despite effective medical treatment, according to a 2009 systematic review, fatality rates from IE are approximately 25% [1]. One study found that 31% of culture-positive IE was caused by Staphylococcus aureus, 17% by viridians group streptococci, 11% by Enterococci, 7% by Streptococcus Bovis, and 5% by other Streptococcus [2]. Systematic review a decade later revealed that the rate of staphylococcal IE had grown in the United States, while the rate of IE caused by streptococcus viridans and culture-negative IE had dramatically decreased[1]. The microbiologic profile of IE depends on the underlying risk factors[3-31].

Immunosuppression, intravenous drug use, poor dental hygiene, degenerative valve disease, and rheumatic heart disease are all risk factors for developing community-acquired infections. Intravenous drug use, which accounts for around 10% of infectious endocarditis cases, suggests frequent inoculation with skin flora such as S. aureus and S. epidermidis, with S. aureus showing a preference for healthy, native tricuspid valves. During the course of its existence, this disease process has consistently shown a preference for males, with a male to female ratio that is nearly 2:1. Patients diagnosed with infective endocarditis now typically have an average

age that is more than 65 years old. Its preponderance in the older population most likely corresponds to the greater frequency of predisposing variables within this demographic, such as prosthetic valves, indwelling cardiac devices, acquired valvular disease, hemodialysis, and diabetes mellitus. Rheumatic heart disease used to be a prominent risk factor; but, in this day and age of antibiotics, it only accounts for a small percentage (less than 5 percent) of all cases. The use of intravenous drugs for recreational purposes is an increasing risk factor that is currently responsible for approximately 10% of all instances of infective endocarditis[2].

Endocarditis can result in a multitude of intracardiac complications. Acute valvular incompetence can cause heart failure symptoms in about one-third of cases. This can happen as a result of an acute perforation of the valve or as a result of the chordae tendineae and papillary muscles becoming compromised. Regurgitation of the mitral or tricuspid valves can cause atrial enlargement and the subsequent development of atrial fibrillation and other supraventricular dysrhythmias. Intracardiac abscesses (14%) and atrioventricular blockages (8%), however, are much less common occurrences[2].

Vegetations may detach from the heart valve, leading to a wide range of sequelae, including ischemic stroke, pulmonary infarct, splenic infarct, intracerebral hemorrhage, meningitis, and intracerebral abscess. The hematogenous dissemination of the infection may result in epidural abscess, discitis, osteomyelitis, perinephric abscess, pyelonephritis, and septic arthritis. The keys to preventing and effectively treating problems connected to IE are repeating blood cultures, controlling the source of the infection, and broadening the antibiotics promptly.

CONCLUSION

This case illustrates the significance of early diagnosis and treatment of life-threatening infective endocarditis, which have a very subclinical and vague presentation. To avoid the catastrophe associated with infectious endocarditis, prompt identification and treatment are crucial. In addition to increasing the risk of cardiac complications, bacteremia is associated with various systemic complications, such as epidural abscess in our instance. The importance of a multispecialty strategy in the optimal care of this multifaceted infection cannot be overstated.

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