ISSN: 2634 - 6796

Journal of Cardiology Research Reviews & Reports

SCIENTIFIC Research and Community

Case Report

Open Access

Eustachian Valve Infective Endocarditis: A Rare Consequence of Implanted Venous Access Ports

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ABSTRACT

We present a case of eustachian valve endocarditis in an elderly male who presented after removal of a central venous access port. This case report also reviews the prevalence of right sided endocarditis, diagnosis, common microorganisms, predisposing risk factors and medical management. Eustachian valve endocarditis was first described in 1986 by Edwards and from 1986 to 2017, only 37 cases have been reported— 46% of which were caused by intravenous (IV) drug use and 24% by indwelling intravenous lines. Other risk factors for the development of eustachian valve endocarditis include rheumatic heart disease, pacemaker wires and immunologic compromise. This case illustrates the importance of recognizing risk factors other than IV drug use in the pathogenesis of right-sided endocarditis, especially in cases involving the eustachian valve.

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Received: February 25, 2022; **Accepted:** March 05, 2022; **Published:** March 15, 2022

Keywords: Eustachian Valve, Endocarditis, Right-sided endocarditis.

Introduction

The eustachian valve (EV) is an embryologic remnant found at the junction of the inferior vena cava and the right atrium. It forms a thick ridge, inferiorly placed in the right atrium [1].

Embryologically, the valve plays a role in directing oxygen-rich blood from the IVC away from the tricuspid valve and toward the foramen ovale [2]. After birth it regresses completely, partially, or persists altogether in some individuals [2]. This leads to variations in ridge thickness that can be noted on echocardiography. In the absence of structural or functional cardiac abnormalities with remarka4ble clinical outcomes, a persistent eustachian valve does not warrant surgical intervention [1]. This structure has been called "the valve of the IVC," and its importance is noted in literature related to trans-catheter procedures, closure of the foramen ovale and atrial septal defects [1, 2]. It can be thickened and cause difficulty in placement of closure devices. In cases where a patient also has a concomitant patent foramen ovale, the presence of the EV greatly increases the risk of stroke [1]. Literature further highlights the importance of recognizing the EV and the role it plays in right-sided endocarditis. Eustachian valve endocarditis is an extremely rare consequence of this embryologic remnant that is likely underdiagnosed [3]. We present a case report of a patient with no history of intravenous drug use and a recent transvenous port removal who was hospitalized for persistent bacteremia due to eustachian valve infective endocarditis.

Case Presentation

The patient was an 86-year-old man with adenocarcinoma of the lung that was metastatic to the cerebellum, status post chemotherapy and radiation treatments, who presented with nondescript, diffuse right shoulder pain for three days. He described the pain as "stabbing" and worse with movement. He felt the pain was reproducible, however he was noted to be tender to palpation only in the clavicular area where a prior venous catheter port was placed. He reported a regimen of 14 weeks of once-weekly chemotherapy with Lambrolizumab. He had also completed a course of radiation in July 2021 directed at the base of his skull for the cerebellar metastases. He denied a history of intravenous drug use. The patient endorsed fevers and chills for three days but denied dizziness, headache, chest pain/tightness, diaphoresis, shortness of breath, abdominal pain, nausea, vomiting, diarrhea, or dysuria. He was found to be febrile on initial presentation. Blood cultures drawn in the Emergency Room eventually grew gram positive cocci in clusters in two out of two sets. He met criteria for severe sepsis on admission and was placed on empiric antibiotics. T-max was 101.5°F with a heart rate of 103. Blood pressure was stable in the 140s systolic. Chest radiograph was without acute abnormality. Cervical spine and shoulder radiographs noted spondylosis in C5-C6 and C6-C7 with osteoporosis/osteopenia and an incompletely-visualized, mass-like density in the left upper lobe of the lung. On physical examination, HEENT was unremarkable, lungs were clear to auscultation, cardiac exam was without murmur, and abdomen was benign without organomegaly. His extremities were noted to have a few splinter hemorrhages under two nails of his right hand. No skin rash was present except for the occasional ecchymosis on the extremities.

Overnight telemetry monitoring showed paroxysmal episodes of atrial fibrillation (CHADSVASC calculated to be 4) with 21 beats of nonsustained ventricular tachycardia. The patient stated he felt asymptomatic during the episodes. On day two of admission,

repeat blood cultures were collected. At this point, no defined source for the bacteremia had been identified. An MRI of the right shoulder, done to rule out osteomyelitis, was only remarkable for mild diffuse osteopenia. A 2D transthoracic echocardiogram (TTE) was obtained to rule out endocarditis, which demonstrated an ejection fraction (EF) of 55-65%, no valvular abnormalities and no wall motion abnormalities. Blood cultures finalized into penicillin binding protein 2a (PBP2a)-negative staphylococcus aureus (methicillin sensitive) with elevated inflammatory markers of erythrocyte sedimentation rate (ESR) of 53 and C-reactive protein (CRP) of 4. With a third set of blood cultures remaining positive for this gram-positive organism, a transesophageal echocardiogram (TEE) was ordered. Cardiology was consulted for the new-onset paroxysmal atrial fibrillation as well. Cardiology deemed the patient an appropriate candidate for anticoagulation and initiated apixaban 5 mg twice daily as well as metoprolol tartrate 25 mg twice daily for rate control.

A standard 2D TEE with spectral and color-flow Doppler was performed using the Omniplane probe through transesophageal and transgastric windows. The study was of a diagnostic quality. The left ventricle was of normal size. There was normal left ventricular wall thickness. Left ventricular systolic function was preserved. EF was noted to be 55-60%. There were no regional wall motion abnormalities. The right ventricle was of normal size and right ventricular systolic function was normal. The interatrial septum was aneurysmal with no evidence of an atrial septal defect. The left atrium was normal in size.

No thrombus was visualized in the left atrial appendage. No left atrial mass or thrombus was visualized. Two out of four pulmonary veins visualized drainage to the left atrium. Right atrial size was grossly normal. There was a mobile, echo-dense mass attached to the chiari/eustachian valve, measuring 0.8 cm by 0.6 cm (Figure 1).

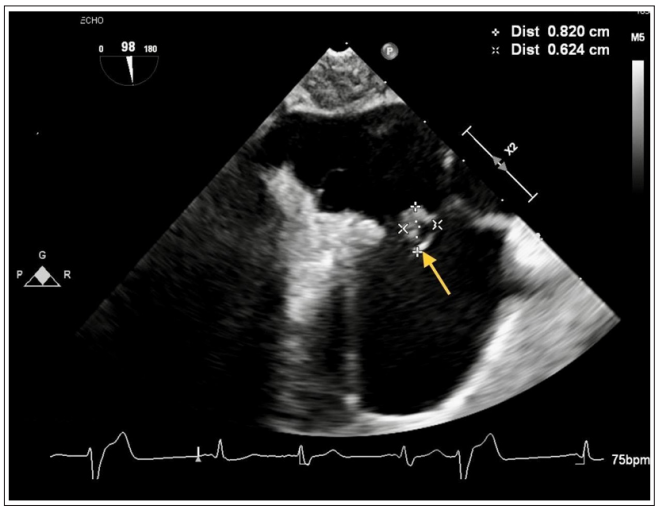

Figure 1: Transesophageal Echocardiogram showing a mobile echo dense mass (yellow arrow) attached to the Chiari-Eustachian valve complex within the right atrium. The top right corner estimates the two dimensional length and width of the mass in centimeters (cm).

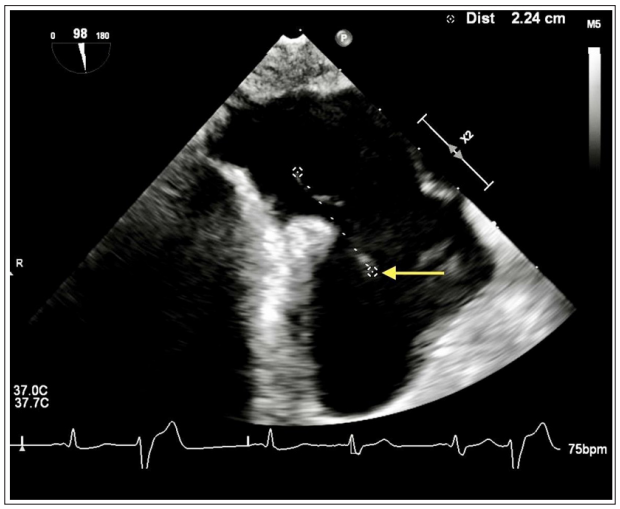

Figure 2: Transesophageal Echocardiogram showing the serpentine shaped attachment (yellow arrow) to the Chiari-Eustachian valve complex within the right atrium

The differential diagnosis for the echocardiographic findings above included a thrombus versus a vegetation. However, given the intracardiac evidence of a vegetation, a typical microorganism (S. aureus) in two sets of blood cultures and persistently positive S. aureus blood cultures drawn more than twelve hours apart, the patient met two of the major criteria for diagnosing endocarditis. Infectious disease was consulted for management of methicillin-resistant Staphylococcus aureus (MSSA) eustachian valve endocarditis, for which 5 weeks of intravenous cefazolin was recommended. A fourth set of blood cultures was negative for organismal growth, and the patient was no longer febrile after the initiation of cefazolin. Both cardiothoracic and vascular surgery were consulted for the EV endocarditis; they determined the patient was not a surgical candidate given clearance of blood cultures and no other apparent complications from the endocarditis. As the patient's condition improved, a peripherally inserted central catheter (PICC) was placed, and he was discharged on intravenous cefazolin to a long term acute care facility.

Discussion

Endocarditis is defined as inflammation of the endocardium, the innermost layer of the heart [4]. This inflammation results in the deposition of fibrin and platelets to form lesions known as vegetations, which can be non-infectious or infectious [4,5]. The diagnosis of endocarditis is based on clinical classification of the modified Duke criteria [5, 6]. The major criteria include echocardiographic evidence of endocarditis by view of a vegetation or an abscess, laboratory evidence of typical microorganisms that cause infective endocarditis in two separate blood cultures or new valvular regurgitation [5, 6]. The minor criteria include risk factors such as intravenous drug use or predisposing cardiac conditions, vascular phenomena such as janeway lesions, immunologic phenomena such as Osler's nodes, fever greater than or equal to 100.4o F, and positive blood cultures that do not meet major criteria [5, 6].

Endocarditis can affect the right or the left side of the heart. However, infective endocarditis most often occurs on the left side of the heart, commonly affecting the mitral or aortic valves [6].

Upon review of the existing literature on endocarditis, right-sided disease accounts for approximately 10% of cases [7]. Classically,

risk factors for right-sided endocarditis include intravenous drug use and recent central venous catheter or intracardiac device placement. These predisposing factors commonly result in tricuspid valve endocarditis and very rarely involve the eustachian valve. Endocarditis involving this embryologic remnant was first described in 1986 during an autopsy by Edwards et al [7]. In 2017, there were 37 cases described in 29 case reports [7]. An updated search of the National Library of Medicine results in 53 total case reports.

The most common organism seen in right-sided endocarditis is Staphylococcus aureus, accounting for 60-90% of cases [7]. When looking at reports of patients who had right-sided endocarditis of the eustachian-chiari network, 44% of these patients had a history of intravenous drug use, 52% did not have involvement of other cardiac structures and 70% required a transesophageal echocardiogram for accurate diagnosis [7].

The pathogenesis of right-sided infective endocarditis involves repeated trauma to the endothelial lining, which encourages the deposition of matrix molecules, serum fibronectin and fibrinogen and subsequent binding of these materials to microorganisms [5, 6]. These microorganisms further coat foreign substances like catheters and foreign particulates that are injected during intravenous drug use [6]. Staphylococcus aureus, for example, contains surface proteins that allow it to adhere to these matrix molecules. This can explain why S. aureus appears to be the most commonly isolated organism in this condition [8]. In order to diagnose a right-sided infective endocarditis, transesophageal echocardiography is the gold standard [8]. However, using a transthoracic approach will also provide valuable information, especially in the case of tricuspid valve involvement [8].

Right-sided infective endocarditis is associated with better clinical outcomes when compared with left-sided IE [9]. Reasons for this may include younger patient age and less hemodynamic consequences with tricuspid valve dysfunction when compared to mitral or aortic valve IE, allowing right-sided IE to be better clinically-tolerated. Such consequences include systemic embolization, abscess formation, and drug-resistant infection [10].

Conclusion

All valves, including the vestigial valves, and the right side of the heart should be carefully interrogated during echocardiography in patients with a high suspicion of endocarditis. While TTEs are typically performed as initial imaging, TEEs have more sensitivity and can identify smaller valvular vegetations or lesions. While intravenous drug use is the most common risk factor for right-sided infective endocarditis, it is not the only risk factor that clinicians should routinely assess. Prolonged venous access ports are of equal importance in the determination of a patient's risk. This case not only highlights the clinical need to recognize and address all risk factors that can cause right-sided endocarditis, but also emphasizes the utility of TEEs in identifying endocarditis involving the eustachian complex.

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