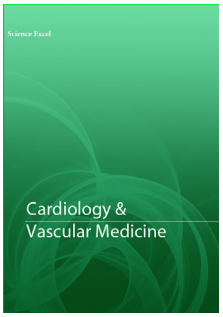
Cardiology and Vascular Medicine

Case Report



Enterococcal Prosthetic Valve Infective Endocarditis Presenting as Complete Heart Block with Intramyocardial Abscess Causing Asystolic Cardiac Arrest

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Abstract

The extension of an intracardiac abscess causing complete heart block is a rare complication of infective endocarditis that is associated with a high mortality. Early identification of conduction abnormalities and a low threshold for suspecting infective endocarditis is crucial to provide prompt management to prevent intracardial extension of infection. We report a case of a patient presenting with complete heart block in the setting of profound hyperkalemia, and was then found to have enterococcal prosthetic valve endocarditis, which was complicated by an intracardiac aortic root abscess which led to asystolic cardiac arrest. The development of heart block in endocarditis serves as a marker for poor prognosis and can signify progression of infection. Management therefore requires immediate pacing, antibiotic delivery to lessen infectious burden, and evaluation for consideration of surgical options such as valve replacement. It is therefore recommended for patients with endocarditis complicated by conduction abnormalities or intracardial abscesses to be treated by a multi-disciplinary team consisting of cardiologists, cardiothoracic surgeons, and infectious disease specialists.

Introduction

Infective endocarditis (IE) is a pathological condition in which microorganisms cause damage to the endothelium of the heart and septic vegetations to form [1]. If left untreated, it can become a fatal condition, as mortality for prosthetic valve endocarditis (PVE) is 20-40% and rates of recurrent PVE is 6-15% for patients [1]. Because of the many end-organ dysfunction manifestations, IE is best managed utilizing a multi-disciplinary approach among cardiologists, cardiothoracic surgeons, and infectious disease physicians.

The incidence of IE is about 1 case per every 1,000 hospitalizations [2]. Common risk factors associated with IE include structural cardiac abnormalities, intravenous drug use, catheter-associated bacteremia, immunosuppression, and prolonged surgery. The most common etiology associated with IE are structural heart abnormalities with mitral valve prolapse with regurgitation being the most common.

PVE accounts for 10-20% of all cases of IE. The majority of PVE are caused by streptococcal species such as S. aureus or coagulase-negative staphylococci [3]. Enterococcus or fungi associated PVE is rare but has a higher mortality. Enterococcal PVE is increasing in prevalence and is more commonly seen in patients with a history of genitourinary or gastrointestinal procedures. PVE is more likely to be associated with pathogens that produce a biofilm to shield the infection from antibiotic delivery making infection eradication difficult. These infections usually require mechanical disruption of the biofilm in surgery to help clear the infection [4]. Here, we present a case of enterococcal associated PVE.

Case presentation

We present a case of a 71yo M with history significant for Covid-19, coronary artery disease and aortic stenosis status post bioprosthetic aortic valve replacement, HTN, who presented to our institution as a transfer from an outside hospital for escalation of care. Initially, the patient presented to outside hospital for worsening shortness of breath and was found to have Covid. Of note, the patient was fully vaccinated and boosted. At the outside hospital, the patient received dexamethasone, 2 doses of remdesivir, and antibiotics for concern of a superimposed bacterial pneumonia. The patient was

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intubated due to worsening respiratory status and hospital course was further complicated by hyperkalemia to 7.1 mmol/L requiring dialysis. Furthermore, the patient was bradycardic down to the 40s. Electrocardiogram (EKG) obtained exhibited high degree heart block in the setting of metabolic acidosis and hyperkalemia which necessitated transvenous pacing (TVP) and transfer to our institution for escalation of care.

Upon presentation to our institution, the patient was normotensive, afebrile, bradycardic with a pulse of 55 bpm, hypoxic with a pulse oximetry 90%, and with an unremarkable physical exam. CXR showed findings consistent with pneumonia and the patient was placed on broad spectrum antibiotics. Labs were significant for a pH of 7.2 on arterial blood gas and hyperkalemia up to 6.8 mmol/L. The patient's EKG showed third degree heart block, a left bundle branch block, widening of the QRS complexes, and hyperacute t waves. The patient went into asystolic cardiac arrest and cardiopulmonary resuscitation (CPR) was immediately started with chest compressions and epinephrine. Return of spontaneous circulation (ROSC) was achieved and multiple vasopressors were started for pressure support. Electrophysiology (EP) was consulted for the high degree heart block and recommended placement of a MICRA leadless pacemaker once the patient was stable.

Blood cultures came back positive for enterococcus faecalis in both bottles, making contamination unlikely. The patient was de-escalated from broad spectrum antibiotics to ampicillin and ceftriaxone. A trans-esophageal echocardiogram (TEE) was performed due to possible concern for infective endocarditis and showed a LVEF 54% and a 3 mm hypermobile vegetation on the anterior leaflet of the mitral valve along with thickening of the posterior aortic root. Cardiothoracic surgery was consulted regarding bioprosthetic aortic valve infective endocarditis complicated by aortic root abscess and, after reviewing the patient in conjunction with the endocarditis team, deemed him a candidate for a redo aortic valve replacement (AVR) and mitral valve replacement (MVR).

The patient was extubated and EP successfully implanted a leadless MICRA AV pacemaker and removed his TVP. The patient's clinical course improved over the next couple of days as his latest arterial blood gas showed a resolution of his prior acidemia, normalization of his potassium levels, and stabilization of his creatinine with a non-oliguric urine output allowing for dialysis to be discontinued.

The patient was stable enough to be transferred to the floors prior to his surgery. Cardiology saw the patient for pre-op risk stratification and left heart catheterization. Coronary angiography revealed non-obstructive coronary artery disease. Cardiology commented that the patient was at high risk for perioperative cardiac complications due to the patient's age, prosthetic valve endocarditis, acute renal insufficiency requiring dialysis, and periannular complications. The RiskE score was calculated at 29 representing a 40% predicted in-hospital post-op mortality.

The patient successfully underwent re-operative AVR/MVR and explantation of leadless MICRA permanent pacemaker with placement of permanent epicardial pacing leads on the ventricle and atrium. EP then connected the surgically placed epicardial leads to a dual chamber permanent pacemaker generator. Post-operatively, the patient endorsed worsening dyspnea and CXR showed flash pulmonary edema. The patient underwent aggressive diuresis with resolution of symptoms and was told to follow-up as an outpatient to EP after discharge.

Discussion

The clinical manifestations of IE are variable and depend on the virulence of the causative pathogen but, classically, the hallmarks of the condition are fever and a new murmur which is found in over 85% of patients [5]. The patient can have elevated erythrocyte sedimentation rate, C-reactive protein, rheumatoid factor, or even elevated Venereal Disease Research Laboratory test titers, however, these are all non-specific markers for IE. Our patient did not have these lab abnormalities and with the exception of a mild leukocytosis

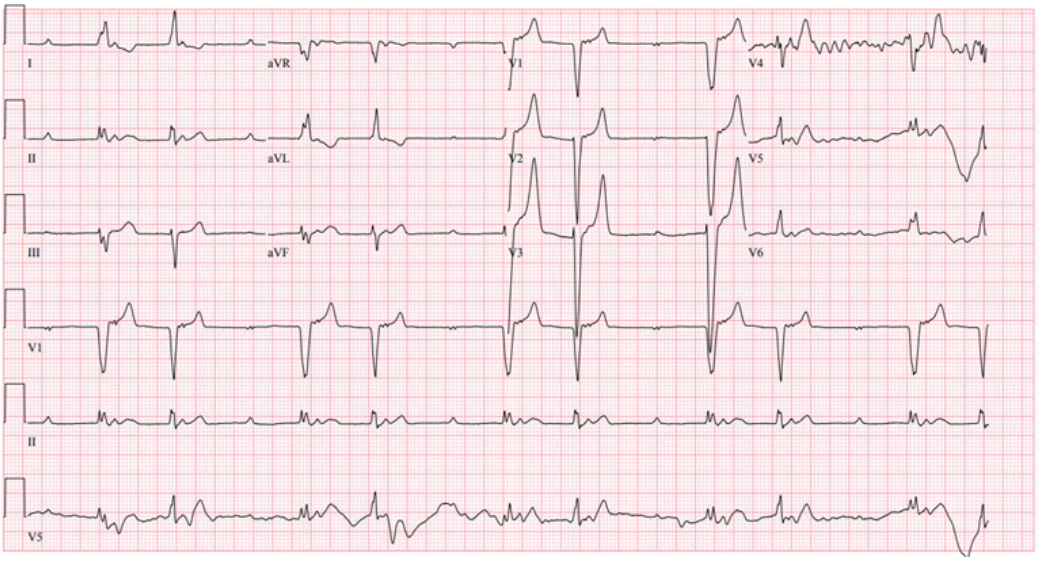

Figure 1. *EKG showing third degree heart block, widening of QRS complexes, and a left bundle branch block.*

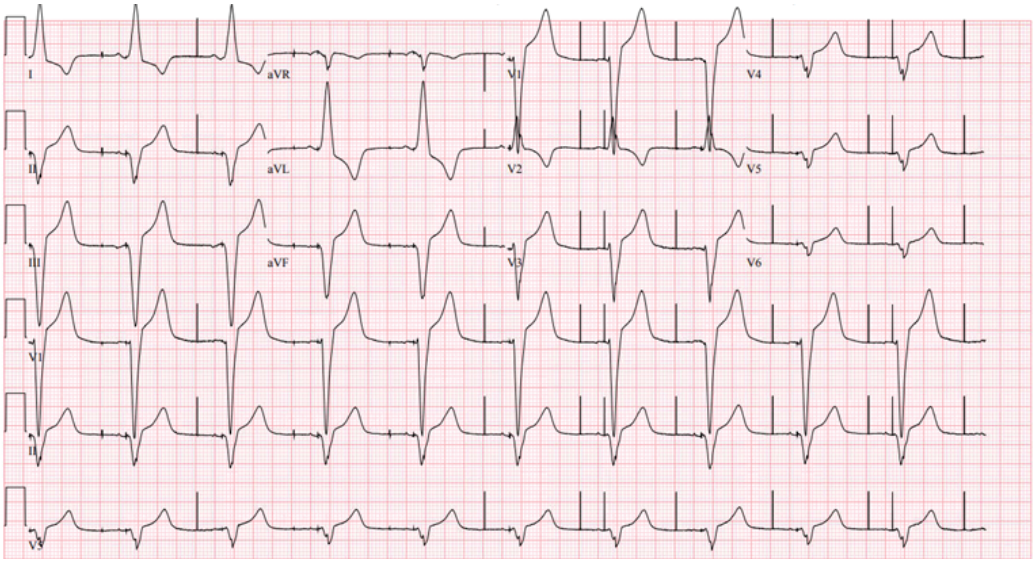

Figure 2. *EKG showing an AV dual paced rhythm after dual chamber permanent pacemaker implantation.*

which was most likely multifactorial in etiology, infectious workup was unremarkable.

The beginning of vegetation formation starts with endothelial injury triggering platelets and fibrin products to adhere to one another causing nonbacterial thrombotic endocarditis. The presence of microorganisms in the blood can then infect the sterile platelet-fibrin nidus resulting in IE [6].

The vegetations that result on the endocardium can have a number of complications. They can interfere with cardiac valves and prevent complete closure resulting in a valvulopathy, a murmur on physical exam, or worsening of regurgitation causing congestive heart failure (CHF) if significant chordal involvement is present. The vegetations may dislodge causing septic peripheral embolizations to the kidneys, brain, spleen, or limbs [5].

The infection can also spread to surrounding structures and disrupt the conduction system leading to arrythmias and heart block as well as abscesses and aneurysms. EKG testing may show conduction abnormalities which serves as a marker for intramyocardial extension of infection. Discovery of a new atrioventricular block carries a 77% positive predictive value for abscess formation with 42% sensitivity [6]. Conduction abnormalities are an ominous prognosticator of patient mortality due to invasive intramyocardial extension of infection leading to potentially fatal arrythmias or heart block. Our patient's initial presentation of heart block was thought to be secondary to hyperkalemia but evidence of vegetations and an aortic root abscess makes the etiology of his asystolic cardiac arrest most likely multifactorial. Conduction abnormalities usually co-exists with intracardiac abscesses with valve-ring abscesses being a classic complication of PVE. Risk factors for such abscesses include PVE, CHF, persistent fevers, and history of intravenous drug use [6].

About 50% of patients with IE develop severe complications that require surgical intervention. There is now growing evidence to support that PVE treatment should combine both medical management with antibiotic therapy along with surgical valve replacement. In doing so, the survival rate increases with fewer relapses and lower late-endocarditis related mortality than with antibiotics alone [7]. The patient presented with not only severe conduction abnormalities such as complete heart block which lead to asystolic cardiac arrest, but also with a bioprosthetic heart valve - both of which are class I recommendations for surgical intervention for IE [8].

The general prognosis of patients with PVE depends on a variety of factors such as the virulence of the causative pathogen, extent of any valvular structure involvement, and duration of infection [9]. However, there exists consensus that certain factors such as CHF, non-streptococcal disease, aortic valve involvement, prosthetic valve infection, older age, and abscess formation all predispose the patient to worse outcomes [10]. Of note, our patient had the majority of these poor prognostic factors.

Given the severity of complications from IE, it is imperative to have a high index of suspicion for this diagnosis. The Duke criteria is currently the most sensitive and specific diagnostic schema available for clinicians and is organized into major and minor criteria [7]. Of note, our patient had both sets of major criteria giving him a definitive pathologic diagnosis of IE. However, because there can be other intracardiac masses besides vegetations such as myxomas, clots, rheumatoid nodules, and nonbacterial endocarditis, the clinician should interpret echocardiographic pictures within the clinical context of the patient.

Conclusion

Our patient presented with severe metabolic abnormalities and was found to have enterococcal PVE along with intramyocardial extension of the infection causing an aortic root abscess. This was further complicated by EKG showing complete heart block, which eventually led to asystolic cardiac arrest with ROSC achieved after CPR and epinephrine administration. The patient was stabilized and underwent a re-operative AVR/MVR surgery as well as dual chamber permanent pacemaker generator implantation with normalization of EKG abnormalities and resolution of his symptoms.

This case was relatively unusual due to a variety of factors. The presentation of the patient was atypical as he had a normal physical exam without a murmur or any immunologic or

vascular phenomena. He also did not have a fever nor was he endorsing flu-like symptoms, nausea, fatigue, or weight loss which would be consistent with a subacute IE clinical schema. The patient's laboratory workup, besides a slight leukocytosis, was unremarkable and the patient was not immunosuppressed nor taking any drugs that suppress his immune system. The causative pathogen causing his IE was rare, as enterococcal species represent less than 5% of all cases of PVE. Furthermore, there was no recent history of any genitourinary or gastrointestinal procedures making the source of the initial infection unclear.

Given the fatal complications if not recognized, IE should be suspected early in a patient that presents with conduction abnormalities even in the absence of typical infectious symptoms or laboratory workup. Invasive IE disease is a rare, but fatal etiology of conduction abnormalities and should be suspected even in patients without a history of intravenous drug use. Our patient had profound electrolyte disturbances to explain the cause of his heart block but further investigation revealed enterococcal PVE spreading to affect the conduction system of the heart with an aortic root abscess. In the clinical context of enterococcal bacteremia, prosthetic aortic valve, and heart block, these findings may represent endocarditis with nascent root abscess. Such patients are usually treated with a combination of medical management and surgical intervention and thus, a multi-disciplinary approach among cardiologists, cardiothoracic surgeons, and infectious disease specialists should be taken.

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