

Isolated Native Pulmonic Valve Infective Endocarditis with *Staphylococcus Lugdunensis*

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Abstract Infective endocarditis (IE) is a life-threatening inflammation of the endocardial surface of the heart usually caused by an infection in a patient with a pre-existing heart condition. Isolated pulmonic valve IE cases without risk factors comprise a very small entity of cases. Bacteremic speciation with a coagulase-negative staphylococci, although not as common as its coagulase positive counterpart, carries significant pathogenic potential and deserves regard. *Staphylococcus lugdunensis* commonly lives on a person's skin that is typically harmless when it remains outside the body but associated with high morbidity and mortality when present in the bloodstream. Our case describes a patient with IE of the pulmonic valve without risk factors cultured with the virulent *Staphylococcus lugdunensis*.

Keywords: *infective endocarditis, staphylococcus lugdunensis, pulmonic valve, native valve*

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1. Introduction

Infective endocarditis (IE) attributable to common pathogens and right or left-heart predilection with associated risk factors is well known. However, isolated right-sided infective endocarditis without risk factors cultured by an uncommon bacterial speciation is less reported. We describe a case of infective endocarditis of the pulmonic valve in the absence of risk factors due to a coagulase-negative staphylococci shown to have a fatal outcome. The aim of this case report is to recognize that isolated right sided infective endocarditis occurs in patients in the absence of risk factors and to distinguish that *Staphylococcus lugdunensis* can be associated with high pathogenicity linked with high morbidity and mortality.

2. Case Presentation

A 72-year-old male presented with large volume bright red blood per rectum complicated by septic shock with positive blood cultures. Medical history is significant for multiple comorbidities consisting of persistent atrial fibrillation on anticoagulation, congestive heart failure, coronary artery disease with prior CABG, diabetes mellitus, hypothyroidism, hypertension, and history of a diverticular bleed ten years ago. On initial physical exam, the patient appeared chronically ill, pale, irregularly irregular, and 2/6 systolic murmur. The gastrointestinal

bleed was suspected to be of diverticular source complicated by anticoagulation. Patient was admitted to the intensive care unit and started on pantoprazole and octreotide infusion and transfused with packed red blood cells. Urgent endoscopy revealed no active bleed with erosive duodenitis and colonoscopy revealed extensive diverticulosis. Further work up on abdominal CT angiogram revealed no active bleed with no plans for invasive interventions. Patient remained hypotensive despite adequate blood product repletion. Blood cultures returned positive for gram positive cocci with initiation of empiric intravenous antibiotics. Transthoracic echocardiography (TTE) revealing moderately thickened pulmonic leaflets with vegetation present on the pulmonic valve evident in [Figure 1](#). Vegetation measured 1.55 x 1.32 cm at the pulmonic annulus with an area of 1.62 cm². No perivalvular echolucent area within the valvular annulus or adjacent myocardial structures was noted in the setting of valvular infection, normally consistent with abscess. Other findings on TTE revealed reduced ejection fraction 31-35% and elevated right ventricular systolic pressures consistent with pulmonary hypertension. During his hospital duration, patient was afebrile without leukocytosis since admission. Clinical course was complicated by septic shock requiring pressor support. Due to the patient's unstable status, transesophageal echocardiogram was not performed. Patient had no history of injection drug use, presence of a cardiac implantable electronic device (CIED) or other intravascular device, or cardiac anomaly. Patient progressively decompensated with signs and symptoms of fluid overload with anasarca, bilateral pleural effusions,

elevated jugular venous pressure, and dilated inferior vena cava displaying florid heart failure treated with aggressive diuresis. Pulmonary vascular congestion was evident on the chest x-ray. Concern for low output heart failure prompted inotropic support. Blood cultures returned revealing growth of pan-sensitive *Staphylococcus lugdunensis*. The presumed source of this bacteremia was thought to be due to bacterial translocation from the gut. The patient was deemed hemodynamically unstable for surgical intervention. Measures were taken to wean pressor and inotropic support although the patient's mean arterial pressure and systolic blood pressure did not tolerate. The patient progressively decompensated with worsening volume status, renal function, and increasing pressor/inotropic support. Eventually succumbing to his chronically ill state, he went into cardiac arrest without return of spontaneous circulation and echocardiogram showing cardiac standstill. Cause of death attributable to cardiogenic/septic shock and biventricular heart failure.



Figure 1. TTE parasternal short axis: RV outflow tract view revealing moderately thickened pulmonic leaflets with vegetation present on the pulmonic valve. Vegetation measures at 1.55 x 1.32 cm at the pulmonic annulus with an area of 1.62 cm²

3. Discussion

Infective endocarditis primarily affects those with underlying structural heart disease with a predilection to the left side of the heart or in those with IV drug use with right side preference [1]. Awareness must be made as in this case of endocarditis isolated to the pulmonic valve without predisposing risk factors [2]. While *Staphylococcus aureus* is the typical culprit of right-sided endocarditis, *Staphylococcus lugdunensis* can be just as virulent as its counterpart and should not be discounted as probable contaminants [3,4]. Treating endocarditis requires a multidisciplinary approach and favorable outcomes warrant prompt diagnosis and management, nevertheless even with the proper clinical course it can carry an ominous prognosis as with our patient. This case illustrates a rare case of isolated native pulmonic valve IE in a patient with no predisposing risk factors cultured with a highly destructive coagulase-negative staphylococci associated with high mortality.

4. Conclusion

Infective endocarditis is an infection that enters the blood stream and results in the adhesion of bacteria to valvular surfaces as a vegetation or propagates as systemic emboli. Bacteria gravitate to predisposing structural abnormalities of the cardiac valves. Cases of IE of native valves without risk factors or structural abnormalities are rarer with limited publications. *Staphylococcus lugdunensis*, a component of normal skin flora, can range from a harmless skin colonizer to a life-threatening, invasive pathogen as with infective endocarditis. This organism's virulence is due to the ability to form a protein-based biofilm allowing it to attach to tissue and prosthetic surfaces and protection from antibiotics and host immunity [5]. Pulmonary valve infective endocarditis accounts for 1.5 – 2% of infective endocarditis cases [6]. Data is limited due to very few case reports. Owing to the scarcity of *Staphylococcus lugdunensis* causing IE, the exact prevalence of infective endocarditis with staphylococcus *lugdunensis* is not known. Few literature reviews and case reports have been found with clinical presentations of infective endocarditis with association to the bacterial pathogen *Staphylococcus lugdunensis* and even less isolated to a native pulmonic valve. The consensus is clear in regard to clinical outcomes of IE with staphylococcus *lugdunensis*, that it should not be considered contamination and is associated with high morbidity and mortality [7]. This case exemplifies that IE can occur in patients in the absence of risk factors, isolated to the right side of the heart, and with a bacterial pathogen that is an uncommon microorganism associated with IE although linked to high virulence when detected.

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