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# Case report Pulmonic valve infective endocarditis from staph lugdunensis

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# ABSTRACT

Pulmonic valve endocarditis is an extremely rare entity. Only 1.1% of all cases of all infective endocarditis affect the pulmonic valve. Furthermore, it is even more uncommon for such a disease process to occur in a young and healthy individual without risk factors. It takes a unique case of circumstances to have pulmonic valve infective endocarditis to occur. Staph lugdunensis is one uncommonly isolated organism that has the ability to cause infective endocarditis, who developed isolated pulmonic valve endocarditis caused by Staphylococcus lugdunensis (S. lugdunensis). Based upon our literature review, only 1 other case of S. lugdunensis endocarditis affective the pulmonic valve has been reported.

#### 1. Introduction

Pulmonic valve endocarditis is a rare entity, accounting for approximately 1.1% of all cases of infective endocarditis [1]. The vast majority of cases causing right sided endocarditis are related to IV drug use. Furthermore, right sided endocarditis is more prevalent in individuals with central venous catheter placement, pacemaker implantations, pre-existing valvular disease or congenital cardiac defects. Based on a previous review in 2008, only 90 total cases of isolated pulmonic valve endocarditis had been previously recorded [2]. The vast majority of these cases have one of the above risk factors presents. It is extremely uncommon for an individual to present with this disease process without the aforementioned risk factors. S. lugdunensis is a coagulase negative staph organism that can cause destructive endocarditis at a much higher ratio than other coagulase negative organisms [3]. Although not commonly recognized as a highly pathogenic organism, when isolated, S lugdunensis should be considered as an organism which has high levels of pathogenicity and one which can cause high morbidity and mortality. Further, when destructive valve infective endocarditis is suspected, S. lugdunensis, along with the more commonly suspected Staph aureus, should be considered.

# 1.1. Case

A 20-year-old man with a past medical history of Blount's Disease with orthosis which had been recently removed arrived to the emergency department (ED) complaining of weeklong fever, cough, and headache. His vitals revealed T 100.9F, and labs revealed WBC 6.4 K/ mm3. His physical exam was unremarkable. Chest X-ray showed slight thickening of the minor fissure, suggesting possible inflammation. He was diagnosed with atypical pneumonia and discharged on azithromycin. The next day he was recalled to the hospital after 2 bottles of blood cultures returned positive for pan-sensitive Staphylococcus lugdunensis. He returned to the ED. This time, however he presented with fever, shortness of breath, and convulsions in the ED. His vitals revealed T 104.9F, BP 118/58, P 128, RR 22, SpO2 94% on room air. His WBC was now elevated to 19.3 K/mm3 with neutrophilic predominance and lactic acid was also elevated to 3.64 mmol/L. Physical was remained unremarkable. Chest CT revealed right lower lobe infiltrate, which was presumed to be pneumonia. His repeated blood cultures also returned positive for S. lugdunensis. The presumed source of this bacteremia was his healing wounds from recently removed orthosis. Given concern for possible infective endocarditis, echocardiograms were ordered. Transthoracic echocardiogram revealed a mobile vegetation on the pulmonic valve (Fig. 1) and transesophageal echocardiogram revealed severe pulmonary insufficiency (Fig. 2) with valve thickening and a vegetation consistent with pulmonic valve endocarditis (Figs. 3 and 4). The patient was started on IV antibiotics but he remained febrile, and dyspneic.

He had previously had non-healing ulcers of the lower extremites, but that did not appear to be the case. A CT scan of the lower extremities still was done, and these did not reveal a source of infection. Next, a chest CT was done and revealed pulmonary nodules in the right and left upper lobes concerning for septic emboli. Cardiothoracic surgery was consulted and the patient underwent pulmonary valve replacement with a 27 mm freestyle stent-less porcine bioprosthesis. He continued antibiotic treatment for total of 6 weeks with vancomycin per infectious diseases recommendations. At follow up visit 2 month later,

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Fig. 1. Transthoracic echocardiogram revealing mass on pulmonic valve.



Fig. 2. Transesophageal echocardiogram revealed severe pulmonary insufficiency.



Fig. 3. Transesophageal Echocardiogram showing vegetation on pulmonic valve.

he was clinically improved, echocardiography revealed functioning bioprosthetic pulmonic valve, and he had resolution of bacterial endocarditis.

## 2. Discussion

Isolated right-sided endocarditis is an uncommon entity alone. Overall, right-sided infective endocarditis accounts for approximately 5% of all endocarditis cases [4]. Furthermore, 90% of right-sided endocarditis affects the tricuspid valve [5]. Pulmonic valve infective endocarditis is a form of right sided infective endocarditis, which accounts for only 1.1% of all episodes of infective endocarditis. There are various beliefs as to the reasons for this low prevalence. Some theories include decreased pressures in the right heart system, higher prevalence of venous blood with suboptimal oxygen content for bacterial organisms, and lesser prevalence of congenital heart defects affective valves on the right side [6]. In children, most documented cases of pulmonic valve endocarditis are associated with anomalous pulmonary valve, while in adults, IV drug use has been cited as a key risk factor.

In the scope of right-sided endocarditis, what predisposes the tricuspid valve as being a more regular nidus, other than more direct contact with the right atrium and more immediate exposure to systemic venous blood. Embedded within this already uncommon entity of pulmonic valve infective endocarditis, is the even more uncommon phenomenon of isolated pulmonic valve infective endocarditis in an individual without known obvious risk factors. Of the less than 100 documented reports of pulmonic valve infective endocarditis, very few describe individuals without IV drug use or congenital abnormality [6]. Of the few that have been described, the patients described had other significant comorbid illnesses. For example, one case described an individual on hemodialysis [7]. Another described a case of an individual following orthotopic liver transplantation [8]. It is extremely atypical to identify an individual with no apparent comorbid conditions, who presents with such a disease process.

Isolates obtained from blood cultures in this case grew species of Staph lugdunensis. This is a coagulase negative staphylococcus which is from the saprophyte category normally identified on human skin and soft tissue. It is extremely rare for coagulase negative staph to cause infections. However the discovery of S. lugdunensis has challenged the belief that most coagulase negative staph are benign. S. lugdunensis was first discovered in 1998, and although rare, it has been isolated in a few different severe infections such as prosthetic valve infections [14].

The unique characteristics of S. lugdunensis in the category of coagulase negative staph is tied to its ability to produce pyrrolidonyl l arylamidases. This allows it to bind fibrinogen with extracellular proteins, resulting in agglutination [15]. S lugdunensis IE is typically very uncommon with one study reporting its occurrence in 1.1% of all cases of infective endocarditis [15]. Because of its pathogenesis, S lugdunensis has an ability to affect native valves more so than its coagulase negative counterparts. It is able to cause infection similar to that of staph aureus including characteristics such as difficult traditional response to antibiotics, valve destruction, abscess formation, and high need for surgical involvement. Approximately 51% of individuals with S. lugdunensis IE affecting the pulmonic valve has been described in the literature [7].

Right sided infective endocarditis is an uncommon entity which carries high morbidity and mortality. Clinical presentation can initially be subacute, and any delay in diagnosis can lead to multiple sequela of the disease process. These patients may initially present asymptomatic. However, as disease the disease process begins to develop, these individuals may manifest with a nonspecific febrile illness. They may present with generalized fatigue, lethargy, malaise, and myalgias. It is however, when more systemic symptoms began to develop, that signs of advancing disease are noted. For example, once an individual begins to experience shortness of breath or dyspnea on exertion, the clinician may be concerned about septic emboli to the lungs. If the patient has symptoms of peripheral edema, it may be possible that right sided valve destruction may have occurred, and be contributing to regurgitation of blood from the right heart [9]. Further contributing to the difficulty in diagnosis of right sided endocarditis is the commonly absent heart murmurs. It is important to evaluate all potential aspects included in the modified Duke's Criteria to assist in making a clinical diagnosis. These criteria include both major and minor criteria. Major criteria include positive blood cultures with a typical organism, evidence of endocardial involvement, or new valvular regurgitation. Minor criteria include predisposing heart conditions or IV drug use, fever, vascular phenomena, immunologic phenomena, positive blood culture which does not meet major criteria [10].

Diagnosis of right-sided endocarditis oftentimes becomes dependent



Fig. 4. Transesophageal echocardiogram confirming presents of pulmonic valve vegetation.

on echocardiography. Although transthoracic echocardiogram is often used as an initial modality in those whom right-sided infective endocarditis is suspected, transesophageal echocardiography is more sensitive and specific in identifying this disease process. Echocardiography helps to assess endocardial and valvular structure, as well as analyze for any questionable vegetations or lesions [11].

Treatment of left sided endocarditis has a set of guidelines given the commonality with which it occurs. It is less certain, which cases of right sided endocarditis may require medical versus surgical treatment, although some generally accepted indications have been identified. Such indications include, continued sepsis despite treatment, decompensated right heart failure, paravalvular abscess [12]. Other areas which may support surgical approach include vegetation > 10mm with fever, or outright vegetation > 20mm [13]. However, when approaching pulmonic valve endocarditis, given its rarity, there are no such treatment guidelines. Individuals who present with pulmonic valve endocarditis should be monitored closely and for now, the criteria which are used for treatment of tricuspid valve lesions should serve as a guideline for treating those with pulmonic valve endocarditis. However, this case does highlight a need for developing a standardized approach towards addressing this phenomenon.

## 3. Conclusion

While right-sided endocarditis is typically associated with IV drug use, it can occur in the absence of predisposing factors. Staphylococcus is the major cause of right-sided infective endocarditis [16]. While we typically think of S. aureus specifically, S. lugdunensis is a coagulasenegative species that is as virulent as S. aureus. It is able to directly bind to von Willebrand factor, and thus overcome shear-stress to adhere to cardiac valves, a unique feature it shares with S. aureus and could explain its increased virulence [17].

In a review of the literature, between 6.3 and 27% of patients with blood cultures positive for S. lugdunensis were found to have infective endocarditis, and in one study, the prevalence was 25% for patients that had two positive blood cultures [17]. Two positive blood cultures for S.

lugdunensis thus warrants a workup for infective endocarditis. Though S. lugdunensis is not an organism considered by the Modified Duke Criteria, an argument can thus be made that it should be. One-year mortality for S. lugdunensis bacteremia and infective endocarditis are high, 25.7% and 36% respectively.

Our patient's course reaffirms the volatility of S. lugdunensis and demonstrates the necessity for aggressive treatment even in a young, immunocompetent patient.

#### Disclosures

None to report.

# Informed consent

Obtained.

#### **Conflicts of interest**

None.

#### **Privacy measures**

Obtained to ensure patient privacy.

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#### References

- R.S. Cassling, W.C. Rogler, B.M. Mcmanus, Isolated pulmonic valve infective endocarditis: a diagnostically elusive entity, Am. Heart J. 109 (1985) 558–567.
- [2] N. Katsufumi, N. Osamu, S.N. Dean, Pulmonary valve endocarditis caused by right ventricular outflow obstruction in association with sinus of valsalva aneurysm: a case report, J. Cardiothorac. Surg. 3 (2008) 46.
- [3] J. Etienne, Y. Brun, J. Fleurette, Staphylococcus lugdunensis endocarditis, J. Clin. Pathol. 42 (8) (1989) 892–893 PMID: 2768531.

- [4] P. Chan, J.D. Ogilby, B. Segal, Tricuspid valve endocarditis, Am. Heart J. 117 (1989) 1140–1146.
- [5] W.C. Roberts, N.A. Buchbinder, Right-sided valve endocarditis: a clinicopathological study of twelve necropsy patients, Am. J. Med. 53 (1972) 7–19.
- [6] F.B. Ramadan, D.S. Beanlands, I.G. Burwash, Isolated pulmonic valve endocarditis in healthy hearts: a case report and review of the literature, Can. J. Cardiol. 16 (2000) 1282–1288.
- [7] S. Kamaraju, K. Nelson, D.N. Williams, W. Ayenew, K.S. Modi, Staphylococcus lugdunensis pulmonary valve endocarditis in a patient on chronic hemodialysis, Am. J. Nephrol. 19 (1999) 605–608.
- [8] C.J. Hearn, N.G. Smedira, Pulmonic valve endocarditis after orthotopic liver transplantation liver, Transpl Surg 5 (1999) 456–467.
- [9] C. Harris, B. Croce, S. Munkholm-Larsen, Tricuspid valve disease, Ann. Cardiothorac. Surg. 6 (3) (2017) 294.
- [10] D.T. Durack, A.S. Lukes, D.K. Bright, et al., New criteria for diagnosis of infective endocarditis: utilization of specific echocardiographic findings, Am. J. Med. 96 (1994) 200–209.
- [11] J.A. San Roman, I. Vilacosta, J. Lopez, A. Revilla, R. Arnold, T. Sevilla, M.J. Rollan,

Role of transthoracic and transesophageal echocardiography in right-sided endocarditis: one echocardiographic modality does not fit all, J. Am. Soc. Echocardiogr. 25 (2012) 807–814.

- [12] R. Moss, B. Munt, Injection drug use and right sided endocarditis, Heart 89 (5) (2003) 577–581.
- [13] S. Hecht, M. Berger, Right-sided endocarditis in intravenous drug users. Prognostic features in 102 episodes, Ann. Intern. Med. 117 (1992) 560–566.
- [14] C. Whitener, G.M. Caputo, M.R. Weitekamp, et al., Endocarditis due to coagulasenegative staphylococci: microbiologic, epidemiologic and clinical considerations, Infect Dis Clin North 7 (1993) 81–96.
- [15] I. Anguera, A. Del Río, J.M. Miró, et al., Staphylococcus lugdunensis infective endocarditis: description of 10 cases and analysis of native valve, prosthetic valve, and pacemaker lead endocarditis clinical profiles, Heart 91 (2005) e10.
- [16] K. Akinosoglou, E. Apostolakis, M. Marangos, G. Pasvol, Native valve right sided infective endocarditis, Eur. J. Intern. Med. 24 (6) (2013) 510–519.
- [17] L. Liesenborghs, M. Peetermans, J. Claes, et al., Shear-resistant binding to von Willebrand factor Allows Staphylococcus lugdunensis to adhere to the cardiac valves and initiate endocarditis, J. Infect. Dis. 213 (7) (2016) 1148–1156.