


CASE REPORT



Isolated pulmonary valve endocarditis: truth or myth?

Siamak M. Seraj , Evanpaul Gill and Simranjit Sekhon

Department of Internal Medicine, San Joaquin General Hospital, French Camp, CA, USA

ABSTRACT

Pulmonary valve (PV) infective endocarditis (IE) is an extremely rare disease that involves normal as well as abnormal valves. This condition mostly occurs in patients with underlying predisposing factors. It could be missed if patients do not present with typical features of right-sided endocarditis or in the absence of classic risk factors. The case presented here did not have any known risk factors for IE until surgery and presented mainly with fever, weight loss, and musculoskeletal symptoms.

ARTICLE HISTORY

Received 3 April 2017
Accepted 29 August 2017

KEYWORDS

Pulmonic valve; infective endocarditis; subpulmonic valve stenosis; musculoskeletal manifestation; *Streptococcus sanguinus*; vegetation

1. Case report

A 61-year-old African-American male came to the emergency department with a history of back and hip pain, night sweats, and decreased appetite coupled with an unintentional weight loss of 40 lbs. He also reported feeling fatigued and had generalized malaise. His history was also significant for hemoptysis and pleuritic chest pain.

Five months ago, before the current admission, he had presented to our hospital for similar symptoms. During that time, he underwent a Computed Tomography scan (CT) of the chest that reportedly showed right lower lobe pleural thickening with hilar lymphadenopathy. The patient was then prescribed oral antibiotics for possible community acquired pneumonia and was subsequently discharged. Unfortunately, the patient did not feel better and came to the hospital again for the current admission.



Past medical history was significant for a period of incarceration during which time he had a PPD placed, which was negative. He also endorsed an eight pack-year history of smoking tobacco and a history of remote IV drug use for two years.

On admission, the patient's vitals were remarkable for tachycardia at 106 beats per minute and an oxygen saturation of 98% on 2 liters. On physical exam, a grade IV/VI holosystolic murmur was heard in all cardiac foci. Lungs were clear to auscultation bilaterally. The abdomen was tender to palpation diffusely with normoactive bowel sounds. There was significant tenderness to palpation in the lumbar spine with a positive straight leg test. Digital clubbing of the fingernails was noted

bilaterally. Pertinent labs showed an elevation of white blood cell counts at 14.5 thousand cells per milliliter and sodium of 128. Radiological imaging included a CT scan of the chest that showed multiple opacities bilaterally, more prominent on the right (Figure 1). Patient was then admitted with a diagnosis of right upper lung community acquired pneumonia, with a high suspicion for tuberculosis. Eventually, tuberculosis was ruled out with three negative acid-fast bacillus sputum samples, negative quantiferon screen, and negative nucleic acid amplification test. Blood cultures came back positive for streptococcus sanguinus. Following positive blood cultures, the patient underwent transthoracic echocardiography (TTE) which confirmed the presence of a large and mobile pulmonic valve vegetation, as well as moderate to severe tricuspid regurgitation (Figure 2). Infectious disease consult was obtained and he was started on ceftriaxone 2 grams intravenously per day. The treatment was continued for a total of four weeks. Due to the large size of the vegetation and severe pulmonary valve insufficiency, the patient underwent valve replacement (Figure 3).

2. Discussion

Pulmonic valve (PV) infective endocarditis (IE) is extremely rare, accounting for 1.5% to 2% of the total cases of IE [1,2]. Most data on PV IE is limited to a few small case series and case reports, some published more than 25 years ago [3–5]. Infection may only affect the pulmonic valve or may concomitantly involve the mitral or aortic valve [3,6,7]. In

CONTACT Siamak M. Seraj  sseraj@sjgh.org  Department of Internal Medicine, San Joaquin General Hospital, 500 W. Hospital Rd, French Camp, CA, 95231, USA

© 2017 The Author(s). Published by Informa UK Limited, trading as Taylor & Francis Group.

This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.



Figure 1. CT scan of chest without contrast showing multiple bilateral opacities.

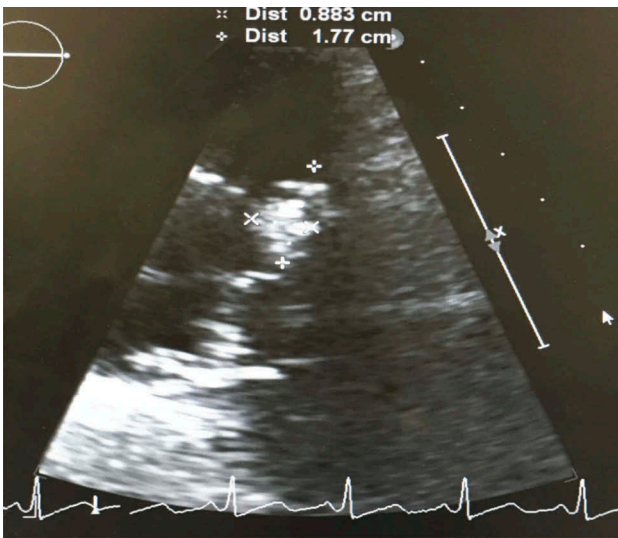


Figure 2. Transthoracic echocardiogram, short axis of aorta view.

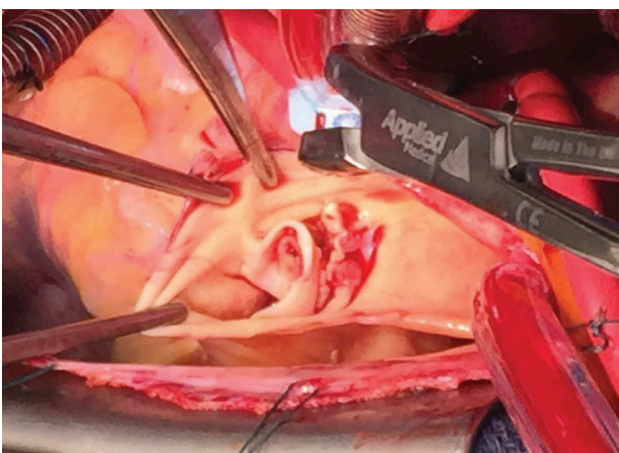


Figure 3. Subpulmonic valve stenosis and PV vegetation during open heart surgery.

addition, PV IE has been associated with both structurally normal and abnormal valves [2,3,6–8]. The rarity of PV IE has been attributed to multiple factors including lower pressure within the right heart, lower incidence of congenital malformations or acquired

valvular abnormalities, lower oxygen content of venous blood and the differences in the endothelial lining and vascularization of the valve [2,9].

The most common risk factor for PV endocarditis is intravenous (IV) drug abuse. However, multiple other risk factors have been reported including male gender, central venous catheters, alcoholism, dental extraction, gonorrhoea, liver or renal transplantation, bowel surgery, colonic angiodysplasia, and congenital heart disease (CHD) [2,9,10]. Immunosuppressive therapy has also recently been proposed as a risk factor [11].

In general, right-sided endocarditis presents with persistent fever and pulmonary symptoms (cough, dyspnea, and hemoptysis), which are mainly due to septic emboli. The pulmonic regurgitant murmur is often a late feature [12]. It has been reported that diagnostic yield of TTE could be as high as 91% [2]. Transesophageal echocardiography (TEE) of PV could be challenging due to its position, which is the most anterior and farthest from the TEE probe in comparison to other heart valves [11]. Given the technical limitations of standard imaging modalities in the diagnosis of pulmonic valve IE, even PET-CT imaging is proposed to further guide management of patients with pulmonic valve endocarditis [13]. The most common microorganisms reported in PV IE are *Staphylococcus aureus*, coagulase negative *Staphylococci*, and group B *Streptococci* [2,11,12].

The case presented here was diagnosed about five months after the first presentation of cough and hemoptysis to ED. Our patient had a remote history of intravenous drug use and no history of alcohol abuse. He had a significant IV/VI holosystolic murmur heard in all cardiac foci which the patient referred to as a congenital finding. Initially, it was unclear if the underlying cause of our patient's symptoms and CT findings were due to tuberculosis or multilobar pneumonia. In addition, we had to exclude *Coccidioidomycosis* since this infection is endemic in our region. Presence of the congenital heart murmur masked the typical pulmonic regurgitant murmur in PV IE and delayed the diagnosis even further. We were only able to make the final diagnosis after the patient was found to have positive blood cultures of *Streptococcus Sanguinus*, which is a member of the Viridans *Streptococcus* group. TTE was obtained immediately and showed 0.88 by 1.77 cm mobile vegetation on the pulmonic valve leaflets with moderate pulmonic valvular regurgitation, which was confirmed with color flow doppler.

Moreover, what also made the diagnosis of PV IE very challenging in this case was the fact that our patient presented mainly with B symptoms like fever, malaise, and severe pain in the lower back and hips. These symptoms, associated with bilateral small opacities in lungs, made it difficult to exclude the possibility of malignancy versus mycobacterial/fungal infection. It is important to realize that IE may initially present with musculoskeletal symptoms, with one third to one half of

patients presenting with this symptom [14]. MRI of the hip and lumbar spine was obtained later which showed a completely normal exam. The etiology of these symptoms is still not completely understood.

Our patient finished four weeks of antibiotic therapy and underwent open heart surgery for removal of vegetation and valve repair. During surgery, patient was further diagnosed with subpulmonic valve stenosis which was likely the main predisposing factor for development of PV IE. Patient's symptoms, including musculoskeletal involvement, were entirely resolved after the treatment.

In conclusion, pulmonic valve endocarditis is an extremely rare infection that may be missed if patients do not have blatant risk factors or typical features of right-sided endocarditis. In this case, patient did not have any known risk factors for IE until surgery and presented mainly with fever, weight loss, and musculoskeletal symptoms. Therefore, an isolated PV IE without any risk factors still remains quite challenging based on our review of the literature and the current case.

Acknowledgements

We thank Drs. Charlie Whitcomb and Ramesh Dharwat for assistance with preparation of this article.

Disclosure statement

No potential conflict of interest was reported by the authors.

ORCID

Siamak M. Seraj  <http://orcid.org/0000-0001-8439-4825>

References

- [1] Lepeschkin E. On the relation between the site of valvular involvement in endocarditis and the blood pressure resting on the valve. *Am J Med Sci.* 1952;224(3):318–319.
- [2] Ramadan FB, Beanlands DS, Burwash IG. Isolated pulmonic valve endocarditis in healthy hearts: a case report and review of the literature. *Can J Cardiol.* 2000;16(10):1282–1288.
- [3] Cassling RS, Rogler WC, McManus BM. Isolated pulmonic valve infective endocarditis: a diagnostically elusive entity. *Am Heart J.* 1985;109(3 Pt 1):558–567.
- [4] Cremieux AC, Witchitz S, Malergue MC, et al. Clinical and echocardiographic observations in pulmonary valve endocarditis. *Am J Cardiol.* 1985;56(10):610–613.
- [5] Bolton WD, Fox ER, Winscott JG, et al. Methicillin-resistant *Staphylococcus aureus* pulmonary valve endocarditis. *Am Surg.* 2009;75(12):1265–1266.
- [6] Nakamura K, Satomi G, Sakai T, et al. Clinical and echocardiographic features of pulmonary valve endocarditis. *Circulation.* 1983;67(1):198–204.
- [7] DePace NL, Nestico PF, Iskandrian AS, et al. Acute severe pulmonic valve regurgitation: pathophysiology, diagnosis, and treatment. *Am Heart J.* 1984;108(3 Pt 1):567–573.
- [8] Tariq M, Smego RA, Soofi A, et al. Pulmonic valve endocarditis. *South Med J.* 2003;96(6):621–623.
- [9] Ranjith MP, Rajesh KF, Rajesh G, et al. Isolated pulmonary valve endocarditis: A case report and review of literature. *J Cardiol Cases.* 2013;8(5):161–163.
- [10] Van der Meer JT, Van Wijk W, Thompson J, et al. Efficacy of antibiotic prophylaxis for prevention of native-valve endocarditis. *Lancet.* 1992;339(8786):135–139.
- [11] Miranda WR, Connolly HM, DeSimone DC, et al. Infective endocarditis involving the pulmonary valve. *Am J Cardiol.* 2015;116(12):1928–1931.
- [12] Schroeder RA. Pulmonic valve endocarditis in a normal heart. *J Am Soc Echocardiogr.* 2005;18(2):197–198.
- [13] Zietlow K, Bazemore TC, Parikh KS. Diagnosing pulmonic valve infective endocarditis with positron emission tomography-CT. *BMJ Case Rep.* 2016(2016); doi: 10.1136/bcr-2015-213491
- [14] Llinas L, Harrington T. Musculoskeletal manifestations as the initial presentation of infective endocarditis. *South Med J.* 2005;98(1):127–128.