

## CASE SERIES

# Native pulmonary valve endocarditis requiring pulmonary valve replacement in adulthood: a case series

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## Abstract

Native pulmonary valve endocarditis is a rare phenomenon as native valve endocarditis tends to typically affect the left-sided heart valves. However, the right-sided heart valves can be affected in patients with a history of intravenous drug use, whereby the tricuspid valve is most commonly affected. We present two cases who were diagnosed with native pulmonary valve endocarditis in the absence of congenital heart disease. In the first case, the native pulmonary valve endocarditis was probably a derivative of compounding factors of an enlarged underlying pulmonary artery and staphylococcal bacteraemia. In the second case, a common causal organism of native valve endocarditis following dental treatment and the resultant echocardiography findings was of significant interest. In summary, native pulmonary valve endocarditis is a relatively rare complication in the adult population, especially in the absence of congenital heart disease.

## INTRODUCTION

Native pulmonary valve endocarditis is a rare phenomenon as native valve endocarditis tends to typically affect the left-sided heart valves [1]. However, the right-sided heart valves can be affected in patients with a history of intravenous drug use, whereby the tricuspid valve is most commonly affected [1]. Valvular endocarditis has a wide clinical spectrum, and the clinical presentation is dependent on a multitude of factors [1]. The sequelae of endocarditis are numerous and indications for surgery are well established according to current guidelines as outlined by European Society of Cardiology and American Heart Association [2, 3]. Complications of native pulmonary valve endocarditis include right heart failure secondary to volume overload due to pulmonary regurgitation. Upon review of relevant literature, there are numerous reports describing the association of native pulmonary valve endocarditis in the setting of congenital heart disease including pulmonary stenosis and secundum atrial septal defect [4], congenital VSD [5, 6], bicuspid aortic valve with patent ductus [7] and clinically silent patent ductus arteriosus [8] but rarely described in its absence. An association

of septic pulmonary embolism associated with right-sided infective endocarditis (RSIE) is well-established [9]. Furthermore, septic pulmonary infarction can occur due to embolism from pulmonary valve vegetations. There is variance in the clinical spectrum of these patients, and they may present fever, dyspnoea, chest pain, cough and haemoptysis as reported by one study analyzing the presenting features and clinical course of patients diagnosed with septic pulmonary embolism [10]. Moreover, septic pulmonary embolism may be present in the absence of RSIE and classified according to the embolic source [11]. Native pulmonary valve endocarditis has been described to occur in a few case reports in the absence of congenital heart disease [12–14]. We present two cases who presented to our institution with cardiac septic pulmonary embolism secondary to RSIE from native pulmonary valve endocarditis.

## CASE DISCUSSION

We present two cases who were diagnosed with native pulmonary valve endocarditis in the absence of congenital heart disease.

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The first patient was 65-year-old gentleman who had presented to our institution with non-specific lethargy, increasing dyspnoea and fever. He had a vague history of an enlarged pulmonary artery diagnosed aged 16 and was under follow-up at another institution. There was no mention of any congenital valvular heart disease. He had no significant co-morbidities. Subsequent investigations confirmed that *Staphylococcus aureus* had been grown from peripheral blood cultures and the patient was placed on intravenous antibiotics. Transthoracic echocardiography had confirmed severe pulmonary regurgitation secondary to large vegetations and a dilated, severely impaired right ventricle which was volume loaded and no other significant valve disease. Coronary angiography demonstrated no flow limiting coronary artery disease. Pre-operative computer tomography imaging had demonstrated several small lung abscesses consistent with a diagnosis of septic pulmonary embolism. The patient had become septic with ongoing pyrexia and organ dysfunction with rise in creatinine to 195 pre-operatively and in the liver function tests with a bilirubin of 31. He had decompensated with evidence of fluid overload requiring diuresis but had become tachycardic and his rhythm changed to atrial fibrillation with a fast ventricular response and the patient had become oliguric. Due to the deterioration in the condition of the patient, a decision was made to operate on an urgent basis and an intra-aortic balloon pump was placed pre-operatively. Operative findings were that of a dilated, volume-loaded right heart, large 3-cm vegetations with a completely destroyed native pulmonary valve and an enlarged pulmonary artery. The native valve morphology did not appear entirely normal, and the native leaflet tissue was thickened and myxoid in appearance. The native valve was excised, and tissue thoroughly debrided and sent for culture and further histopathological analysis. A bioprosthetic valve was implanted using interrupted sutures and the arteriotomy was closed. Due to the severely impaired right ventricular function, the patient was rested on cardiopulmonary bypass and gradually weaned off with modest inotropic support and intra-aortic balloon pump support. The post-operative recovery of the patient was prolonged requiring multi-organ support with vasoconstrictors and haemofiltration. Post-operative atrial fibrillation was managed with antiarrhythmic medications. Following a period of stabilization and improvement, the patient was transferred to the care of the cardiology team for continuing antibiotic therapy.

The second case was a 67-year-old gentleman who had presented to a peripheral hospital with fever following dental treatment which had occurred 1 month prior to admission. He had a past medical history of hypertension and no previous history of congenital heart disease. Peripheral blood cultures had been positive for *Streptococcus viridians*. He was commenced on appropriate antibiotic therapy. Transthoracic echocardiography had confirmed vegetations on the pulmonary valve causing

severe regurgitation and vegetations on the posterior leaflet on the mitral valve causing moderate-to-severe regurgitation. Furthermore, he had biventricular dilatation and the other valves were free of disease. Coronary angiography did not demonstrate any flow-limiting coronary artery disease. Pre-operative CT pulmonary angiogram had confirmed the presence of a septic right lower lobe pulmonary embolus and CT abdomen and pelvis had confirmed the presence of a splenic infarct. Due to ongoing pyrexia in the presence of antibiotic therapy and the presence of septic pulmonary embolism and splenic infarction, the patient was offered inpatient surgery. Operative findings were that of a completely destroyed pulmonary valve with vegetations affecting all three leaflets, vegetations affecting the P1 component of the mitral valve causing subtotal destruction of P1. Furthermore, there was septic embolic in the right lower lobe pulmonary artery. The native pulmonary valve was excised, and valve sent for histopathological analysis and culture and replaced with a mechanical prosthesis due to the small size of the annulus. Pulmonary embolectomy was performed to remove the right lower lobe septic pulmonary embolus. The P1 scallop of the mitral valve was excised and then sent for histopathological analysis and culture. The A1/P1 region was then plicated to complete the repair of the native mitral valve. The patient was weaned off cardiopulmonary bypass on modest inotropic support. His post-operative recovery was satisfactory, and he was transferred back to the peripheral hospital for ensuing antibiotic therapy to complete the course duration intended.

The patient with the staphylococcal pulmonary valve endocarditis was critically ill and in multi-organ failure and had a more prolonged intensive care stay, whereas the patient with the streptococcal viridans endocarditis who required two separate valve interventions was in superior condition pre-operatively and had a relatively uneventful and short intensive care stay.

## DISCUSSION

Native pulmonary valve endocarditis in the absence of congenital heart disease is rare in its occurrence but has been previously described in isolated case reports [12–14]. The description of pulmonary valve endocarditis and associated cardiac septic pulmonary embolism has also been described [9]. In our case series, we had two different patients presenting with the same complication from the native pulmonary valve endocarditis, i.e. septic pulmonary embolism but they presented with vague symptoms, and with different organisms affecting their native valve and their pulmonary valve was replaced with prostheses in both patients. Pulmonary valve repair has also been described for native pulmonary valve endocarditis [15]. This case series described the presence of cardiac septic pulmonary embolism in the presence of native pulmonary valve endocarditis and the absence of congenital valvular heart disease. In the first case,

the native pulmonary valve endocarditis was probably a derivative of compounding factors of an enlarged underlying pulmonary artery and staphylococcal bacteraemia. In the second case, a common causal organism of native valve endocarditis following dental treatment and the resultant echocardiography findings was of significant interest. The combination of mitral and pulmonary valve endocarditis has not been previously described. One could interpret this almost as a 'Graham Steell-esque' consequence of mitral valve regurgitant endocarditis, whereby the classical Graham-Steell murmur is functional pulmonary valve regurgitation as a result of long-standing pulmonary hypertension as a consequence of mitral stenosis [16], the mitral valve endocarditis could have spread in a retrograde fashion via the lungs to the pulmonary valve. Furthermore, the right lower lobe septic pulmonary embolus may further support this assumption. Although valve repair techniques for pulmonary valve endocarditis have been previously described, the pathology and destruction of the native valve necessitated replacement in both these cases [15]. In summary, native pulmonary valve endocarditis is relatively rare complication in the adult population, especially in the absence of congenital heart disease. Our relatively small case series demonstrate successful replacement of the infected native pulmonary valves which had caused cardiac septic pulmonary embolism, and the second case describes a previously undescribed clinical association of pulmonary and mitral valve endocarditis, 'Graham Stell-esque' in its occurrence.

## CONFLICT OF INTEREST STATEMENT

None declared.

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