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CASE REPORT
Other

Septic pulmonary emboli in pulmonary valve endocarditis with concurrent ventricular septal defect and coronary artery disease: a case report

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Background Infective endocarditis (IE) is one of the common causes of life-threatening infections. Compared to left-sided endocarditis, right-sided infective endocarditis is rarer, with pulmonary valve endocarditis much rarer than the tricuspid valve. Its diagnosis poses a challenge, owing to its rarity, low index of clinical suspicion, and lack of availability of appropriate diagnostic measures. Risk factors include indwelling central venous catheter, sepsis, intravenous drug use, pacemaker with lead infection, or ventricular septal defect (VSD).

Case summary We describe a case of pulmonary valve endocarditis that led to septic pulmonary emboli in a patient scheduled for elective bypass surgery for triple vessel disease. There was an incidental finding of VSD on echocardiography, which is also a risk factor for pulmonary valve endocarditis owing to the jet of VSD to the pulmonary valve. The patient was given 4 weeks of antibiotics and subsequently underwent coronary artery bypass graft, pulmonary valve replacement, and VSD closure.

Discussion Our case demonstrated the importance of high clinical suspicion and vigilance of diagnosing pulmonary valve endocarditis when dealing with pyrexia of unknown origin in a patient with a congenital VSD as VSD-associated pulmonary valve endocarditis remained a rare disease. Besides, an active search for clinical and radiological signs of pulmonary embolization is necessary in patients with right-sided endocarditis especially those with large and mobile vegetation. A conservative approach or valve repair is recommended for most patients with right sided IE affecting the tricuspid or pulmonary valve.

Keywords Infective endocarditis • Pulmonary valve • Ventricular septal defect • Pulmonary emboli • Case report

ESC Curriculum 4.7 Pulmonary regurgitation • 4.11 Endocarditis • 9.5 Pulmonary thromboembolism

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Highlights/Key Learning points

- Pulmonary valve endocarditis is rare. Thus, high clinical suspicion and vigilance are crucial during echocardiography to accurately diagnose pulmonary valve endocarditis.
- A ventricular septal defect is a risk factor for pulmonary valve endocarditis.
- Septic pulmonary embolus is a well-recognized complication of pulmonary valve endocarditis.
- Pulmonary valve repair preferred as preservation of native pulmonary valve is recommended if clinically feasible.

Introduction

Infective endocarditis (IE) is one of the common causes of life-threatening infections.[1] Compared to left-sided endocarditis, right-sided infective endocarditis is rarer, with pulmonary valve endocarditis much rarer than the tricuspid valve.[1] Its diagnosis poses a challenge, owing to its rarity, low index of clinical suspicion, and lack of availability of appropriate diagnostic measures. Intravenous drug use is often implicated in adult cases of infective endocarditis in the presence of a structurally normal heart.[2] Septic pulmonary embolism is a well-characterized complication of pulmonary valve IE, and the presence of pulmonary infiltrate on the initial chest X-ray should raise clinical suspicion of this complication.[3,4] Overall, the prognosis of pulmonary valve IE is more favourable than left-sided IE, and most can be managed conservatively.[5,6]

Timeline

3 months before admission	Coronary angiogram was done and noted triple vessel disease.
	Echocardiogram noted incidental findings of ventricular septal defect.
Day 0	Electively admitted for CABG and VSD repair.
Day 1	Investigated for pyrexia of unknown origin (PUO). Echocardiogram noted huge mobile mass at pulmonary valve, diagnosed as infective endocarditis.
	Blood culture grew *Streptococcus gordonii*.
Day 2	Computed tomography pulmonary angiogram (CTPA) showed pulmonary emboli.
Day 29	Persistent fever and repeated CTPA and CECT thorax showed pulmonary emboli with consolidation.
	Continued antibiotic for 6 weeks.
Day 49	CABG, pulmonary valve replacement with VSD closure done.
Day 58	Well, discharged home with warfarin.
Day 63	Well and asymptomatic during clinic review. Good wound healing. Echocardiogram showed bioprosthetic pulmonary valve no leakage.

Case report

A 40-year-old gentleman with underlying hypertension and dyslipidemia was admitted for typical angina and shortness of breath. He was diagnosed with non-ST segment elevation acute coronary syndrome. Global registry of acute coronary events score was 48 with 0.6% mortality at 6 months. He then underwent a coronary angiogram which revealed triple vessel disease with left main stem involvement (*Figure 1A–D*). The synergy between percutaneous coronary intervention (PCI) with taxus (SYNTAX) score was 44. Transthoracic echocardiography (TTE) showed an incidental finding of outlet perimembranous ventricular septal defect (VSD, Supplementary material online, Video 1) with left to right shunt. We had discussed the case among the Heart team. In view of high Syntax score with a perimembranous VSD, we decided to subject the patient to elective coronary artery bypass graft (CABG) surgery with VSD repair. He was discharged with aspirin, clopidogrel, rosuvastatin, perindopril, bisoprolol, and pantoprazole.

During readmission for elective CABG and VSD repair, he complained of intermittent fever for the past month despite multiple medical visits. Physical examination revealed a diastolic murmur at the left upper sternal edge. His vital signs were stable, no significant findings on respiratory examination, and there were no peripheral stigmata of infective endocarditis. He was diagnosed with pyrexia of unknown origin with underlying congenital heart disease for further evaluation. He was not taking any illicit drugs and there was no history of recent dental extraction.

Full blood count showed leukocytosis with a white cell count of 14×10^9/L and raised C-reactive protein (CRP) of 95 mg/L. Repeated transthoracic echocardiogram showed a highly mobile mass at the pulmonary valve measuring 2.3 × 1.2 cm (Supplementary material online, Video 2) with free flow pulmonary regurgitation (*Figure 2A* and *B*), the previously identified outlet perimembranous VSD measuring 1.2 cm, Qp:Qs of 1.4, with a left to right shunt was present (*Figure 3A–C*). A computed tomography pulmonary angiogram (CTPA) showed vegetation within the main pulmonary trunk, attaching to the pulmonary valve leaflets, with distal embolization involving bilateral posterobasal segments of the lungs, which has caused septic atelectasis (*Figure 4A* and *B*). Subsequent blood cultures grew *Streptococcus gordonii*. The patient was diagnosed with pulmonary valve endocarditis following Modified Duke's criteria, having fulfilled two major criteria (blood culture and echocardiogram positive for IE) and three minor criteria (predisposing heart condition, fever, and vascular phenomenon). Despite 3 weeks of appropriate antibiotics, he had persistent fever with rising CRP. We then repeated CTPA and contrast enhanced computer tomography (CECT) lung

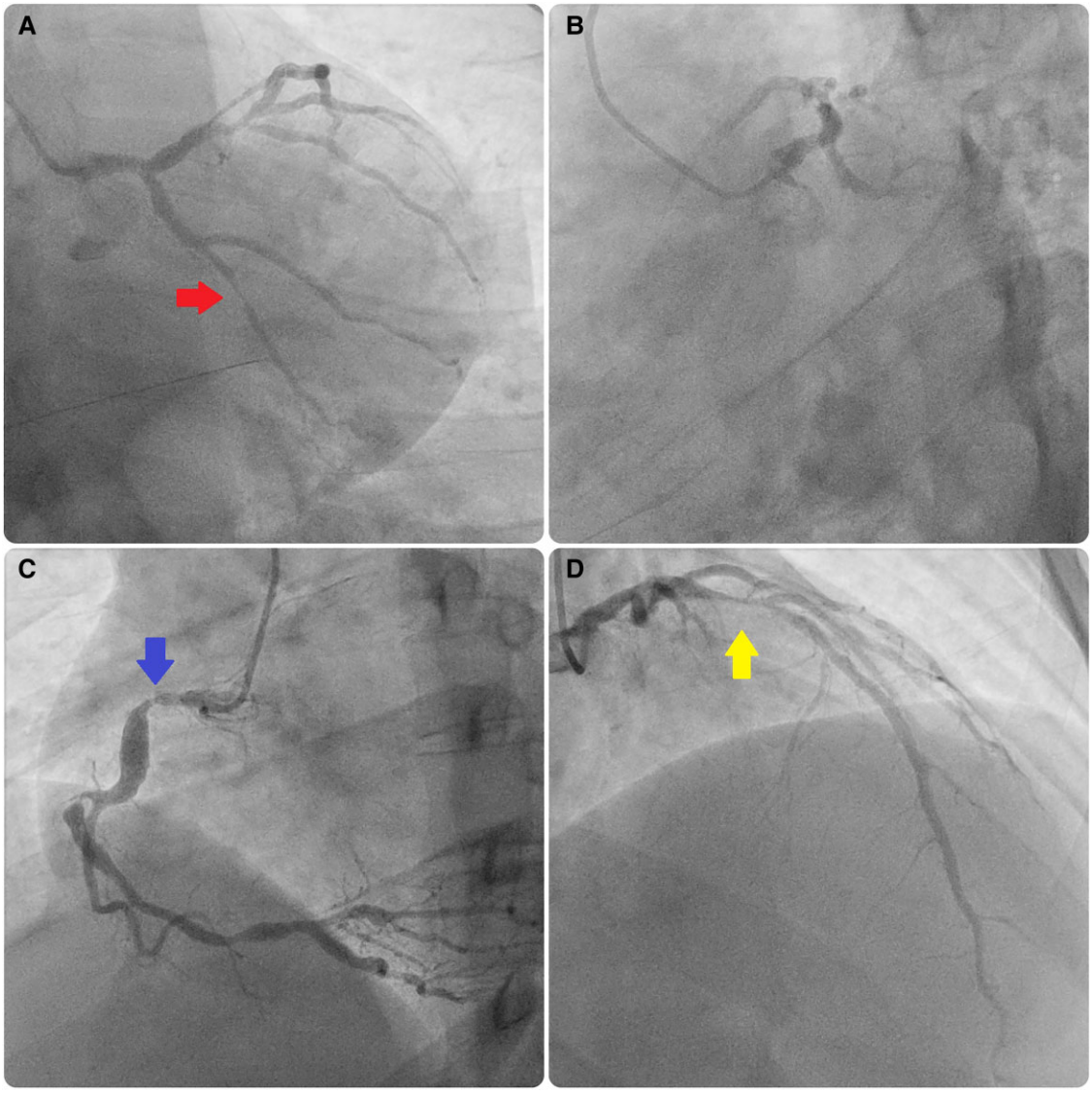

Figure 1 (*A*) Moderate stenosis distal left main with severe stenosis mid left circumflex artery (red arrow). (*B*) Moderate stenosis distal left main. (*C*) Severe stenosis proximal right coronary artery (blue arrow) with ectatic segment. (*D*) Severe stenosis proximal left posterobasal artery at D1 junction (yellow arrow).

to assess possible pulmonary complication and unfortunately; it showed dislodgement of the vegetation from the pulmonary valve into the left posterobasal pulmonary artery, resulting in left basal consolidation and pleural effusion. There were no signs of lung abscess. The repeated TTE showed smaller, but non-resolving 1.0 × 0.8 cm vegetation with persistent free flow pulmonary regurgitation despite 1 month of antibiotic treatment.

He was then treated with total of 6 weeks of intravenous crystalline Penicillin for *S. gordonii*. After being optimized medically, he successfully underwent CABG surgery with pericardial bioprosthetic pulmonary valve replacement (Supplementary material online, Video 3) and VSD closure. He was discharged home 9 days post-operation.

Six weeks post-operation follow-up showed good surgical site wound healing with no sign of systemic infection, and the patient was angina free. A repeated transthoracic echocardiogram 1 month post operation showed a good left ventricular ejection fraction of 55% with an intact bioprosthetic pulmonary valve. His latest transthoracic echocardiogram 1 year post operation showed an intact bioprosthetic pulmonary valve with trivial pulmonary regurgitation with no residual VSD (Supplementary material online, Video 4).

Discussion

Right sided IE is rare, accounting for only 5–10% of all IE cases.[5] Pulmonary valve endocarditis accounts for 2% of all cases of IE, which is 10 times less common than tricuspid valve IE.[3,7] The rarity of pulmonary valve IE can be attributed to several factors. First, since the right heart carries venous blood with lower oxygen content and

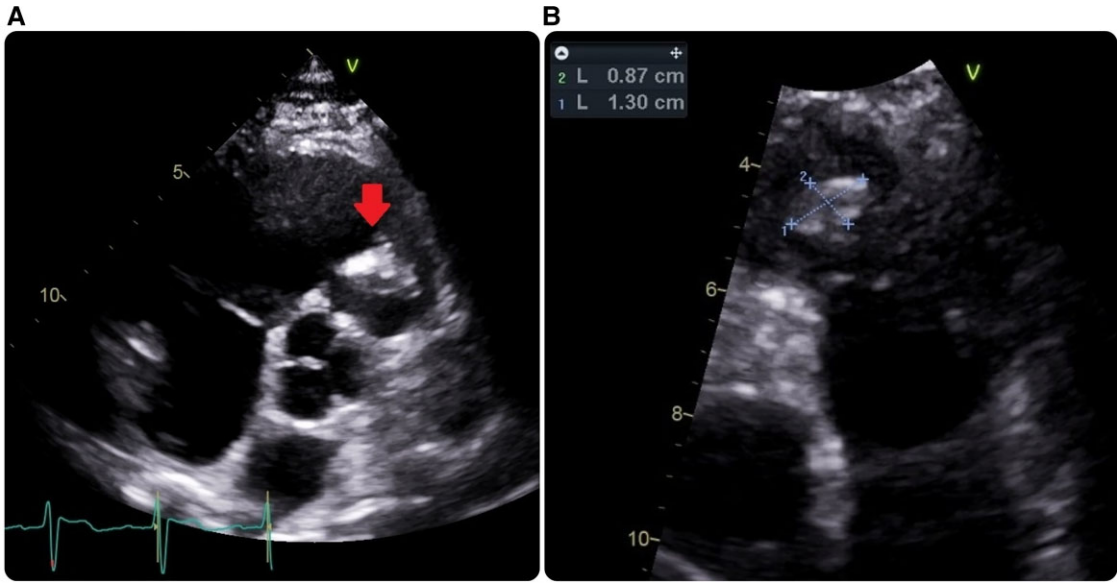

Figure 2 (*A*) Aorta pulmonary artery view showing pulmonary valve vegetation. (*B*) Pulmonary valve vegetation measuring 1.30 × 0.87 cm.

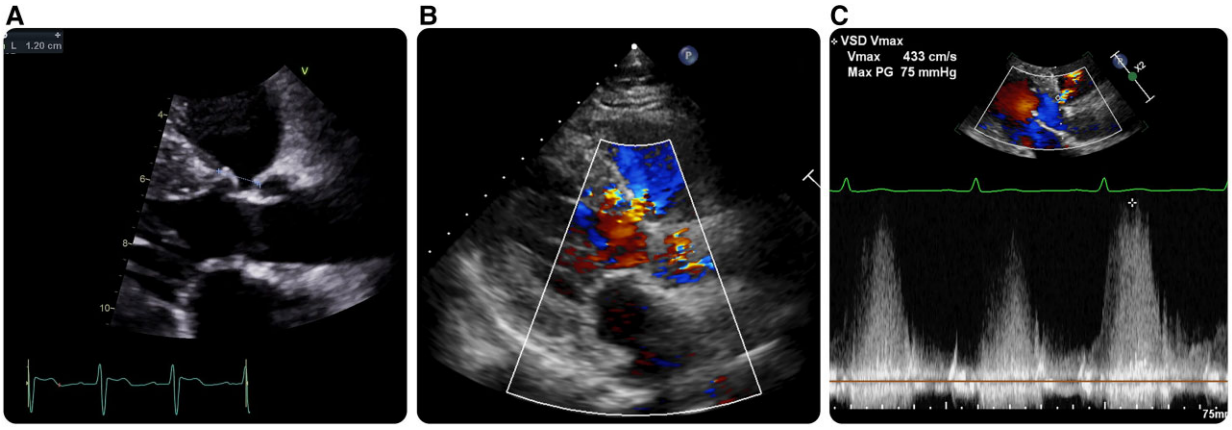

Figure 3 (*A*) Ventricular septal defect measuring 1.20 cm from parasternal long axis view. (*B*) Colour Doppler through ventricular septal defect. (*C*) Continuous wave Doppler through ventricular septal defect.

lower pressure gradient, protecting it from the development of IE.[2] In addition, the pulmonary valve has a lesser incidence of congenital malformation, making it a less popular site for IE.[2] Patients with congenital heart defects with left to right shunts are prone for right sided IE, owing to the high pressure gradient between left and right hearts.[3] The resultant endocardial damage on the right heart predisposes the patient to IE.[3] On the other hand, occurrence in adults with healthy hearts can be due to intravenous drug use, indwelling catheters, sepsis, and pacemaker with lead infection, all of which have become more prevalent in the recent years.[2] Among them, intravenous drug use is the most common cause of pulmonary valve IE. In fact, tricuspid valve abnormalities, such as tricuspid regurgitation, prolapse, and thickening, are increasingly found in patients with repeated drug injections.[6] Our patient has a history of untreated VSD, which is a predisposing factor for pulmonary valve IE. The turbulent jet of blood through the defect may repetitively strike the pulmonary valve, causing endocardial damage and making it vulnerable to IE.[3,8,9]

Patients with right-sided infective endocarditis may present with fever, respiratory symptoms, bacteremia, or pneumonia, especially with lower lobes involvement due to septic emboli.[3] It had been suggested that patients with multiple lung cavitary lesions with fever should be worked up for right-sided IE with trans-oesophageal echocardiography even in the absence of significant risk factors.[10]

An active search for embolization is always recommended in patients with endocarditis, especially in patients with large (>10 mm) and mobile vegetations, just like the case we described.[3,11] Pulmonary septic embolization is a common complication of right-sided IE, and computed tomography scan is the imaging

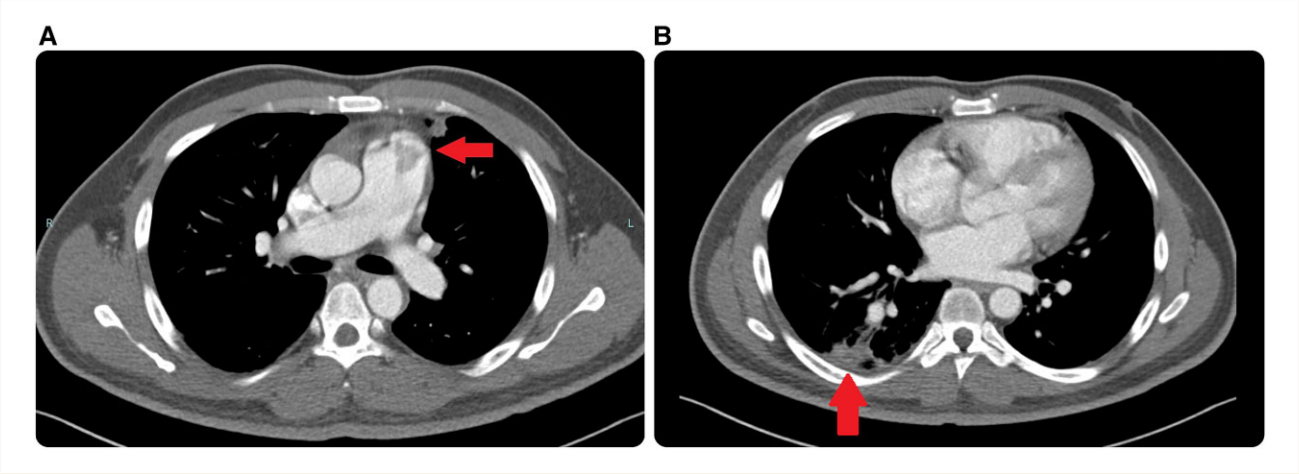

Figure 4 (*A*) Computed tomography pulmonary angiogram (CTPA) showed vegetation within the main pulmonary trunk, attaching to the pulmonary valve leaflets (red arrow), (*B*) Pulmonary consolidation at posterobasal segment.

modality of choice.[3] In a retrospective analysis conducted by Hecht and Berger[4] in 1992, they found approximately 55% of patients with chest radiograph infiltration on presentation consistent with pulmonary septic emboli. Our patient had embolization of vegetation into the main pulmonary trunk, as well as in subsegmental branches, resulting in septic atelectasis, pneumonia, and pleural effusion. He also had continued pulmonary septic emboli despite a long course of appropriate antibiotics.

Owing to the rarity of right-sided IE, there is much less data and guidelines than left-sided IE.[5,6,12] In general, isolated right-sided IE carries a better prognosis than its counterpart.[5] An intravenous antibiotic is the mainstay of treatment in right-sided IE, and they typically respond to 4–6 weeks course of parenteral antibiotics.[6,8,13] Surgical intervention in right sided IE is only indicated when it is caused by microorganisms that are difficult to eradicate, e.g. fungus, persistent bacteremia more than 7 days despite adequate antimicrobial therapy, recurrent pulmonary emboli with or without concomitant right heart failure, perivalvular abscess, large persistent tricuspid valve vegetation (>20 mm), or right heart failure secondary to severe tricuspid regurgitation.[6] In our case, the patient underwent surgical intervention due to multifactorial. Despite 6 weeks of appropriate antibiotics, he had persistent fever with rising CRP and recurrent pulmonary emboli. His concomitant triple vessels disease with high SYNTAX score and VSD also warrants him for concurrent CABG surgery and open VSD closure surgery.

The preservation of a native pulmonary valve is recommended whenever possible and if valve replacement is unavoidable; either homograft or xenograft is preferred.[2] A pericardial valve was used in our patient, as it reported to lasts longer in pulmonary position than a homograft or porcine valve.[7]

Conclusion

Our case demonstrated the importance of high clinical suspicion and vigilance of diagnosing pulmonary valve endocarditis when dealing with pyrexia of unknown origin in a patient with a congenital VSD. Besides, an active search for clinical and radiological signs of pulmonary embolization is necessary for patients with right-sided endocarditis, especially those with large and mobile vegetation. Last but not least, a conservative approach or valve repair is recommended for most patients with right-sided IE affecting the tricuspid or pulmonary valve. Still, if unavoidable, the use of a homograft or xenograft is preferred.

Lead author biography

Dr Lim Wei Juan graduated from University Malaysia Sarawak (UNIMAS) in 2012 and completed his internship training in 2014. He subsequently joined cardiology department as medical officer. He then joined medical department to complete his Membership of Royal College of Physician (MRCP) in 2016. He undergone his speciality training and joined National Heart Institute in 2021 to continue his dream in cardiology.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Consent: The authors confirm that consent for submission and publication of this case report including the images, laboratory work and associated text has been obtained from the patient in line with COPE guidance.

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