Pulmonary Valve Endocarditis: Always Look on the (B)right Side!

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INTRODUCTION

Right-sided infective endocarditis (IE) constitutes a distinct subset within the spectrum of endocarditis, accounting for a smaller fraction of cases than left-sided IE. Despite its relatively lower incidence, right-sided IE poses unique challenges in both diagnosis and treatment. Unlike left-sided IE, which primarily affects the mitral and aortic valves, right-sided IE predominantly affects the tricuspid valve (TV) and rarely the pulmonary valve (PV) and is often associated with intravenous drug use (IVDU) and intracardiac devices as prevalent predisposing factors. Diagnostic imaging, particularly echocardiography, plays a central role in the timely detection and characterization of right-sided IE. However, anatomical factors may complicate imaging and necessitate the use of adjunctive modalities for accurate diagnosis and also to search for the source of systemic embolism. In this context, we present an interesting clinical case of PV IE, highlighting the diagnostic challenges, therapeutic considerations, and clinical outcomes associated with this condition.

CASE PRESENTATION

We present a 64-year-old man with a history of type 2 diabetes mellitus, coronary artery bypass graft, chronic total occlusion of the right coronary artery, and high-grade stenosis of the superior mesenteric artery. The patient was an active smoker (40 pack-years) with chronic obstructive pulmonary disease GOLD II/B and was addicted to alcohol. They presented to our institution with general weakness, weight loss (20 kg), chills, and fever. Upon arrival, body temperature was 37.7°C, blood pressure 110/62 mm Hg, and heart rate 89 bpm. The patient appeared cachectic with a body weight of 55 kg. Heart auscultation revealed an early diastolic low-pitched murmur on the left upper sternal border. Bilateral breathing sounds were diminished, and there was no peripheral edema. Electrocardiogram showed a sinus rhythm and signs of right ventricular hypertrophy. Blood chemistry revealed an elevated white blood cell count (16,000/μL) and C-reactive protein (303 mg/L), iron-deficiency anemia (Hb 9.8 g/dL), mildly elevated liver tests, and elevated N-terminal pro b-type natriuretic peptide (2,030 pg/mL). Computed tomography (CT) of the chest showed a large consolidation with air bronchogram in the right upper lobe (Figure 1) as well as bilateral patchy subpleural consolidations in both lower lobes and bilateral pleural fluid. Pulmonary emboli were not present.

Empiric antibiotic therapy was started using amoxicillin-clavulanic acid 4 × 1 g for a suspected pneumonia. However, blood cultures were positive for *Enterococcus faecalis* while sputum culture remained negative. Antibiotics were switched to high-dose amoxicillin 6 × 2 g and rocephine 2 × 2 g, and echocardiography was requested.

Transthoracic echocardiography (TTE) revealed large and mobile masses attached to the PV (0.9 × 1.9 cm) prolapsing into the right ventricular outflow tract (RVOT; Figure 2A, Video 1). Color-flow Doppler revealed continuous to and fro laminar flow (Figure 2B, Video 2) and diastolic flow reversal in the branches of pulmonary arteries suggesting massive pulmonary regurgitation (PR).

Continuous-wave Doppler through the PV showed a dense PR signal with a triangular shape, early termination of PR flow, severely reduced pressure half-time (86 msec), and almost equal diastolic to systolic velocity-time integral ratio (consistent with severe PR; Figure 2C). Using Doppler volumetric method, we calculated a regurgitant volume of 114 mL and regurgitant fraction of 71%. The right ventricle (RV) was severely dilated with septal D shaping during diastole (Figure 2D; Video 3) and normal systolic function (tricuspid annular plane systolic excursion, 20 mm). No masses were observed on the TV, and there was mild regurgitation with normal inferior vena cava dimensions and collapsibility.

Transesophageal echocardiography (TEE; both midesophageal and transgastric windows) confirmed these findings (Figures 3 and 4, Videos 4–7). Careful inspection of the TV was normal without signs of endocarditis.

Because there was no hemodynamic instability or uncontrolled infection, the decision of the endocarditis and heart team was to start first with an etiologic workup to exclude malignancy and only if this was excluded to consider an operative repair. The patient denied IVDU, and drug screening was negative. All teeth had previously been lost, and the oral examination did not show an infectious focus. Urine sediment and culture were normal. The patient had a recent enteritis a few months prior to presentation, so bacterial translocation from the gastrointestinal tract was the assumed source of infection. Colonoscopy found 2 subcentrimetric polyps, which were resected. Positron emission tomography with CT (PET-CT) 1 week after the start of antibiotics showed increased fluorodeoxyglucose uptake of the pulmonary consolidation zones and mediastinal lymph nodes, but no hyperenhancement of the valves. Coronary CT angiography showed patency of the bypass grafts.

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VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE on admission, zoomed subcostal aortic valve short-axis and PV long-axis view, demonstrates the mobile masses on the PV.
Video 2: Two-dimensional TTE with color-flow Doppler, zoomed subcostal aortic valve short-axis and PV long-axis view, demonstrates severe PR.
Video 3: Two-dimensional TTE, apical 4-chamber view, demonstrates normal left ventricular size and function with severe right heart dilatation.
Video 4: Two-dimensional TEE, midesophageal RVOT (58°) view, demonstrates a dilated pulmonary artery and the suspected PV vegetative masses.
Video 5: Two-dimensional TEE, transgastric RVOT, biplane orthogonal long-axis (85° and 175°) views, demonstrates the suspected PV vegetative masses.
Video 6: Three-dimensional TEE volume-rendered, en face view from the RVOT perspective, demonstrates the suspected PV vegetative masses.
Video 7: Three-dimensional TEE volume-rendered with transillumination photorealistic display, en face view from the RVOT perspective, demonstrates the suspected PV vegetative masses.

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Heart surgery was performed 4 weeks after the start of intravenous (IV) antibiotics. A redo sternotomy was done. Visual inspection of the PV (Figure 5) confirmed massive vegetations on the cusps with extensive valvular destruction. The annulus was not affected, and there was no abscess. After resection of all the cusps with preservation of the annulus, a PV replacement was performed using a bioprosthesis.

The postoperative course in the intensive care unit was complicated by pneumonia, chronic obstructive pulmonary disease exacerbation, and delirium. Pulmonary bioprosthetic function was normal. On the tenth postoperative day the patient was transferred to the standard ward for continuation of IV antibiotic therapy. Intraoperative cultures remained negative. Further hospital course was uncomplicated, and the patient was discharged home 18 days postsurgery. The total length of hospitalization was 50 days.

DISCUSSION

Right-sided IE accounts for a smaller proportion, approximately 5% to 10%, of all IE cases compared to its left-sided counterpart, resulting in a relative paucity of published information.[1]

The lower incidence of right-sided IE, compared with left-sided IE, may be explained by the relatively low prevalence of pathologic conditions affecting the right valves, including congenital malformations, differences in the properties and vascularization of the right endothelium, and lower pressure gradients and jet velocities across the right heart valves, lower right wall stress, and reduced oxygen content in venous blood.[2] The etiology of right-sided IE primarily involves IVDU, intracardiac devices, and central venous catheters, which have become increasingly prevalent over the past 2 decades.

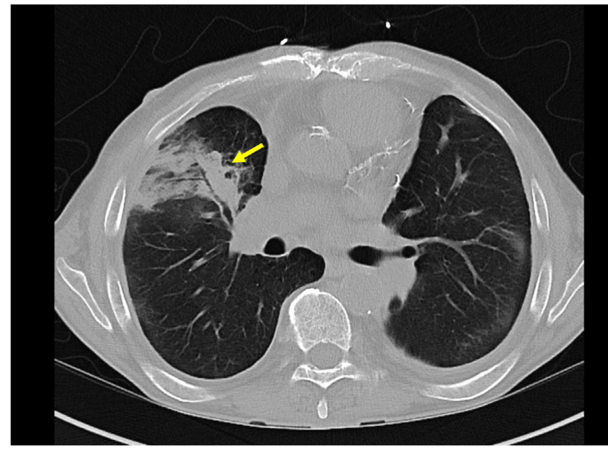

Figure 1 Chest CT scan, axial display, demonstrates pulmonary emphysema with consolidation and air bronchogram in the right upper lobe (*arrow*).

Vegetations can be found anywhere in the endocardium; however, they are most commonly found on the atrial side of the TV, on cardiac device leads, and on the ventricular side of the PV, which is usually involved considerably less than the TV. The TV is responsible for 90% of right-sided IE instances associated with IVDU. While PV IE may coexist with TV IE, isolated PV IE is rare, accounting for less than 2% of all IE cases.[3,4]

Common predisposing factors for PV IE include IVDU, sepsis, immunosuppression, catheter-related infections, congenital heart conditions. and alcoholism. However, in up to 28% of PV IE cases, predisposing factors fail to be identified.[5] Alcohol addiction was the only predisposing factor identified in our patient. Our patient had no recent central catheters or congenital disorders as previous echocardiographic studies showed no structural or functional abnormality of the PV. Preexisting PR in a patient with chronic lung disease and pulmonary hypertension is a possible substrate for IE. However, PR was not seen on the patient's previous echocardiographic studies.

Echocardiography serves as the primary imaging modality for diagnosing right-sided IE, with TTE offering valuable insights due to the anterior localization of right-sided structures. In addition to standard views, subcostal or modified views are often required to obtain a clear overview of the inflow and outflow tracts of the RV, Eustachian valve, and posterior leaflet of the TV.[6] However, in some patients, anatomical factors may lead to suboptimal image quality limiting the sensitivity of TTE, and therefore, interrogation using TEE is necessary.[3] Despite its anterior location in the chest and the bigger distance to the transesophageal probe, the PV can be visualized in the upper esophageal, midesophageal, and transgastric windows to varying degrees of success. Although the diagnosis of PV IE could be made by TTE in this case, TEE ensures with higher confidence that other structures are not involved. This is important as it would influence the surgical approach. Other imaging techniques, such as fluorodeoxyglucose-18 PET-CT, can also be helpful, but in our case the diagnosis was already clear following echocardiography.[7] It is of interest that in our case PET-CT was negative, confirming its poor sensitivity (about 20%) in native valve endocarditis as an important limitation.

The main complication of PV IE in the presented case was severe valvular regurgitation with dilatation of the RV. At admission the patient also had bilateral patchy consolidations in both lower lobes

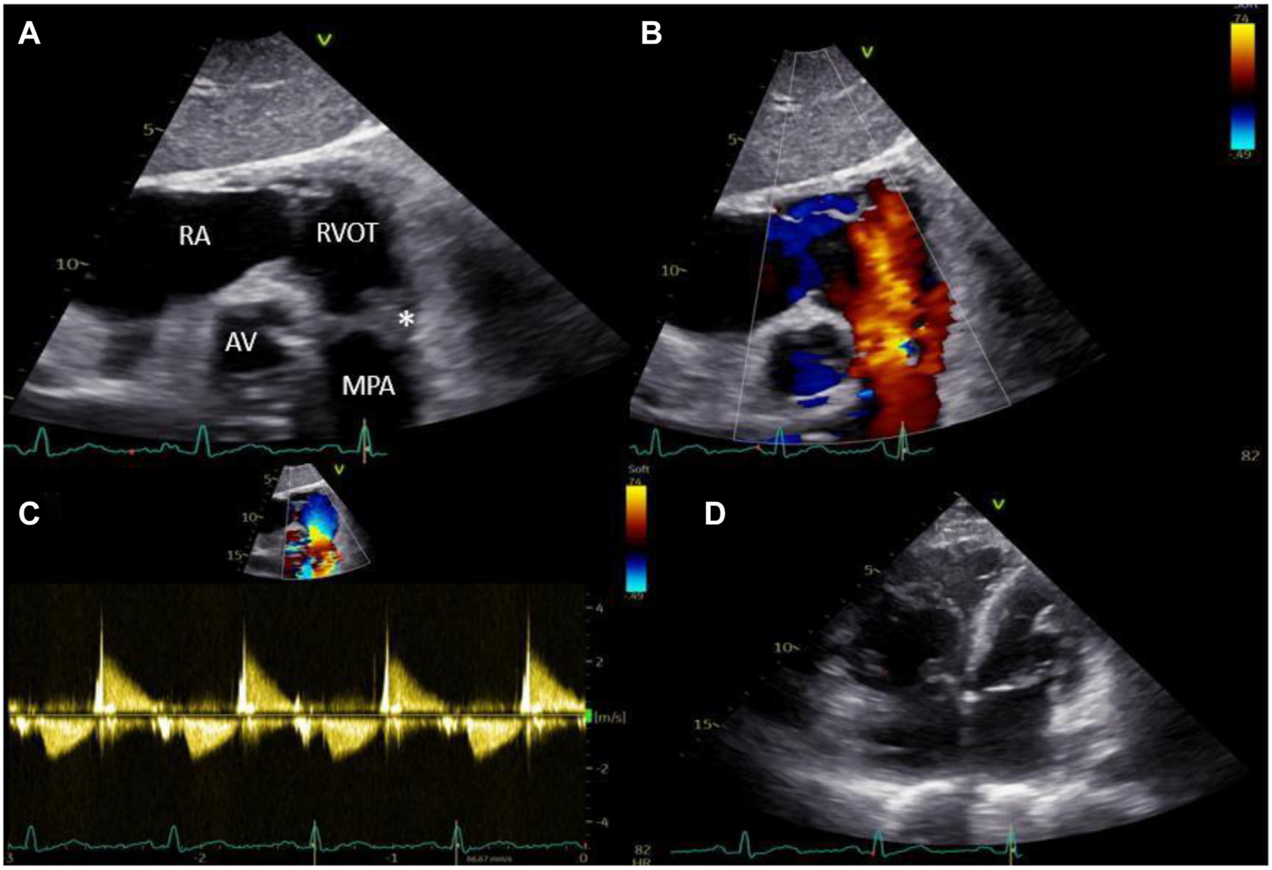

Figure 2 Transthoracic echocardiogram on admission. Two-dimensional zoomed subcostal AV short-axis diastolic view without **(A)** and with **(B)** color-flow Doppler, demonstrates a PV mass (*) and a broad regurgitant jet with laminar flow. **(C)** Continuous-wave Doppler through the PV demonstrates a dense triangular PR signal with early termination of regurgitant flow consistent with severe PR. **(D)** Two-dimensional apical 4-chamber diastolic view demonstrates right ventricular dilatation with a right ventricular cardiac apex. *AV*, Aortic valve; *MPA*, main pulmonary artery; *RA*, right atrium.

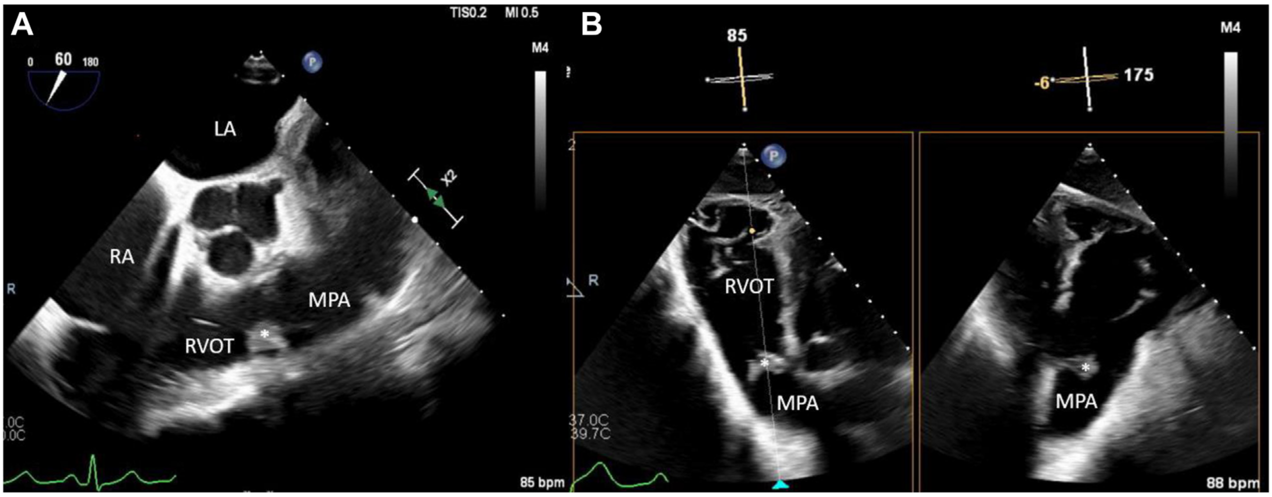

Figure 3 Transesophageal echocardiography before surgery. Midesophageal RVOT short-axis (60°) diastolic view **(A)** and transgastric biplane orthogonal long-axis (85° and 175°) diastolic views, demonstrate a PV mass (*). *LA*, Left atrium; *MPA*, main pulmonary artery; *RA*, right atrium.

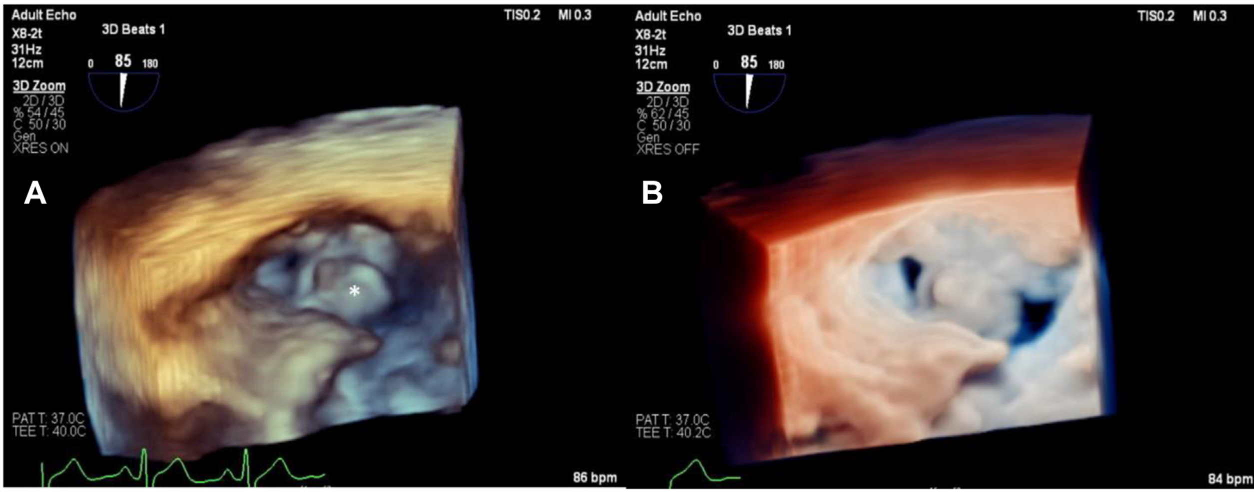

Figure 4 Three-dimensional TEE of the PV in diastole, single-beat zoom acquisition with **(A)** volume rendering (volume rate, 31 Hz) and **(B)** transillumination photorealistic displays, demonstrates the en face structure of the PV from the RVOT perspective to highlight the suspected vegetative masses (*). Images were acquired from the transgastric RV outflow view as seen in Figure 3B.

Figure 5 Surgical specimen of resected PV demonstrates the extensive vegetations (*arrows*).

and bilateral pleural fluid on chest CT. In the literature, lung involvement is due to septic embolization, which may occur in 80% of cases and can range from minor atelectasis to large infiltrates, pleural exudates, and cavitation, generally involving the lower lobes.[8,9] The microorganism causing right-sided IE in our patient was *E. faecalis.* This finding is rather unusual, as the bacteria most frequently associated with right-sided IE are *Staphylococcus aureus* (60%–90% of cases), streptococcus, and coagulase-negative staphylococci. In addition, in the context of chronic alcoholism, *S. pneumoniae* is the most common causative micro-organism of IE.[3,10]

While IV antibiotics remain the cornerstone of treatment for right-sided IE, surgical intervention may be warranted in cases of extensive valvular damage or large valve vegetation.[3] Pulmonary valve replacement for IE is rare and poorly described in the literature.[4,11] Evidence suggests that right-sided IE is associated with better clinical outcomes compared to left-sided IE, with lower rates of systemic embolization, abscess formation, and drug-resistant infections.[10] Currently, mortality rates are generally between 5% and 10%, even without surgery. However, despite low in-hospital mortality, right-sided IE carries a high risk of recurrence, particularly in IVDU populations.[3]

CONCLUSION

This case shows a rare example of native PV IE due to *E. faecalis* in chronic alcoholism. In summary, right-sided IE poses unique challenges in diagnosis and treatment compared to left-sided IE. Despite its lower incidence, it is associated with specific predisposing factors such as IVDU and intracardiac devices. Echocardiography remains crucial for diagnosis, although anatomical complexities may necessitate additional imaging modalities.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

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DISCLOSURE STATEMENT

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SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2024.05.003.

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