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Isolated Pulmonic Valve Endocarditis: Case Report and Review of Existing Literature on Diagnosis and Therapy

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INTRODUCTION

Right-sided endocarditis accounts for 5%-10% of all cases of infective endocarditis (IE),[1] and intravenous (IV) drug use remains its most common predisposing factor.[2] Pulmonic valve involvement in IE is uncommon,[3] and not only is it the least commonly involved valve in IE,[2] with prevalence of around 2%,[4] but "isolated" pulmonic valve endocarditis (PVE) is an even more rare complication of IE.

We report a case of a middle-aged male patient, with recent history of IV drug abuse, who was subsequently found to have a large isolated pulmonic valve vegetation. In line with current medical practice, he was managed with IV antibiotics, but he then went on to develop severe pulmonic insufficiency (PI) during his complicated hospitalization.

While IE is known to cause a previously normal valve to become stenotic or insufficient, there are rarely any data in the preexisting medical literature on severe, hemodynamically significant insufficiency complicating isolated PVE despite adequate medical therapy. Furthermore, PVE usually has a benign course, but it can present with diagnostic difficulty, and the decision to manage complicated PVE conservatively versus surgically, in the absence of robust guidelines, remains challenging.

CASE PRESENTATION

A 46-year-old male patient with a past medical history of type I diabetes mellitus, hepatitis C infection, and IV drug use presented to the emergency department with a 2-week history of generalized malaise, productive cough, abdominal pain, nausea, and vomiting. On admission, he appeared acutely ill and had persistent tachycardia with high-grade fevers. His blood work was significant for a white blood cell count of 38,400/μL with 92% neutrophils, as well as sodium of 125 mmol/L, glucose of 352 mg/dL, albumin of 2.6 g/dL, creatinine of 1.34 mg/dL, lactic acid of 4.6 mmol/L, and an elevated troponin at 0.08 ng/mL. Urine drug screen was positive for cannabinoids and amphetamines. Computed tomographic imaging of the chest, abdomen, and pelvis showed multiple bilateral cavitary lung lesions concerning for septic pulmonary emboli and bilateral pleural effusions.

The patient was found to have methicillin-sensitive *Staphylococcus aureus* bacteremia, and he was started on broad-spectrum antibiotic therapy, which was eventually deescalated to IV vancomycin. Transthoracic echocardiography (TTE) was performed due to suspicion for endocarditis in the setting of positive blood cultures and septic emboli; however, it did not reveal any valvular abnormalities and showed a normal ejection fraction of 60%-65%. To determine the source of infection and due to the worsening clinical status of the patient, a transesophageal echocardiography (TEE) was performed to rule out endocarditis. The TEE revealed a 2.1 $\times$ 2.7 cm pulmonic valve vegetation, partially attached to the wall of the pulmonic outflow tract, but without any notable PI (Figure 1; Videos 1-4).

Aortic, mitral, and tricuspid valves were structurally and functionally normal. The decision was made to wait for resolution of sepsis and for the cultures to become negative before considering surgery. In the interim, serial TTEs showed progression of PI from mild to moderate. Moreover, the patient's respiratory status declined due the development of a loculated left-sided pleural effusion as well as a right-sided hydropneumothorax (Figure 2), and he was emergently intubated.

The pleural effusion was managed with tube thoracotomy and video assisted thoracoscopic surgery. The decline in his respiratory status made him an unsuitable candidate for pulmonic valve replacement during his hospitalization. He was eventually extubated, and the chest tube was removed upon clinical stability and radiological improvement in the size of right hydropneumothorax and loculated pleural fluid at the left base.

He remained hospitalized for completion of IV antibiotic course for 6 weeks after the blood cultures became negative. TTE performed before discharge showed severe PI secondary to the pulmonary valve vegetation (Figure 3, Video 5), which by this time had enlarged to 4 $\times$ 1 cm and was noted to be prolapsing into the (RVOT) and pulmonary artery.

Right atrium and right ventricle appeared mildly enlarged, with slightly depressed right ventricular systolic function. The inferior vena cava demonstrated <50% collapse consistent with elevated right atrial. In addition, rapid flow deceleration during the diastole, which is suggestive of severe PI, was noted (Figure 4).

Surgical intervention was deferred due to the risk of prosthetic valve infection secondary to patient's lack of will to abstain from IV

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VIDEO HIGHLIGHTS

Video 1: TEE midesophageal short-axis view of the heart showing the pulmonic valve vegetation.
Video 2: TEE midesophageal long-axis view showing the pulmonic valve vegetation.
Video 3: TEE sweep from the short-axis view to the long-axis view showing the pulmonic valve vegetation.
Video 4: TEE midesophageal long-axis view of the right ventricular outflow tract showing pulmonic vegetation involving the pulmonic cusp and the pulmonary annulus.
Video 5: TTE short-axis view at the basal level showing the pulmonic valve vegetation and pulmonic regurgitation.

View the video content online at www.cvcasejournal.com.

drugs after discharge. Subsequently, he was discharged and close follow-up with a multidisciplinary team was set up for drug rehab and surveillance of pulmonic valve vegetation and severe insufficiency.

DISCUSSION

PVE accounts for only 1.5%-2.0% of all cases of IE.[3] PVE has rarely been reported in the literature in the absence of concurrent involvement of other cardiac valves,[5] and its low incidence is thought to be secondary to multiple physiologic mechanisms.[6] It is known to occur most frequently in association with IV drug abuse,[4,7-9] and the most commonly isolated causative agents include *Staphylococcus aureus*, coagulase-negative Staphylococci, and group B Streptococci.[4,10]

PVE commonly manifests clinically as fever in the setting of predominantly pulmonary symptoms such as dyspnea, pleuritic chest pain, cough, and hemoptysis.[8] PI is a late development of PVE and is detected on physical examination in about 50% of patients with PVE as a low-pitched, diastolic murmur that is easily missed.[7,10,11] The absence of signs and symptoms of cardiac disease often leads to delayed diagnosis, especially in patients lacking traditional risk factors or concurrent involvement of other valves.[7]

Given the low incidence of PVE, there are scant data on the diagnostic test of choice. One study suggests that TTE can be used to diagnose isolated PVE in as many as 91% of cases.[4] Miranda *et al.*,[9] in their review of nine patients who were diagnosed with PVE between 2000 and 2014, reported that TTE was diagnostic in all six patients, whereas one out of seven TEEs mistook a pulmonic valve vegetation for an RVOT thrombus.[9] In a subsequent study involving 17 patients, Miranda *et al.*[12] noted that TTE and TEE were diagnostic in 62% and 57% of patients, respectively; however, the diagnostic accuracy increased to 88% when both the modalities were combined. Conversely, in our case TTE failed to detect any pulmonary valve abnormalities, whereas TEE detected the 2.7 × 2.1 cm vegetation only a few days later.

Doppler and echocardiographic parameters are used to grade severity of PI, but no single Doppler and echocardiography parameter is specific enough to quantify PI severity, so integration of multiple parameters is required to grade PI. It is acknowledged that severe PI

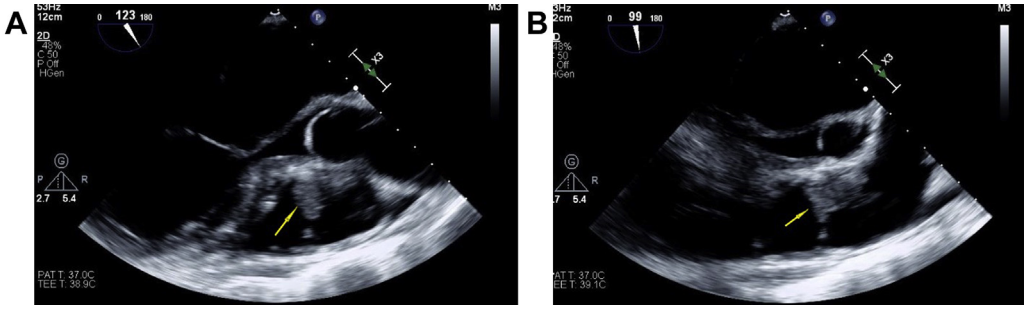

Figure 1 **(A, B)** TEE showing a large vegetation on the pulmonic valve (*yellow arrows*).

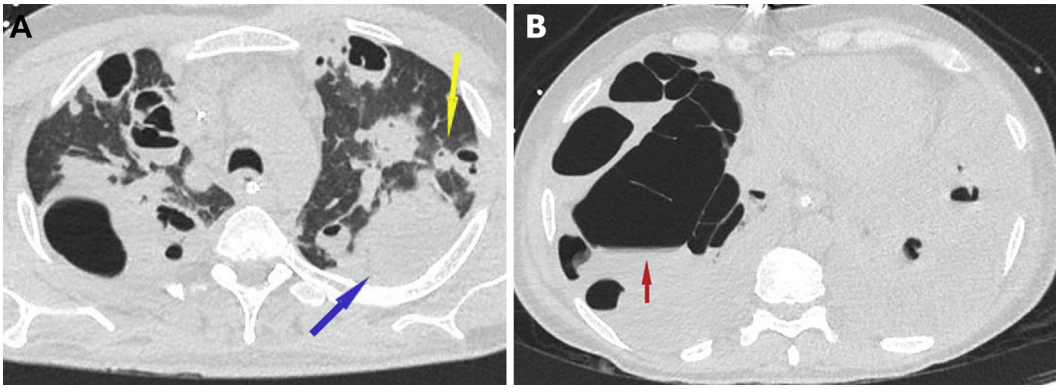

Figure 2 **(A, B)** Computed tomographic chest imaging without contrast showing extensive bilateral cavitary nodules and consolidations (*yellow arrow*), with a large right loculated hydropneumothorax (*red arrow*) and large left loculated pleural effusion (*blue arrow*).

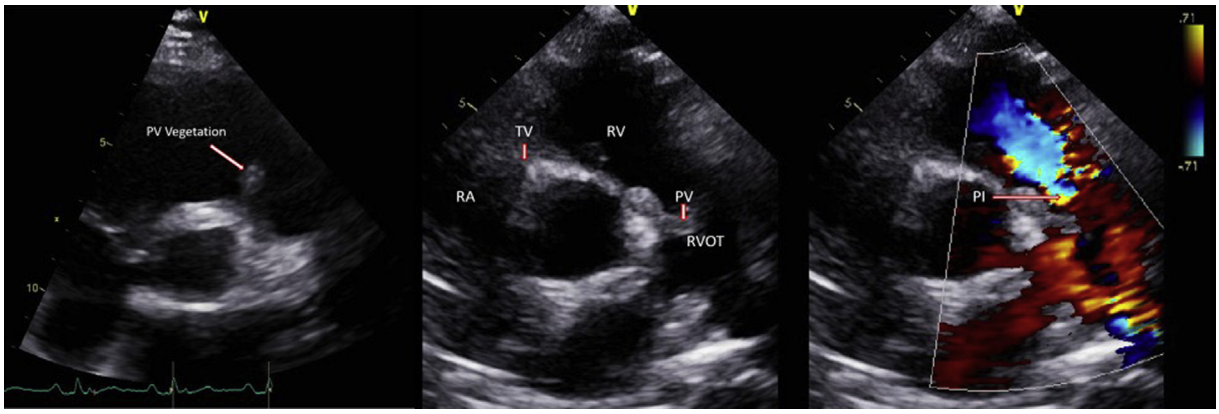

Figure 3 TTE showing pulmonic valve (PV) vegetation causing severe PI. *RA*, Right atrium; *RV*, right ventricular; *TV*, tricuspid valve.

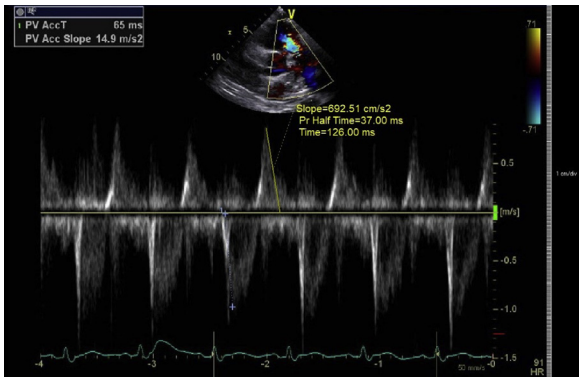

Figure 4 Doppler velocity trace depicting rapid deceleration rate, as shown by the steep PR slope, with extension into late diastole, which is indicative of severe PR. *PR*, Pulmonic regurgitation.

is challenging on spectral color Doppler because of lack of turbulence, but, despite lacking specificity, the rapid deceleration rate of the continuous-wave Doppler signal with termination in mid to late diastole is often compatible with severe PI.[13]

Cardiac magnetic resonance imaging is an excellent modality for evaluation of the pulmonic valve and pulmonary artery and for quantification of PI severity with direct phase-contrast method. It is also the preferred method for quantitation of right ventricular volume and function, as the right ventricle is frequently dilated in chronic severe PI.

Endocarditis involving the pulmonary valve usually has a benign, reversible course and typically responds to parenteral antibiotic therapy administered for a period of 4-6 weeks.[14] Surgery is mostly reserved for cases where there is persistent bacteremia, complications such as abscess formation and recurrent septic pulmonary embolism, and relapse. It is recommended that early surgical intervention should be considered in hemodynamically unstable patients, as well as in those with vegetations larger than 2 cm and in whom the causative agent is Staphylococcus.[15] As was clearly illustrated by our patient's clinical course, wherein surgical intervention was not an option due to his multiple comorbidities and complicated clinical course, PVE can be complicated by the development of stenotic or insufficient valvular lesions and consequent cardiovascular compromise despite adequate antibiotic therapy.

CONCLUSION

Isolated PVE has a low incidence and mandates a multidisciplinary approach to its management because of the complexity of its presentation, lack of understanding of the natural history of the disease, and lack of clinical guidelines for its diagnosis and management. Our case serves to reiterate that PVE can be missed on TTE, which is traditionally a more reliable diagnostic technique than TEE, and therefore poses an impressive diagnostic challenge. Furthermore, even though PVE has historically been known to respond well to antibiotic therapy, it is important to recognize that larger vegetations are associated with a poor outcome,[15] but the decision to intervene surgically should not be just based on the size of the vegetation.[2] Although not possible with our patient due to his clinical condition and lack of willingness to give up substance abuse, our case nevertheless highlights the need for close monitoring of the lesion, should any evidence for lack of response to culture and sensitivity guided antibiotic therapy emerge.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2019.05.003.

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