

EP CASE REPORT

Premature ventricular complexes from a left ventricular diverticulum: a case report

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Introduction

Safety reasons may prohibit ablation of premature ventricular complexes (PVCs) like in case of a left ventricular diverticulum (LVD). Left ventricular diverticulum complications include thrombosis, embolism, rupture, heart failure, ventricular arrhythmias, and valvular abnormalities. This report presents a case of high PVC burden originating from the LVD.

Case presentation

A 54-year-old Asian male presented with palpitations, weakness, chest discomfort, and dyspnoea. He was previously asymptomatic and symptoms progressed over the last 4 years, despite treatment with antiarrhythmic drugs (class Ic/III). The electrocardiogram revealed frequent PVCs with inferior axis and right bundle branch block morphology. The PVC burden was 15% (Holter monitoring).

We performed an electrophysiology study aiming for PVC ablation. We used retrograde arterial access to map the left ventricle and found the earliest myocardial activation (13 ms prior to QRS begin) at the basal part of the lateral wall. We then noticed a local protrusion of the left ventricular (LV) shadow in close proximity and excluded an ischaemic LV aneurysm/coronary stenosis with angiography. Considering the risks of perforation/embolism we withheld ablation and stopped the procedure.

Echocardiography revealed no abnormalities other than some antero-lateral wall thickness. We performed a contrast-enhanced computed tomography that revealed a large trabeculated LVD (33 × 21 × 29 mm) with a small endocardial ostium.

The patient was referred for surgical LVD resection. During the operation the LVD appeared to have muscle and fibrous sleeves (Figure 1A). An *en bloc* resection was performed under extracorporeal circulation and the margins were coagulated prior to a linear plastic repair (Figure 1, 6B). The clinical PVCs diminished immediately and the patient was free of symptoms.

Discussion

Left ventricular diverticula (LVDs) are usually congenital due to a partial cessation in the development of the embryonic tissue.¹ Although most LVDs are diagnosed in early childhood, non-apical LVDs are often isolated, with a good prognosis that may result in later diagnosis.²

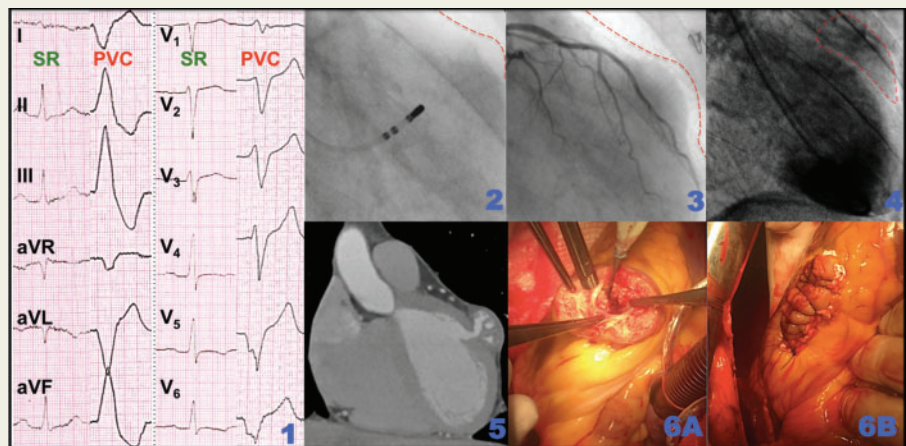


Figure 1 Sinus rhythm (SR) and a premature ventricular contraction (PVC) (1) from a bulging left ventricular wall protrusion (2–4), found to be a diverticulum (5) that was resected (6).

Preoperative investigations did not reveal its presence and suspicion only aroused during the electrophysiological study due to an out-pouching of the LV shadow at the earliest activation site. We came to a conclusion that this might be a diverticulum. Not knowing the thickness of the wall in this area and given the associated risk, the procedure was stopped. Contrast computed tomography revealed a large LVD with a small ostium that could not allow insertion of the catheter. Moreover, the internal structure was characterized by deep trabeculations that could harbour the PVC origin. The precordial PVC morphology and the not so early endocardial activation (only –13 ms) strengthened the suspicion of a distal PVC origin within the LVD.

We decided to refer the patient for LVD resection in order to treat the PVC and prevent possible complications like embolization, heart failure, rupture, or even sudden death.^{2,3} This eliminated the clinical PVCs and the patient remained asymptomatic during follow-up.

Conclusion

Careful imaging is mandatory in patients with PVCs and a suspected LV diverticulum. Symptomatic therapy-refractory PVCs that are related with an LVD can be successfully treated with surgical resection.

Conflict of interest: none declared.

References

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