Left ventricular pseudoaneurysm as a complication of left ventricular summit premature ventricular contraction ablation

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**Introduction**

The development of a left ventricular (LV) pseudoaneurysm as a complication of focal premature ventricular contraction (PVC) ablation has not been previously reported. We describe the case of an LV pseudoaneurysm manifesting as recurrent cardiac tamponade following PVC ablation of the endocardial portion of the LV summit.

**Case report**

A 52-year-old woman with a history of recurrent, symptomatic paroxysmal atrial fibrillation and symptomatic PVCs underwent a combined radiofrequency (RF) pulmonary vein isolation, superior vena cava isolation, and PVC ablation. The patient had a resting heart rate of 35–50 beats per minute with post-PVC accentuated pauses and became fatigued with beta-blocker therapy. The earliest site of PVC activation was mapped to the endocardial portion of the LV summit. The local timing was equally early in the left coronary cusp and immediately below the cusp in the LV endocardium (<20 ms) (Figure 1). Ablation was performed using RF current and a 3.5-mm open-irrigated ablation catheter (Thermocool SmartTouch D/F curve; Biosense Webster, Diamond Bar, CA). Ablation was first performed below the left coronary cusp with titration from 30 W to 50 W owing to late transient suppression (120 seconds, 22–26 g). Given the suspicion of an intramural site of origin with the appearance of far-field local electrograms no earlier than 20 ms, 2 additional 1-minute RF applications were delivered with incomplete suppression at 50 W (temperature limit 42°C). Ablation at 40 W was then performed in the left coronary cusp (8–18 g) under intracardiac echocardiography visualization to ensure adequate distance from the left main coronary artery. Immediate suppression was observed within 1 second of the second RF application. Two consolidation lesions (1 minute each, 4 minutes of total RF) were delivered after PVC suppression. There were no sudden impedance rises during RF ablation and the maximum temperature observed was 35°C for all the applications. The largest impedance drop observed was 29 Ω over 2 minutes at the first endocardial ablation site. Intraprocedural intracardiac echocardiography imaging was notable for a small pericardial effusion that remained stable through the duration of the procedure. The patient was discharged the following day on apixaban 5 mg twice daily, which had been held 1 day prior to the procedure. The patient developed chest pain 3 days post procedure. A transthoracic echocardiogram was normal and the pain was felt to be consistent with pericarditis.

Three weeks post procedure, the patient presented with sudden-onset cardiac tamponade requiring emergent pericardiocentesis. The aspirate was consistent with a hemorrhagic pericardial effusion. A chest CT did not show evidence of cardiac perforation or esophageal fistula. Recurrent pericardial effusion was not seen on serial echocardiograms. The pericardial drain was removed after three days. Anticoagulation was discontinued and the patient was initiated on steroid therapy for presumed hemorrhagic pericarditis.

Five weeks later, the patient presented with recurrent cardiac tamponade requiring repeat pericardiocentesis. A chest computed tomography (CT) was performed and did not show extravasation of contrast, but showed a double layer of pericardial enhancement suggestive of pericardial inflammation. The pericardial aspirate was again consistent with a hemorrhagic effusion. The presentation was thought to be an aggressive case of Dressler syndrome with an elevated C-reactive protein of 75 mg/L (normal <5 mg/L). Pulse steroid therapy paralleled with self-limited drainage

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Cardiac magnetic resonance imaging was subsequently obtained at another institution and revealed a 2.4 × 2.4-cm pseudoaneurysm arising from the basal anterior LV wall immediately below the aortic valve (Figure 2). In retrospect, the initial CT scan also demonstrated this finding; however, the large basal anterior ventricular pseudoaneurysm was misinterpreted as the left atrial appendage (Figure 3). The patient underwent cardiac surgery, during which a 1-cm defect of the basal anterior LV wall was identified by direct visualization and intraoperative transesophageal echocardiography. The LV outflow tract was approached through an aortotomy carefully protecting the native aortic valve. The pseudoaneurysm was readily visible below the left coronary cusp. A bovine pericardial patch was sewn to the neck of the pseudoaneurysm using interrupted, horizontal mattress 4-0 polypropylene sutures. Additionally, the left atrial appendage was excised. Intraoperative transesophageal echocardiography showed complete exclusion of the pseudoaneurysm. The patient did well postoperatively, with no evidence of recurrent pericardial effusion. The patient continued to have intermittent PVCs in follow-up, but she has remained free from AF recurrence after 5 months.
LV pseudoaneurysm, or contained LV rupture, is most commonly seen post myocardial infarction or in association with cardiac surgery or trauma. LV pseudoaneurysm is a rarely reported complication of catheter ablation. Mansour and colleagues describe a case of LV pseudoaneurysm formation near the mitral annulus presenting 15 years after low-energy direct current accessory pathway ablation. Koch and colleagues report a case of a 6.1-cm LV pseudoaneurysm identified in a patient with cardiac sarcoidosis following multiple endocardial and epicardial RF ablations for ventricular tachycardia. This is the first described report of an LV pseudoaneurysm as a complication of PVC ablation manifesting as recurrent cardiac tamponade. The diagnosis of Dressler syndrome is one of exclusion and the rare finding of hemorrhagic effusion post ablation should prompt consideration for this uncommon diagnosis, particularly when recurrent.

This case highlights the importance of considering the potential for life-threatening complications related to focal PVC ablation. Although the thick LV myocardium is generally regarded as resistant to perforation, caution is warranted while ablating in the region of the LV summit with high power and irrigated technology. Whereas the LV summit is associated with higher rates of recurrence, lower power with fewer consolidation lesions may have prevented this complication. It is not clear whether the risk of this complication was increased because of the concomitant pulmonary vein isolation with therapeutic anticoagulation, although the activated clotting time range was 250–300 and the novel oral anticoagulant was held 1 day prior to the procedure. This case provides a reminder of the potential harm that can be caused to patients in the process of an elective treatment of a non-life-threatening condition from this vulnerable region within the heart. Complex ablation procedures performed in tandem in a single procedure may compound the risk for complications, injury, and harm.

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