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A case of premature ventricular contractions-related cardiomyopathy

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Abstract

Premature ventricular contractions (PVCs) are heart beats initiated in the ventricles instead of in the sinoatrial node. A high burden of PVCs can lead to a cardiomyopathy, characterised by reduced left ventricular (LV) systolic dysfunction. We present a case of PVC-related cardiomyopathy where the 65-year-old male was initially seen by his primary care provider for recent onset chest pain and dizziness. His transthoracic echocardiogram showed mild concentric LV hypertrophy and mildly reduced systolic function (LV ejection fraction 43%). There was also mild right ventricular (RV) systolic dysfunction. He was started on a beta-blocker and an angiotensin-converting enzyme inhibitor. A 24-hour Holter monitor showed a very high burden of PVCs (32% of all beats). He continued to have frequent PVCs and his echocardiogram did not improve. He was eventually referred for a PVC ablation. Following the ablation, a repeat Holter monitor showed a marked reduction in PVC burden (<1% of beats) and his echocardiogram had normalised.

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Introduction

Premature ventricular contractions (or complexes) (PVCs) originate from ectopic ventricular sources and these premature beats can cause the sensation of 'fluttering' or 'skipped beats'. PVCs originate in the Purkinje fibres of the ventricles and disturb the regular pattern of conduction of normal sinus rhythm. They may be followed by a 'compensatory pause' resulting in the sensation of a 'skipped beat'. Three or more consecutive PVCs is defined as ventricular tachycardia which, if prolonged, can be fatal.

The causes of PVCs are variable and the exact cause is often unknown. The prevalence of PVCs in the general population is estimated to range from 1–4% based on electrocardiograms (ECG) and 40–75% on Holter monitoring.[1] Patients with PVCs may or may not be symptomatic. The initial diagnosis is usually obtained on a 12-lead ECG which can reveal ectopic beats, in addition to other abnormalities which may be related, e.g. electrolyte derangements, prior myocardial infarction, left ventricular (LV) or right ventricular (RV) hypertrophy. Ambulatory rhythm monitors, such as Holter monitors, can help to correlate arrhythmias with symptoms and also quantify their burden, which can help estimate the chances of developing a cardiomyopathy or other complications. Laboratory and other testing can evaluate for potential aetiologies of the PVCs, e.g. structural heart disease, myocardial ischaemia, electrolyte disturbances, thyroid dysfunction, etc. A high burden of PVCs can lead to a cardiomyopathy, characterised by reduced LV systolic dysfunction.[2] Medical and interventional treatment of the PVCs may therefore need to be considered.

Here, we present a case of PVC-related cardiomyopathy. In this case, the patient had a low LV ejection fraction (EF) and high burden of PVC which, after thorough evaluation, was presumed to be the cause. His symptoms and EF improved after ablation of the PVCs.

Case presentation

A 65-year-old male with no PMH presented to clinic with chest pain that occurred three weeks ago along with a generalised sense of 'feeling unwell' since then. He had also felt intermittently lightheaded and dizzy, in addition to confirming occasional palpitations. His chest pain (described as a 'pressure') had not recurred. He denied dyspnoea, syncope, leg swelling, nausea, vomiting, dysuria, haematuria, abdominal pain and headache.

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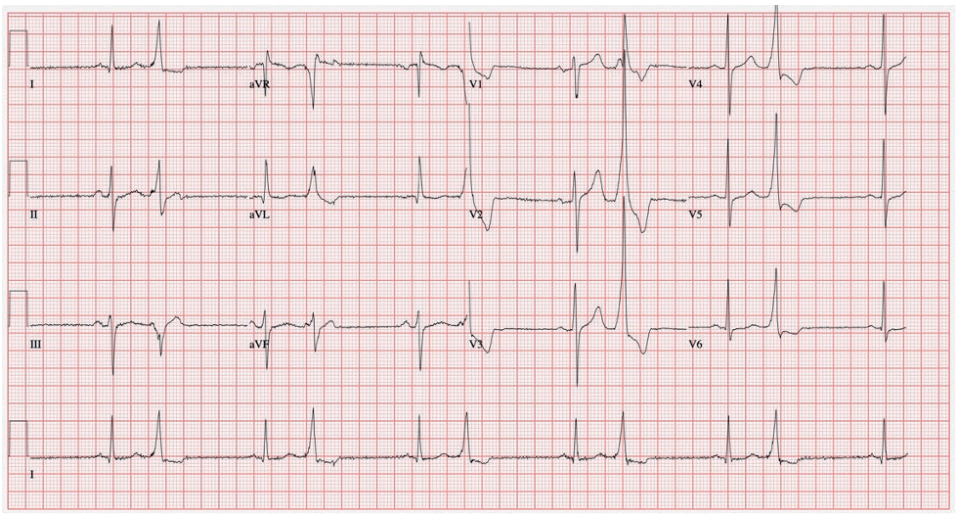

Figure 1 Patient pre-ablation 12-lead ECG

Although he was without recurrent chest pain, his primary provider in the clinic was concerned and he was sent to the Emergency Department. A 12-lead ECG showed sinus rhythm with PVCs present in a pattern of bigeminy (Figure 1). Notable laboratory tests in the Emergency Department showed a normal (undetectable) troponin I × 2, normal comprehensive metabolic panel and a normal thyroid stimulating hormone. Substance use was an unlikely aetiology given that he had no prior history of recreational drug or significant alcohol use, and a urine toxicology screen in the Emergency Department was negative. He had a transthoracic echocardiogram (TTE), which showed mild concentric LV hypertrophy and mildly reduced systolic function (LVEF of 43%) and global hypokinesis. There was also mild RV systolic dysfunction and mild aortic valve sclerosis. Differential diagnoses included an acute process causing LV systolic dysfunction and a high burden of PVCs, such as myocarditis. However, a magnetic resonance imaging scan was not performed because of the normal troponin I and ESR. He was thought to be stable for discharge from the Emergency Department with cardiology follow-up. He was started on metoprolol succinate 25 mg per day and lisinopril 5 mg per day.

Three weeks after presentation at the Emergency Department, he underwent myocardial perfusion scanning and Holter monitoring. The nuclear vasodilator stress test showed no evidence of ischaemia or infarction, but gated images suggested severely reduced LV systolic function at rest and moderate reduction at maximal stress. A 24-hour Holter monitor revealed a heart rate range of 45–87 (mean 60 bpm) without significant pauses, but a very high burden of PVCs (32% of all beats), as well as rare PACs. The patient's diary noted headache, nausea, chest discomfort and back pain, possibly associated with PVCs, but it was difficult to correlate the PVCs to the symptoms given the high burden of PVCs. Metoprolol was continued and lisinopril was increased to 5 mg twice daily, but further medication changes were not tolerated due to borderline low blood pressure and light-headedness.

Three months after his initial presentation, he continued to have palpitations and his echocardiogram was unchanged. Clinic ECGs continued to show frequent PVCs and the patient was referred to the electrophysiology clinic. The electrophysiology consultant felt that given the patient's

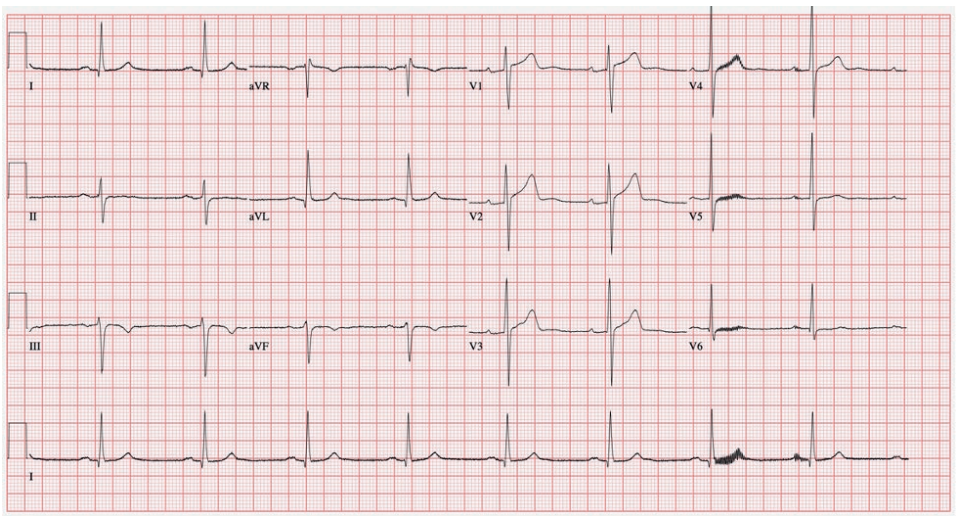

Figure 2 Patient post-ablation 12-lead ECG

prior workup and Holter findings, it was likely that the patient had a PVC-related cardiomyopathy, and that even if it was not the primary aetiology, a high burden of PVCs could still be detrimental to the patient's prognosis. The patient was offered ablation therapy vs a trial of antiarrhythmic medications, but the patient was fearful of possible side effects of antiarrhythmic medications and chose ablation. At the time of the ablation procedure, three-dimensional anatomic-electrophysiologic mapping was performed and the PVCs were localised to the postero-septal mitral annulus. Notable ECG features that suggested this location were the width of the QRS complex, slurred upstroke of the QRS complex (resembling that of Wolff–Parkinson–White's delta wave), and early r-wave precordial transition.

Radiofrequency energy was used to create precise 'burns' to the area with catheters. This resulted in immediate suppression of the PVCs, and recurrences were not able to be induced even with high dose isoproterenol infusion. There were some occasional PVCs noted, but these were from different origins. A repeat Holter monitor performed one month later showed normal sinus rhythm ranging from 39–96 bpm (mean 53 bpm). PVCs represented <1% of beats with an isolated VE couplet, and PACs represented <1% of beats with few SVE couplets. Repeat 12-lead ECG also confirmed the absence of PVCs (Figure 2). At a follow-up appointment, the patient reported he had been asymptomatic since the ablation. A repeat echocardiogram obtained thereafter showed that both LV and RV function had normalised (LVEF was 58%).

Discussion

PVCs are common and usually asymptomatic or minimally so. Until recently, PVCs were thought to be benign if no structural heart disease was present. However, this belief is changing as we discover more associations with cardiovascular pathology. There is a growing body of evidence that reveals a high PVC burden may result in a cardiomyopathy. Moreover, the patterns seen in the mean heart rate of Holter monitors imply that the cardiomyopathy induced by PVCs is different to tachycardia-induced cardiomyopathy.[3] As such, we report a case of a patient with PVCs who developed a PVC-mediated cardiomyopathy which resolved with ablation.

PVC-induced cardiomyopathy was brought into focus by Duffee et al. when LV systolic dysfunction improved after pharmacological suppression of PVCs in patients thought to have idiopathic dilated cardiomyopathy.[4] Pharmacological therapies, such as beta-blockers or non-dihydropyridine calcium channel blockers, are traditionally initiated when the patient is symptomatic.[5] PVC-induced cardiomyopathy is important to consider when LV dysfunction is diagnosed, especially in older adults where PVC prevalence dramatically increases.[5] If suspected, rhythm monitoring (e.g. 24–72 hour Holter monitoring) should be performed to quantify the PVC burden. Although there is no uniformly accepted definition, PVCs in excess of 10–15% or more than 10,000/day on monitoring has been quoted as thresholds for a 'high burden'. Stress testing/angiography (for coronary artery disease), a laboratory evaluation (e.g. serum protein electrophoresis/urine protein electrophoresis/free light chains (amyloid), thyroid stimulating hormone (hypo/hyper-thyroidism), human immunodeficiency virus, iron studies (anaemia)), and other imaging (e.g. cardiac magnetic resonance imaging) can be used to exclude other causes of LV dysfunction, and a careful family history can help to rule out familial dilated cardiomyopathy.

Evidence suggests that PVC suppression can improve heart function in patients with symptomatic PVC.[5] After lifestyle modification, such as reducing or eliminating stimulant use (e.g. caffeine and other medications), therapies are split into medications and catheter ablation. Catheter ablation is quickly becoming a first-line therapy for PVC-mediated cardiomyopathy, with beta-blockers and non-dihydropyridine calcium channel blocker medications also being options.[5,6] However, most cardiologists would not use calcium channel blockers in patients with LV systolic dysfunction. In a study of 30 patients with remote myocardial infarction referred for cardioverter-defibrillator implantation, 15 of the patients had a PVC burden ≥5% of all QRS on 24-hour Holter monitoring. In that setting, catheter ablation improved LVEF from 0.38 ± 0.11 to 0.51 ± 0.09 compared to the control group of 15 patients.[7] Patients should be monitored following treatment (either medically or ablatively) to assess both LV function and PVC frequency.

Hyman et al. recently performed a cohort study of 20 patients with suspected PVC-related cardiomyopathy who had previously undergone ablation, but still had a high PVC burden by treating them with Class IC agents (flecainide or propafenone).[8] In follow-up, they showed significant reductions in PVC burden and an increase in LVEF, without complications (sustained ventricular arrhythmias or sudden death).[8]

Conclusion

In patients with a high burden of PVCs who are diagnosed with LV systolic dysfunction, and after other causes of it have been excluded, PVC-induced cardiomyopathy should be strongly considered as the cause. As such, treatment should be initiated that may include catheter ablation, pharmacotherapy or both. Here, we presented a case of premature ventricular-related cardiomyopathy with decreased systolic function and a high burden of PVCs, both of which resolved following PVC ablation.

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