Premature Ventricular Complexes in Apparently Normal Hearts

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KEYWORDS

• PVC-induced cardiomyopathy • Catheter ablation • Idiopathic ventricular fibrillation

KEY POINTS

- Although generally considered benign, epidemiologic studies have consistently shown PVCs to be associated with a worse prognosis and higher morbidity and mortality than those patients without PVCs.
- Diagnostic evaluation in the patient with PVCs and no known structural heart disease should consist of complete history; physical; 12-lead ECG; 24-hour Holter monitor; echocardiogram; and where appropriate advanced imaging with exercise stress test, cardiac MRI, cardiac PET scan, and endocardial voltage mapping to exclude structural heart disease, define the morphology and frequency of the PVCs, and if appropriate plan for ablation.
- Options for management for patients with PVCs include watchful waiting if asymptomatic and normal ejection fraction with at least yearly evaluation and echocardiography, β-blockers, calcium channel blockers, antiarrhythmic medications, or catheter ablation.
- Catheter ablation is potentially curative in patients with high-risk or malignant PVCs including PVC-induced ventricular fibrillation.
- Catheter ablation is superior to medical therapy for PVC-induced cardiomyopathy and after successful ablation most show improvement in EF after 3 to 12 months.

PREMATURE VENTRICULAR COMPLEXES IN STRUCTURALLY NORMAL HEARTS: PRESENTATION AND DETECTION

Premature ventricular complexes (PVCs), also referred to as ventricular premature beats or ventricular extrasystoles, are common and prevalent in individuals with and without structural heart disease. The prevalence of PVCs depends on the characteristics and comorbidities of the population, the method by which the population is studied, and the duration of observation. In a review of healthy Air Force recruits with no known structural heart disease the prevalence of PVCs on surface electrocardiogram (ECG) was 2.4%.[1] When monitored for 24 hours with ambulatory Holter, the prevalence of PVCs reported in individuals without structural heart disease ranges from 40% to 100%.[2–6] Population studies, such as the Atherosclerosis Risk in Communities (ARIC) and a subset of the Multiple Risk Factor Intervention Trial (MRFIT), demonstrated a higher prevalence of PVCs in men than women, African Americans compared with white persons, and an increased

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prevalence in individuals with hypertension. Increasing age was noted to be associated with higher PVC burden with a 34% increase for each 5-year increment increase in age.[5,7] The mechanism of PVCs in patients with a structurally normal heart seems to be cyclic adenosine monophosphate–mediated triggered activity.[8–10]

Symptoms

Symptoms are mild or absent in many patients; however, in some individuals symptoms are disabling. Common symptoms include palpitations and dizziness; frequently these symptoms may be caused by the PVC or the subsequent compensatory pause. The pause allows time for greater calcium uptake by the myocardium and a sinus beat following PVC is hypercontractile. Symptoms may be attributed to the PVC, the compensatory pause, hypercontractile sinus beat, or a combination of all. Other symptoms include lightheadedness and near syncope with the onset of ventricular bigeminy and a sudden decrement to one half of the hemodynamically effective heart rate (Fig. 1). A sensation that the heart has "stopped" or pulsation in the head or neck can be noted. Pulsation of the head or neck may correlate with the physical examination finding of "cannon" A waves because a PVC may trigger simultaneous contraction of the atria and ventricles and contraction of the atria against closed mitral and tricuspid valves. Symptoms of heart failure or abrupt syncope are concerning for high-risk forms of PVCs, such as those producing cardiomyopathy or PVC-induced ventricular fibrillation (VF) and should prompt further diagnostic evaluation.

Diagnostic Evaluation

Personal and family history should focus on excluding known structural heart disease including early coronary artery disease; previous myocardial infarction; valvular heart disease; and excluding any family history of inheritable conditions including channelopathies, hypertrophic cardiomyopathy, or arrhythmogenic right ventricular cardiomyopathy. Endocrine disorders, such as thyroid, adrenal, or pituitary abnormalities, may uncommonly precipitate or affect the burden of PVCs. A history of sarcoidosis in any organ system or obstructive sleep apnea may require further evaluation for structural heart disease. Social history should screen for illicit drug use and over-the-counter medications that may contain stimulant or sympathomimetic compounds. A full history of herbal medications with package inserts should be reviewed. Some patients may identify triggers in food or drink that correlate with increased burden of PVCs.

Diagnostic evaluation of patients with symptoms of PVCs should be directed at confirmation of PVC as a cause of symptoms, quantifying the daily burden as number or percent of all beats and determining the different morphologies of the PVCs. Initial evaluation should consist of a 12-lead ECG and ambulatory monitor of at least 24 hours, with longer duration if absence of symptoms during monitoring period. Patients with intermittent symptoms may need 30-day monitoring or rarely implantable loop recorder to document the presence of PVCs and correlation with symptoms. Patients should have transthoracic echocardiogram to identify significant structural heart disease. Additional imaging with cardiac MRI or cardiac PET scan may be considered in patients with multiple morphologies of PVCs or abnormal surface ECG to help identify such conditions as arrhythmogenic right ventricular dysplasia, amyloidosis, or cardiac sarcoidosis.[11,12] Treadmill stress testing allows characterization of morphology of PVCs, response

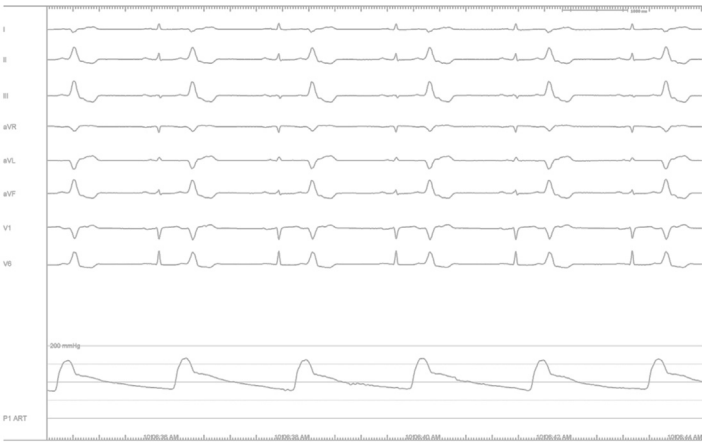

Fig. 1. Nonperfused PVC producing a sudden decrease in heart rate and presyncope. Surface ECG leads and arterial pressure tracing are shown.