Mini Review
Volume 4 Issue 4 - October 2017
DOI: 10.19080/JOJCS.2017.04.555643

JOJ Case Stud
Copyright © All rights are reserved by Saurabh Mehrotra

Radiofrequency Ablation in Frequent Ventricular Ectopy

Saurabh Mehrotra1*, Rakesh Sharma2 and Sundeep Mishra3

1Department of Cardiology, Postgraduate Institute of Medical Education and Research, India
2Senior Resident, Postgraduate Institute of Medical Education and Research, India
3Department of Cardiology, All India Institutes of Medical Sciences, India

Submission: August 16, 2017; Published: October 23, 2017
*Corresponding author: Saurabh Mehrotra, Associate Professor, Department of Cardiology, Postgraduate Institute of Medical Education and Research, Chandigarh, India, Email: rhythm_divine46@yahoo.com

Abstract
Ventricular premature beats (VPBs) are common findings and occur in broad spectrum of population including subjects with structurally normal hearts and those with cardiac disease, independent of severity [1]. The incidence of VPBs in subjects with structurally normal hearts varies according to observational studies. Incidence of VPBs was 7.8% in participants during evaluation of 12-lead ECGs in a large healthy military population, with a much lower incidence in age group below 20 (4.6%) compared to those older than 50 years of age (21.7%) [2]. Hinkle et al. [3] reported that the incidence of asymptomatic ventricular arrhythmias was 62% in a mixed population of healthy individuals and patients with known heart disease.

Introduction
Ventricular premature beats (VPBs), also referred to as ventricular premature complexes are early depolarization’s of ventricular myocardium arising in a variety of situations. VPCs/NSVT are common and occur in a broad spectrum of the population including patients without structural heart disease and those with any form of cardiac disease, independent of severity [1]. The incidence of VPBs in subjects with structurally normal hearts varies according to observational studies. Incidence of VPBs was 7.8% in participants during evaluation of 12-lead ECGs in a large healthy military population, with a much lower incidence in age group below 20 (4.6%) compared to those older than 50 years of age (21.7%) [2]. Hinkle et al. [3] reported that the incidence of asymptomatic ventricular arrhythmias was 62% in a mixed population of healthy individuals and patients with known heart disease.

Ventricular arrhythmias occurring in structurally normal hearts are labelled as idiopathic ventricular arrhythmias (VA) and accounts for approximately 10% of the patients with ventricular tachycardia (VT) [4]. Outflow tract arrhythmias are the most common type of idiopathic VA and more than 70-80% of idiopathic VTs or VPBs arise from right ventricular (RV) OT. Prognostic implication of VPCs may vary depending on underlying heart disease, left ventricular functions, age of patient and associated co-morbidities. Prognosis is usually favourable in patients with structurally normal heart and age below 30 years.

Also frequent isolated ectopic beats, mostly originating from the right ventricular outflow tract have been reported as a cause of tachycardia induced cardiomyopathy, a reversible form of congestive heart disease that resolves after elimination of the culprit arrhythmia either by medical treatment or by Radiofrequency Ablation (RFA). As most of VA arise from RVOT so our further discussion will primarily focus on ventricular arrhythmias arising from right ventricular outflow tract.

Prevalence
The prevalence of VPBs is directly related to the study population, method of detection and duration of observation. In patients with no known heart disease, VPBs occur in approximately 1 percent of routine 12-lead ECG of 30 to 60 seconds duration [2,4]. When 24-hour ambulatory monitoring is used, up to 80 percent of apparently healthy people have occasional VPBs [5,6]. The frequency of VPBs increases with increasing age and also in patients with underlying heart disease. There is an age-related increase in the prevalence of VPBs in normal individuals and those with underlying heart disease [2,6,7]. The occurrence of frequent VPBs accounting for more than 20 percent of overall heart beats is rare, seen in less than 2 percent of patients [8].

Symptoms
Ventricular ectopic activity is commonly encountered in clinical practice. Usually, it is not associated with life-threatening
consequences in the absence of significant structural heart disease. However, frequent ventricular ectopic beats can be extremely symptomatic and even incapacitating in some patients because of palpitations or dizziness. VPBs rarely cause hemodynamic instability, except when associated with severe left ventricular systolic dysfunction or when occurring with bradycardia.

VPBs are associated with several characteristic findings on history; physical examination and electrocardiogram. Symptoms variously reported include palpitations, pounding sensation in the neck, dyspnoea, dizziness, pre- syncpe, syncope, reduced exercise capacity and decreased quality of life. Other symptoms described by patients may include coughing, claudication and dysphagia.

Increased VPB/NSVT burden may cause tachycardia induced cardiomyopathy with heart failure symptoms. Cardiomyopathy induced by atrial tachyarrhythmias has been well described [9-12]. Although the mechanism for tachycardia-induced cardiomyopathy secondary to atrial tachyarrhythmias with fast ventricular activation is well understood, the mechanisms for PVB-induced cardiomyopathy are less clear. Besides LV dyssynchrony due to LBBB during PVBs, other causes such as increased oxygen consumption [13] have been implicated. Furthermore, the so-called apical-to-basal “squeezing effect” in systole during physiological activation of the LV is disrupted during VT that occurs later than the SR transition. A V2 transition ratio >0.6 predicted an LVOT origin with 95% sensitivity and 100% specificity. For patients referred for catheter ablation of OTVT, this simple ECG measurement might be performed in the office both to help plan an ablation strategy and to enhance patient counselling with regard to procedural time, potential outcome, and risks associated with arterial access, mapping, and ablation. One might argue that the V2 transition ratio is cumbersome for everyday use in clinical practice. In the electrophysiology lab, this measurement is easily made with digital calipers available on any clinical electrophysiologic recording system. For more practical clinical use a precordial transition during the PVB/VT that occurs later than the SR transition excludes an LVOT origin with 100% accuracy. This simple measure can be easily used by any cardiologist or electrophysiologist when counselling patients about PVB ablation.

In case of RVOT VPBs, 12-lead surface ECG is crucial for identifying the origin of PVBs/VTs from this anatomically complex region [21]. Resting 12-lead ECGs in sinus rhythm are usually normal, with up to 10% of patients presenting with incomplete or even complete right bundle branch block (RBBB).

How to Diagnose Origin of VPBs

Detailed intracardiac electrical mapping has demonstrated that the vast majority of outflow tract VPBs/VTs originate from the anterior and superior septal aspect of the right ventricular outflow tract (RVOT), just inferior to the pulmonic valve [17,18]. Less commonly, the site of origin can be localized to the right ventricular (RV) infundibulum, RV free wall, and posterior aspect of the interventricular septum. In approximately 10% to 15% of cases, the arrhythmia originates from the left ventricular outflow tract (LVOT) and can be mapped to the region of the aortic cusps [19,20] (1,8–10). Rarely, outflow tract VPBs/VTs can be ablated from within the anterior inter ventricular vein, aorto-mitral continuity, or the root of the pulmonary artery.

In addition to the overall frequency of VPBs, QRS duration as well as epicardial site of origin of VPBs appears to play a role in the development of cardiomyopathy and are associated with outcomes following catheter ablation. Wider QRS complexes appear more likely to result in cardiomyopathy with a lower overall burden of VPBs while also being associated with longer times to normalization of LV systolic function following ablation, while epicardial VPB origin also appears to predict delayed LV function recovery.

The incidence of PVB-induced cardiomyopathy is higher in older patients [16] and the pathophysiology is less well understood. It is often more difficult to determine whether VPBs are a result of reduced LV function or the cause. It is well described that PVBs/NSVTs are a common finding on 24-hour holter recordings in patients with ischemic or dilated cardiomyopathy. The indication for primary ICD implantation in these patients to reduce the arrhythmogenic risk for SCD is dependent on LV function and not arrhythmia burden. The increased risk for SCD is present even if no arrhythmias have been documented.
Typically, PVBs originating from the RVOT have an inferior axis with left bundle branch block (LBBB) morphology, and a late R/S transition at V4 in the precordial leads. A QRS duration<140ms is suggestive of a PVB with a ‘septal’ origin, whereas a QRS duration>140ms favours a ‘free wall’ origin, particularly when notches are seen in the down stroke of the QRS of the inferior leads [22].

Diagnostic Evaluation

The diagnostic evaluation of patients with symptoms suggesting VPBs includes an electrocardiogram (ECG) or ambulatory cardiac monitoring, if VPBs are not recorded in ECG. Also 24 hour holter monitoring is best accepted approach to quantifying the frequency of VPBs as a percentage of total heart beats and determine if they are monomorphic or polymorphic.

Echocardiography should be performed focusing on the presence or absence of underlying structural heart disease and ventricular systolic functions. It is important to distinguish the benign VPBs originating in the right ventricular outflow tract from those related to arrhythmogenic right ventricular cardiomyopathy, as the VPB morphology may be quite similar but the prognosis entirely different. Catheter ablation can be quite an effective treatment in the first case, while patients with the last condition often need an implantable cardioverter-defibrillator as protection from sudden cardiac death. ARVC should generally be suspected in patients with a family history of sudden death and/or T wave inversion in the right precordial leads.

Exercise treadmill stress test to evaluate the response of the VPBs to exercise, determine the VPB morphology, determine if sustained or non sustained ventricular tachycardia (VT) can be induced with exercise, as well as screen for underlying ischemia. Catecholamine-sensitive VPBs may increase during exercise, as well as those related to ischemia; more commonly, however, VPBs are suppressed during exercise and remerge during recovery phase. Catecholamine or exercise-induced VPBs respond well to beta blocker therapy.

Correctable causes or triggers should be sought by clinical history (inquiring about possible underlying cardiovascular diseases, but also about use of alcohol or caffeine-containing beverages, or illicit drugs, etc) and/or laboratory testing (electrolyte levels, thyroid stimulating hormone [TSH]). For documented nocturnal VPBs, sleep apnea needs to be considered and polysomnography performed, when indicated. Further testing is indicated only when this initial evaluation identifies significant abnormalities that require further evaluation.

Treatment

In persons with frequent VPBs, evaluation and management is based on symptomatic status and presence or absence of underlying structural heart disease which has prognostic significance and may require specific therapy. There is no clear evidence that VPB suppression with beta blockers or antiarrhythmic drugs improves overall survival in patients who have no symptoms and have not had a major arrhythmic event. Thus, the only indications for the use of beta blockers or antiarrhythmic drugs for VPB suppression are for symptomatic patients or for patients with cardiomyopathy felt to be possibly related to frequent VPBs.

According to the current guidelines for the management of symptomatic PVCs [23], beta-blockers are the drug of choice. However, the efficacy of beta-blocker therapy (namely atenolol and metoprolol) is generally modest, with a reduction of PVC burden between 10-25% [24,25]. The efficacy of calcium channel antagonists such as verapamil varies in several reports, with reasonable efficacy in patients with idiopathic VT [26-28] but it is less effective in patients with only PVCs, most likely as a result of different underlying mechanisms [25]. Following these, Class I antiarrhythmic drugs such as propafenone or flecainide are recommended. Although treatment with antiarrhythmic drugs in symptomatic patients with structurally normal hearts may be reasonable [23], they are contraindicated in patients with cardiomyopathy due to their proarrhythmic effects [29]. In these patients, beta-blockers and amiodarone are the only antiarrhythmic drugs available. However, due to the frequent and significant side effects associated with amiodarone [30], it should only be administered in patients refusing catheter ablation or after failed catheter ablation. Since catheter ablation of RVOT PVB/VTs is a highly effective therapy with a very low complication rate, this should be the treatment of choice.

Patients found to have underlying structural heart disease will typically receive medical therapy specific to their disease process. In many cases, this therapy will also reduce the frequency of VPBs. Examples of therapy which can reduce the frequency of VPBs are beta blockers, which improve survival in patients with a prior myocardial infarction or heart failure, and antihypertensive therapy, which may induce regression of LVH in patients with hypertension.

For patients with symptomatic VPBs in whom beta blockers or calcium channel blockers have not resulted in symptomatic improvement, additional therapeutic options include antiarrhythmic medication and radiofrequency catheter ablation. For most patients, either approach is a reasonable first choice. However, for patients with frequent VPBs associated with left ventricular dysfunction, radiofrequency ablation is preferred modality. In patients with high PVCs burden (>20%PVCs/24 hours) is associated with increased risk of developing left ventricular dysfunction so prophylactic ablation may be proposed even when patients are asymptomatic.

Outcomes of Radiofrequency Ablation

In general, the success rate of catheter ablation of arrhythmias originating from the RVOT is reported to be high [24]. In most studies, the acute success rate is reported to
be >80% [23]. Also after successful ablation the recurrence rate is generally not exceeding 5% even after long-term follow-up [23,31-35].

References


23. Aliot EM, Stevenson WG, Garrote JMA, Bogun F, Calkins CH, et al. (2009) EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). Europace 11(6): 771-817.


