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Original Article

Outflow tract ventricular premature beats ablation in the presence or absence of structural heart disease: Technical considerations and clinical outcomes

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ABSTRACT

Background: Premature ventricular beats (PVBs) are early depolarization of the myocardium originating in the ventricle. In case of very frequent PVBs, patients are severely symptomatic with impaired quality of life and are at risk of pre-syncope, syncope, heart failure, and sudden cardiac death particularly in the presence of structural heart disease. Ventricular outflow tracts are the most common sites of origin of idiopathic PVBs especially in patients without structural heart disease. We examined the role of radiofrequency catheter ablation in suppression of monomorphic PVBs of outflow tract origin in the presence or absence of structural heart disease, and its impact on improvement of left ventricular (LV) systolic function.

Methods: Thirty-seven highly symptomatic patients with PVBs burden exceeding 10% were enrolled, provided that PVBs are monomorphic, originating in ventricular outflow tracts and regardless the presence or absence of structural heart disease. Patients were divided into 2 groups according to PVB site origin (RVOT vs. LVOT). 3D electro-anatomical mapping modalities were used in all patients employing activation mapping technique in the majority of cases. Acute success was considered when PVBs completely disappeared or when residual sporadic PVBs ≤ 1 beats/min or ≤ 10 beats/30 min after RF ablation. Patients were followed up for a mean period of 5.4 ± 1.2 months with long-term success defined as complete disappearance or marked reduction by more than 75% in the PVBs absolute number on 24 h holter monitoring.

Results: Mean age of the study group was 39.9 ± 12.97 years, including 22 (59.4%) males. PVBs originated in RVOT in 17 cases and in LVOT in the remaining 20 cases. Prevalence of structural heart disease and consequently shortness of breath was higher in LVOT group. Initial ECG localization matched EP localization in the majority (94%) of cases. R wave duration index was the only significant independent predictor for RVOT origin with cut off value of <0.3 (P = 0.0057) upon multivariate analysis. Acute success was encountered in 32 (86%) patients with all cases of failure in the LVOT group. Recurrence occurred in 5 (15%) cases without significant difference between both groups. All cases of recurrence had residual PVBs at the end of the procedure. 18 cases out of the study group showed significant improvement of their EF (>5%) at the end of the follow-up period with no significant differences between both groups (p = 0.09). A linear correlation was observed between PVBs burden at follow up and magnitude of improvement of LV EF, particularly in patients with resting LV dysfunction and increased LV internal dimensions.

Conclusions: RF ablation is an effective and safe method for elimination of outflow tract PVBs irrespective of their origin and the presence or absence of structural heart disease. PVBs burden after ablation appears to be the main determinant for reversal of PVB induced myopathy particularly in those with increased LV internal dimensions.

Abbreviations: PVB, premature ventricular beats; LVOT, left ventricular outflow tract; RVOT, right ventricular outflow tract; SCD, sudden cardiac death; EF, ejection fraction; VT, ventricular tachycardia; EP, electrophysiological.

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1. Introduction

Premature ventricular beats (PVBs) are early depolarization of the myocardium originating in the ventricle. In patients with no underlying heart disease, PVBs are considered benign with very good prognosis. However, when PVBs are very frequent many patients are severely symptomatic with impaired quality of life and are at risk of pre-syncope, syncope, and heart failure. On the other hand, in the presence of structural heart disease PVBs represent increased risk of sudden cardiac death (SCD).[1,2]

ACC/AHA/ESC 2006 Guidelines for management of patients with ventricular arrhythmias and the prevention of SCD stated that beta blockers should be used as primary therapy in the management of ventricular arrhythmias and the prevention of SCD. In addition, ablation of asymptomatic PVBs may be considered when PVBs are very frequent; to avoid or treat PVB induced cardiomyopathy.[3]

PVBs originating in the ventricular outflow tract usually appear in patients without structural heart disease. They may present in the form of isolated or incessant PVBs, or as tachycardia (up to 80% of idiopathic ventricular tachycardia (VT)). The main causal mechanism is triggered activity, but re-entry or abnormal automaticity mechanisms have also been postulated. Beta blockers or Verapamil usually show only limited effectiveness in controlling this type of PVBs. Radiofrequency ablation can be effective, but is hampered by the fact that this PVBs has limited and unpredictable inducibility.[4,5]

We investigated the role of radiofrequency catheter ablation in suppression of monomorphic PVBs of outflow tract origin in the presence or absence of structural heart disease, and its impact on improvement of left ventricular (LV) systolic function.

2. Methods

2.1. Study population

Thirty-seven patients with very frequent (>10% PVBs burden documented on holter monitoring[6]) monomorphic PVBs, originating from right or left ventricular outflow tracts, in the presence or absence of structural heart disease, who are still symptomatic despite antiarrhythmic therapy including beta blockers, were enrolled in the current study. Patients with concomitant atrial arrhythmias, thyrotoxicosis, hypertrophic cardiomyopathy with septal thickness exceeding 14 mm, non-revascularized coronary artery disease, heart failure patients with NYHA class 3 or 4, and those with non-outflow tract PVBs were excluded.

2.2. Methodology

Detailed history (symptoms, full medical treatment, and family history of SCD), full clinical examination, and laboratory investigations (serum electrolytes, thyroid profile) for exclusion of reversible PVBs causes were done in all cases.

Standard 2D echocardiographic examination was done at baseline to exclude structural heart disease, occasionally after the procedure in case of suspected complications, and after 6 months of follow up of LV systolic function (calculated by modified Simpson method). Improvement of EF $\geq$ 5% compared to baseline was considered significant for further statistical analysis.

Twenty-four hours ambulatory ECG monitoring was done for assessment of absolute PVBs number, PVBs burden (calculated as number of PVBs/number of total heartbeats per 24 h $\times$ 100), and to exclude other life threatening arrhythmias. Holter monitoring was repeated 6 months after ablation or whenever significant symptoms were encountered for detection of early or late recurrence.

2.3. Twelve lead ECG

After exclusion of myocardial ischemia, initial localization of PVBs origin was done using different algorithms including PVB transition in chest leads, V2 transition ratio (calculated as the percentage R-wave during VT: (R/R + S) VT divided by the percentage R-wave in sinus rhythm (SR): (R/R + S)SR), and R wave duration index (calculated by dividing the QRS complex duration by the longer R wave duration in lead V1 or V2). PVB duration, and coupling interval and axis in inferior leads were also recorded.[7–9]

2.4. Electrophysiological study (EP study) and radiofrequency (RF) ablation

EP study and ablation were done under local anesthesia after stoppage of antiarrhythmic drugs for at least 6 half-lives. Systemic anticoagulation was maintained by intravenous administration of heparin (initial bolus of 75 U/kg IV followed by 1000 U per hour) throughout the procedure.

Three Dimensional electro-anatomical mapping was done for all cases using either the CARTO 3 mapping system (Biosense, Diamond Bar, CA, USA) or the Ensite NavX® system (St Jude Medical, Inc, St Paul, MN) according to physician preference and availability. Three Dimensional compatible ablation catheters, (Thermocouple 4 mm tip 7F for Ensite NavX system and Thermocool 3.5 mm 8F for CARTO 3 system) were used. In addition a multi-electrode (quadripolar or decapolar) catheter was introduced into the RV (apex or RVOT) to be used for pacing and as a reference catheter if needed.

2.5. Mapping techniques

In case of frequent PVBs an activation map during PVBs of the chamber of origin was created; the ventricle was plotted by dragging the mapping catheter over the endocardium. The site of earliest ventricular activation (red isochrones in CARTO 3 or white isochrones in NavX) with a local ventricular electrogram preceding the surface QRS onset by at least 25–30 ms was targeted by ablation. Voltage maps were also created especially in patients with structural heart disease for identification of scar tissue (areas with local voltage < 0.5 mv).

Mapping was always started in the RV even if PVBs were suspected to originate from LV. Mapping timing reference was either stable multi-electrode catheter as coronary sinus decapolar catheter for NavX system, or 12 lead surface ECG for CARTO 3 system.

In cases of infrequent PVBs encountered, drug provocation with epinephrine (0.1 mcg/kg/min) was used. If PVBs remained infrequent, pace mapping protocol was performed at different locations within the designated chamber. Ablation was attempted at sites with perfect pace maps 12/12 in comparison with 12 lead ECG recording of clinical PVBs.

2.6. Ablation, post ablation study and follow-up

RF energy was delivered in a temperature-controlled mode for 60–120 s at each ablation site with a maximum temperature of 48°c and a maximum power of 30–50 W. In case of aortic cusp origin, coronary angiography was done first and radiofrequency was adjusted to maximal power of 30 W.

Acute success was considered when PVBs completely disappeared or when residual sporadic PVBs $\leq$ 1 beats/min or $\leq$10

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