Arrhythmogenicity of radiofrequency ablation: Symptomatic non-sustained ventricular tachycardia and frequent premature ventricular ectopics after accessory pathway ablation

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A B S T R A C T

Radiofrequency ablation (RFA) has emerged as the preferred treatment modality with high success rate in cases with WPW syndrome. Arrhythmogenic complications are rarely reported after RFA, except for early or late recurrence of accessory pathway (AP) conduction. We present a unique case where the AP was successfully ablated, however, a new monomorphic PVC of similar morphology to the pre-excited beats developed within 30 min of RFA. She required medical management with sotalol to overcome her worsening symptom on follow-up. The ectopics resolved after 4 months.

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1. Introduction

Radiofrequency catheter ablation (RFA) has emerged as preferred modality of treatment for symptomatic Wolff Parkinson White (WPW) syndrome. Premature ventricular contractions (PVC) in a structurally normal heart is another pathology which is well managed by RFA when medical therapy fails. They usually originate from outflow tract and valve annulus of both right and left ventricle. The association of these two conditions or one presenting after an overt pathway is ablated is not very common. We present a unique case where PVC started soon after successful ablation of accessory pathway. The patient reported back with very frequent ventricular ectopics and occasional NSVT which made her symptomatically worse than her original paroxysmal palpitation due to atrioventricular reentrant tachycardia.

2. Case

A 46-year-old lady presented to out-patient department with recurrent episodic palpitations. Her baseline ECG showed positive delta in limb lead I with R/S > 1 in V1 suggestive of left sided pathway. (Fig. 1A). She was advised electrophysiology study (EPS) and radiofrequency ablation (RFCA).

Informed consent was taken before procedure. After stopping anti-arrhythmic drug for three days, EPS was performed under fasting state with local anaesthesia. Anticoagulation during procedure was maintained using Intravenous Heparin. One quadripolar and one decapolar diagnostic catheter was placed into right ventricular apex and coronary sinus (CS) respectively through right femoral vein. We paced the atria rapidly and noted maximal pre-excitation suggestive of left posteroseptal AP. After baseline EPS, tachycardia was induced by programmed stimulation with 2 atrial premature depolarisations (APDs) from CS. It was a short VA tachycardia with eccentric activation, AEGM (electrogram) in CS 5–6 being earliest (Fig. 1B). The response to ventricular overdrive pacing (VOP) was VAHV and his synchronous PVC (HSP) terminated the tachycardia. Thus, it was an orthodromic
Fig. 1. A. Baseline ECG suggestive of WPW syndrome. B. Maximum pre-excitation during CS pacing. C. Tachycardia intracardiac EGM.

Fig. 2. A. During RFA Intracardiac EGM shows separation of AV and disappearance of preexcitation. B. Fluoroscopy in LAO 30° of the successful ablation site. C. Fluoroscopy in RAO 30°, of the successful ablation site.

Fig. 3. A. Post RFA, PVC appeared on table during waiting period. Note, the morphology is same (12/12 match) with the maximum preexcited beats in Fig. 1B. B. Intracardiac shows negative HV, with QRS onset appearing much before A signal, hence, ruling out PAC and confirming diagnosis PVC.
atrioventricular reciprocating tachycardia (AVRT) mediated by left posterior accessory pathway. We accessed the mitral annulus antegradely using trans-septal approach. The annulus was mapped with a 7F, 4 mm tip, B curve, ablation catheter (Biosense Webster, USA) and SL-1 sheath (St. Jude Medical, Saint Paul, MN, USA). The earliest signal was defined using shortest AV/VA interval with QS-pattern on unipolar electrogram (EGM) at left posteroseptal area (Fig. 2A, B, C). Once the site was confirmed we delivered RF energy while pacing atria from CS 7–8 to monitor maximal pre-excitation. The manifest pathway conduction disappeared after 3 sec and we consolidated the lesion using same 40 W and maximum temperature of 60 °C (non-irrigation). After RF application, there was no accessory pathway ventriculoatrial (VA) conduction at ventricular pacing. A ventricular programmed pacing showed no VA conduction of AP and AV node, and no other SVT was induced by an atrial and ventricular programmed stimulation. While we were planning to shift out the patient after removing catheters, she developed frequent PVCs. The morphology of those beats were identical to her pre-excitation pattern (Fig. 3A). The possibility of premature atrial complex (PAC) with conduction via AP (with reappearance of antegrade conduction) was suspected. The catheters were repositioned to confirm the diagnosis. The HV interval of wide QRS beat was negative. The QRS onset preceded the atrial EGM ruling out PAC with AP conduction (Fig. 3B). Intravenous adenosine was given which showed transient AV block without any antegrade pathway conduction.

The treatment options were mapping and ablation of the PVC or medical management. It was a shared decision to try conservative management. The patient was discharged next day with few PVC and minimal symptoms on tablet bisoprolol (5 mg once daily). However she became very symptomatic and visited ER with frequent PVCs. The morphology indicated that the ectopics were originating from areas around the ventricular insertion site of pathway. We evaluated ablation signals and noted that during initial ablation we were predominantly on atrial side but while delivering consolidation lesions we would have gone more on ventricular aspect. We advised 24 hours holter to quantify VPC burden. Her holter monitoring recorded frequent PVC, bigeminy, couplets and occasional NSVT (Total PVC burden 57%) [Appendix 1]. We again discussed about EP study and ablation but the patient wished to continue anti-arrhythmic drugs (AADs). We started her on tablet sotalol (40 mg twice daily). Over 3 months she experienced gradual improvement in her symptoms. As the PVC burden reduced to <1% after 4 months, sotalol was discontinued. She is asymptomatic at 9 months follow-up.

3. Discussion

The above case is a unique experience where our patient had worsening symptoms despite successful intervention. This happened due to appearance of frequent PVC and occasional monomorphic NSVT.

WPW syndrome is known to have arrhythmic associations. It is reported that 15% of adults and children followed up for 10 years had atrial fibrillation (AF) and 0–0.6% suffered sudden cardiac death (SCD) per year due to ventricular fibrillation [1–4]. Some of these findings were traced to genetic links [5,6]. There is enough data suggesting remission of paroxysmal atrial fibrillation after AP ablation [7].

There has been case reports of accessory pathway co-existing with PVCs which subsided with single ablation [8]. This was probably due to disturbance of electrical activity of focal cardiac muscle cells close to ventricular insertion of pathway. In our case the PVCs appeared only after ablation. Though the exact mechanism is not known, it is presumably a result of spontaneous firing of ventricular myocytes in the area of ablation. We felt this is similar to junctional automaticity during AVNRT or atrio-fascicular pathway ablation. The other possibility is micro-reentry. The RF lesion has ragged edges which is less clearly demarcated from underlying normal myocardium. This produces non-uniform anisotropy and slow conduction resulting in reentry tachycardia [9]; [10]. Mujovic et al. has elaborated on new onset arrhythmias after AP ablation in a cohort of 124 patients in whom 7.3% were PVC [11]. When we analysed this group we noted 2 patients had septal

Appendix. 24 Holter after ablation suggestive of frequent monomorphic PVC and bigeminy.
pathway ablation which were followed by PVCs starting at least after one month. Our case had immediate appearance of PVC which was managed medically. The arrhythmia spontaneously resolved similar to triggered VPCs/right ventricular outflow VT (RVOT VT). The final answer to define mechanism will require further research involving more patients with this kind of presentation.

Author contributions
DB and AK performed the case. DB and SSM wrote the manuscript and developed the images. SM and RS offered significant clinical inputs during the case and writing.

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Consent
Consent has been taken from patient.

Declaration of competing interest
None.

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