Chapter 13
Slowly Conducting Posteroseptal Accessory Pathway

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Abstract Long RP narrow QRS tachycardia with negative P waves in the inferior leads, in addition to CS ostium AT and atypical AVNRT be aware of PJRT.

PJRT

A 32-year-old lady, with frequent episodes of narrow QRS tachycardia and failed ablation elsewhere, presented to out clinic for more evaluation and redo ablation.

ECG revealed episodes of narrow QRS tachycardia, with rate about 110 bpm and long RP-short PR, negative P wave in the inferior leads. Patient had a history of frequent and long-standing episodes of palpitations. Echocardiography revealed mildly reduced LVEF.

EPS was done, using three quadripolar catheters, for recoding HRA, His and RV potentials, and a decapolar catheter for recording CS potentials. A long steerable sheet (Agillis) was used for the passage of the ablation catheter.

With AES a narrow QRS tachycardia was induced with a rate of about 110 pbm compatible with patient's clinical arrhythmia, with long RP-short RP; and 1:1 AV association. The earliest site of atrial activation was in CS 9–10; but VA signal was not so fused.

His refractory ventricular pacing was done during arrhythmia and resulted in the advancement and resetting of arrhythmia, consistent with the presence of a concealed AP's corporation in the arrhythmia (Fig. 13.1).

Another His refractory PVC resulted in the termination of the arrhythmia (Fig. 13.2).

HV interval was normal during sinus rhythm and with atrial overdrive pacing; suggesting that the accessory pathway had only retrograde conduction.

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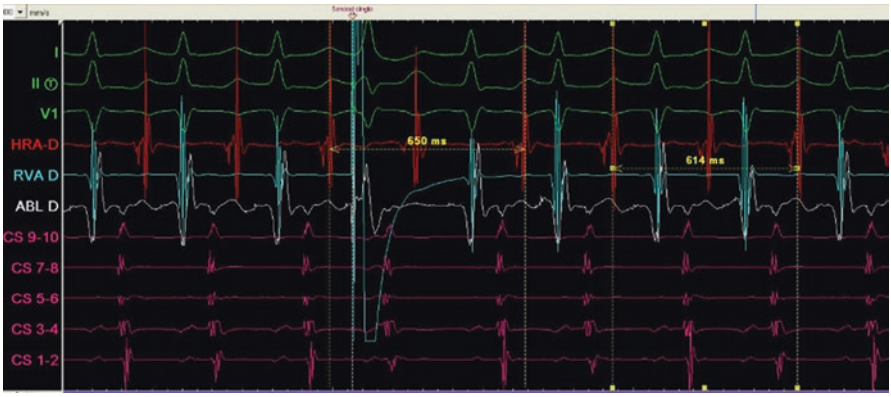

Fig. 13.1 His refractory pacing resulted in advancement and resetting of the arrhythmia infavor of the presence of an accessory pathway that is incorporating in the arryhthmia

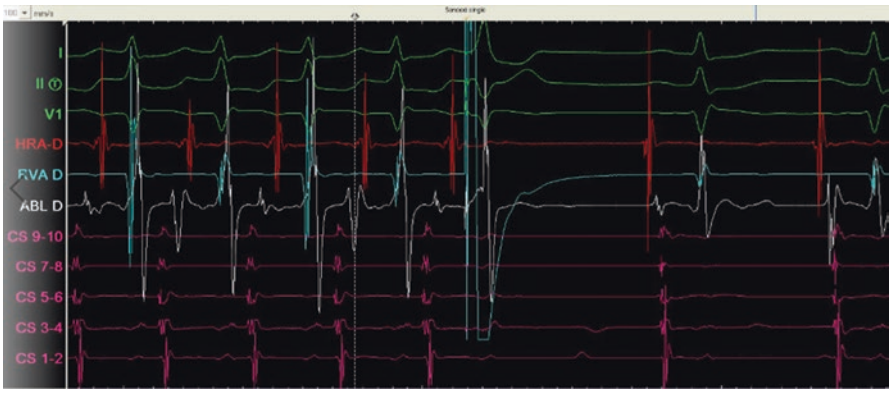

Fig. 13.2 Arrhythmia termination with His refractory PVC, that is strongly in favor of accessory pathway-mediated tachycardia that was PJRT in this case

Entrainment of the arrhythmia with ventricular overdrive pacing during arrhythmia was done and resulted in VAV response.

Also, His refractory ventricular pacing revealed the presence of an accessory pathway, but ventricular extra stimulation showed decremental conduction, so decremental, slowly conducting AP was considered as culprit pathway, resulting in PJRT.

In addition, AH interval during atrial pacing (after Isuprel infusion) at the tachycardia cycle length was within 20–40 ms of the tachycardia in PJRT, in contrast with atypical AVRNT, that is more than 40 ms (Fig. 13.3).

Results of differential septal sequential pacing were not conclusive.

Mapping of the right posteroseptal area for finding the earliest A was begun, after meticulous mapping, RF application in the site of atrial activation immediately

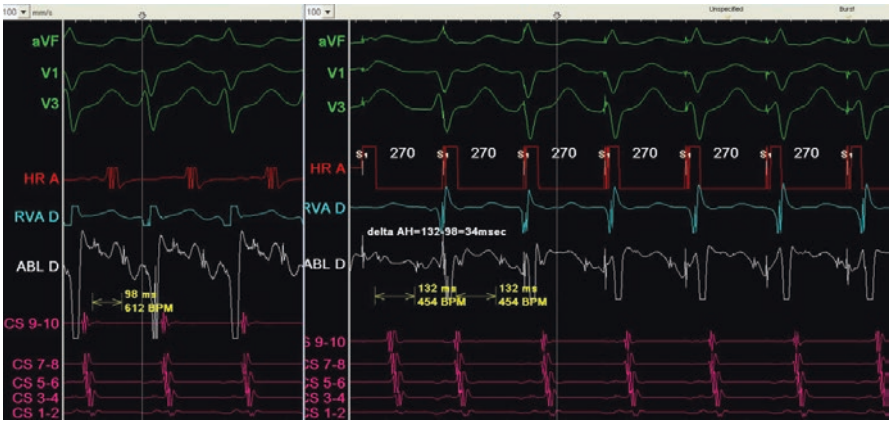

Fig. 13.3 AH interval during HRA pacing is within 20–40 ms of tachycardia AH interval (34 ms in this case)

resulted in the termination of the arrhythmia. Ventricular pacing showed VA dissociation immediately and after 30 min.

Half an hour after ablation VA was dissociated.

Discussion

Posteroseptal accessory pathways account for 25–30% of APs. Because of the complex anatomy of the posteroseptal area ablation of these types of APs is time consuming with greater fluoroscopic time [1].

The AP is located in the posteroseptal RA or CS proximal in 76% of cases, in 12% in mid septal location, and in the remainder is in right or left posterior or lateral [2, 3].

CS diverticula are present in about 9–10% of patients [3].

Patients often present with arrhythmia due to posteroseptal accessory pathways, but the presence of the CS diverticulum is not as reason for the presence of AP.

There are some electrocardiographic clues for discrimination of right from left septal accessory pathways in the WPW cases with antegrade conduction; such as negative or isoelectric delta wave in V1, with abrupt transition in V2 and deeply negative delta in III and aVF; but in left side delta is positive in V1 or $R > S$ in V1 is present, and aVF is less negative. These are not discriminating during orthodromic reciprocating tachycardia or in concealed accessory pathways [2, 3].

PJRT manifests as long RP tachycardia, for more than 12 h/day with typically inverted *P* waves in the inferior leads [4].

About 80% of the slowly conducting and decremental accessory pathways responsible for PJRT are located at the posteroseptal region [5, 6].

The differential diagnosis for PJRT is atrial tachycardia of the CS ostium and atypical from of AVNRT (fast–slow) type [7].

With ventricular overdrive pacing a VAAV post-pacing response indicates AT and could rule out PJRT.

With His refractory pacing, the atrial activation advancement or delay or termination of the arrhythmia without conduction to the atrium are in favor of PJRT, and could rule out atypical AVNRT and AT.

Differential septal sequential pacing as a variant of septal versus apical RV pacing has not been studied in patients with decremental APs [8].

In addition, AH interval during atrial pacing at the tachycardia cycle length is within 20–40 ms of the tachycardia in PJRT, in contrast with atypical AVRNT, that is more than 40 ms.

During ablation in long RP tachycardia, the target site for ablation is mostly the right posteroseptal area. There is another clue for the right target site for ablation, that is the presence of a sharp followed by blunt component of the earliest retrograde atrial electrogram, in contrast with blunt followed by sharp electrogram suggesting the left site of the septum as the target.

The shortest VA time and negative unipolar atrial electrogram are the best targets for ablation.

Despite the complex nature of the anatomy of the posteroseptal area, the success role for ablation is about 93%. Rarely heart block could be occurred due to damage to the AV nodal artery [9].

For ablation within the coronary sinus, the proximity to the right coronary artery may be problematic, and in such situations, coronary angiography is recommended. In sites with less than 2 mm distance of the coronary artery, cryoablation is the choice.

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