The Importance of Previous Ablation

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Introduction

A 24 year old male had a witnessed cardiac arrest at home and was successfully resuscitated by the ambulance crew and subsequently cooled on ITU. Initially there were concerns about his neurological status, however, these had resolved by the time he was transferred to our institution.

The patient had no previous symptoms suggestive of pre-existing heart failure. He had never had palpitations, presyncope or syncope and there was no family history of sudden cardiac death. CMR performed 7 days post arrest demonstrated a degree of biventricular dysfunction and dilation thought to be secondary to the arrest. There was no late gadolinium enhancement to suggest the presence of a cardiomyopathic process.

The 12 lead ECG (Figure A) showed sinus rhythm with normal QT interval, no evidence of pre-excitation, normal R wave progression, no J point elevation suggestive of Brugada Syndrome, no evidence of Epsilon waves and unremarkable T wave morphology. Early repolarisation (see arrows - Figure A) in the inferior, and intermittently in the lateral, leads was noted. While early repolarization is known to be associated with idiopathic VF, it is also recognized to be a normal variant. His treadmill exercise test was normal as was the Ajamaline challenge.

A diagnosis of idiopathic VF was made and a dual chamber ICD was implanted without complication.

Procedure 1

Six weeks after ICD implant he was admitted with multiple shocks from his device. Interrogation showed tachycardia with a 1:1 relationship of cycle length 260ms/230bpm – well within the VF zone. The stored VEGM morphology closely matched that of his sinus rhythm EGM.

He was taken to the lab for an EP study. Quadripolar catheters were advanced to the HRA and RV, hexapolar to the His and decapolar to CS, all via the RFV. Baseline intracardiac intervals were normal. Ventricular pacing showed eccentric atrial activation. Retrograde conduction was non-decremental. Anterograde conduction was decremental through the AV node. Figure B shows narrow complex tachycardia induced during catheter manipulation with CL 285ms with earliest retrograde activation in CS 3-4 (see arrow – Figure B). Atrial advancement was demonstrated with HSVPB,
ventricular entrainment resulted in a VAHV response. These findings were consistent with orthodromic AVRT utilising a left free wall accessory pathway. Trans-septal puncture was performed; an irrigated ablation catheter was placed on the lateral mitral valve annulus at approx. 3 o’clock (Image 1). Ablation was performed during V pacing with initial VA prolongation then a shift in activation to a concentric pattern with the earliest atrial activation on the His catheter shown on the last two beats in Figure C. Concentric decremental VA conduction persisted for 25 minutes before the catheters were removed.

Procedure 2

Two months later he returned with further shocks from his ICD. Interrogation showed a 1:1 tachycardia again with the same VEGM morphology to sinus rhythm and it was assumed the accessory pathway had recovered. Back to the lab we go.

Quadripolar catheters were once more advanced to the HRA and RV, hexapolar to the His and decapolar to CS. Narrow complex tachycardia was easily induced, shown in Figure D. This time, however, note the completely different VA interval compared to the orthodromic AVRT seen in Procedure 1. Earliest atrial activation now appears on the catheter in the RA (probably closer to the annulus given the far-field V signal) – certainly not consistent with AVRT utilising a left lateral accessory pathway.

Further testing was performed. HSVPB had no effect on the atrial activation time which would be expected in all forms of AVNRT but would be expected to advance or delay the AEGM in AVRT. Ventricular entrainment resulted in a clear VAHV response. The SA-VA difference was 60ms, see González-Torrecilla et al (JCE, 2011) and the ventricular entrainment, González-Torrecilla et al (Heart Rhythm, 2006) with cPPI – TCL = 100ms, both consistent with an accessory pathway.

The next manoeuvre performed was para-hisian pacing (Hirao et al, Circulation, 1996) - Figure E; note His only capture on the first beat which is clear when comparing Stim-RVA EGM (see green arrow – Figure E) to Stim-RVA on subsequent beats (Obeyesekere et al, JCE, 2011). This also suggests the presence of an accessory pathway because the difference in Stim->A time is only 20ms. This extension reflects longer intraventricular activation time of the V at the AP site due to loss of Purkinje fibre capture (seen by Stim-V prolongation when His capture is lost i.e. S3V3 – S2V2 = 20ms).

So where is it? The VA interval is long in the CS, long at the His and long on the catheter in the RA. What next?

Figure F shows ventricular pacing with the decapolar catheter pushed as far around the CS as possible seen in the top right of Image 2. It now becomes apparent; retrograde atrial conduction shows eccentric activation with double potentials (A & A’) on the decapolar catheter.
Ablation at the site of earliest atrial activation, anterolateral in the LAO projection, abolishes the pathway. 12 months later the patient remains asymptomatic.

DISCUSSION

This case is an example of localised block in the mitral isthmus location caused by a previous procedure hindering location of the same or another nearby accessory pathway. Testing performed suggested the presence of an accessory pathway; VAHV response negative for AT, para-hisian pacing and ventricular entrainment were negative for AVNRT and positive for an accessory pathway. There was a long VA in all intracardiac EGMs until the decapolar catheter was pushed further in to CS which demonstrated double potentials. Admittedly a single endocardial ablation lesion placed on the MV annulus generating this level of block is unusual. Typically multiple lesions using irrigated ablation with high power, high flow rates and application of energy to the endocardium and epicardium via the CS are required for complete mitral isthmus block.

This case report highlights the importance of comprehensively scrutinising all prior procedures.

Figures and Images

Figure A - 12 lead ECG.

Figure B – Orthodromic AVRT.

Image 1 – Ablation site in the left anterior oblique projection.

Figure C – V pacing post ablation.

The first three beats show fusion with sinus rhythm.

Figure D – Narrow complex tachycardia.

Figure E – Para-Hisian pacing.

Figure F – V pacing with decapolar catheter pushed around CS

Image 2 – Decapolar catheter pushed around to anterolateral CS.

References


