INNOVATIVE TECHNIQUES

COMPLEX CASE STUDY

Unmasking Primary Atrial Myopathy During Ablation of Atypical Flutter

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ABSTRACT. A patient without any cardiac history and a structurally normal heart was referred for symptomatic “atrial flutter.” Standard activation and entrainment techniques confirmed the presence of an atypical right-sided flutter. Electroanatomic mapping uncovered the presence of extensive right atrial myopathy and aided with disclosing the location of the critical isthmus and its subsequent ablation.

KEYWORDS. Atypical Flutter, Atrial Tachycardia, Atrial Myopathy.

A 51-year-old man with no medical or cardiovascular history presented with persistent “atrial flutter” of unknown duration (Figure 1). The patient did not have any record of atrial fibrillation or obstructive sleep apnea. Non-invasive cardiac evaluation was unremarkable; normal left ventricular function and chamber sizes were confirmed. Owing to sustained symptoms of tachycardia, fatigue and diminished exercise tolerance he was referred for catheter ablation. A duodecapolar catheter was placed posterior to the tricuspid annulus (TRA) from the right femoral vein; a decapolar catheter was inserted into the coronary sinus from the right internal jugular vein; and a hexapolar catheter was placed across the TA into the right ventricle from the left femoral vein. After catheter insertion it became apparent that the presenting rhythm was atrial tachycardia (AT) with a cycle length of 280 ms. The typical “high–low” activation sequence characteristic of typical atrial flutter was not seen. To determine the exit site location, entrainment mapping was performed first from the distal coronary sinus, which resulted in manifest fusion and a lengthy post-pacing interval (Figure 2a); differential pacing confirmed the origin of the AT to be in the right atrium (Figure 2b).

Most notable however was the absence of any electrogram activity on the distal electrode pairs of the duodecapolar catheter. To exclude recording equipment errors, the catheter to cable and cable to pinbox connectivity was verified to be operational. Contact with the ablation catheter (open irrigated tip, 3.5 mm) in the region of interest confirmed this area to be indeed electrically silent. Anatomically, this area corresponded to a broad region of the crista terminalis affected by scar. Subsequently, point-by-point voltage and activation mapping of the right atrium was conducted with CARTO3 system (Biosense Webster, Diamond Bar, CA) creating a three-dimensional shell of the right atrium.1 The acquired image reflects the presence of a large band of scar extending from the area just inferior to the superior vena cava to anterior to seven o’clock on the TRA. Further mapping of the cavotriscuspid isthmus uncovered islands of scar with high voltage channels coursing through them; fragmented (Figure 3a–c) electrograms were observed in these locations, which proved to be earliest in activation (Figure 4). Ablation 0.5 cm posterior to the TRA between four and five o’clock terminated the AT on the second application (Figure 5). Despite aggressive attempts at reinduction, the AT remained non-inducible. The patient had normal sinus node function. At 4 weeks’ follow-up the patient had not had any recurrence and was off drug therapy. The etiology of the atrial myopathy remains unknown and could not be readily explained.2

In conclusion, a previously asymptomatic right atrial myopathy spawned AT in this patient. The presence of...
Figure 1: Twelve-lead electrocardiogram of the presenting rhythm initially suggests atrial flutter.

Figure 2: Entrainment from the distal coronary sinus results in a long post-pacing interval (a); this interval is significantly shorter when pacing from the low right atrium (b). LAS: halo catheter positioned behind tricuspid annulus; HiS: septal leaflet of tricuspid valve; RVA: right ventricle; ABL: ablation catheter.
Figure 3: An activation map of the right atrium in a right anterior orthogonal view depicts the distribution of scar along the crista terminalis (grey) tissue (a). A caudal view of the same map indicates that earliest activation (red) is posterior to the tricuspid valve annulus (b). A propagation map represents slowed conduction (red) through this zone (c).

Figure 4: Double arrows point to a highly fragmented electrogram that was activated earliest and at the site of termination.
myopathy in the left atrium could not be excluded as this chamber was not explored. Entrainment pacing and three-dimensional mapping were complimentary in identifying the critical isthmus and its subsequent ablation.

References


Figure 5: The arrow points to the site of termination on the electroanatomic map. Red denotes application of ablation lesions. The intracardiac recordings reflect prompt restoration of sinus rhythm after the second application.