CASE REPORT

Ectopic nodal structures in a patient with atrial tachycardia originating from the mitral valve annulus

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We report a case, which we believe to be rare, of adenosine-sensitive atrial tachycardia (AT) originating from the mitral valve annulus. The patient, a 73-year-old woman, died of unrelated cause 4 years after radiofrequency (RF) ablation therapy. Histologically, fibrous replacement of atrial musculature by mature collagenous tissue produced by the RF current was observed at the left inferior atrioventricular junction. In serial sections that included the coronary sinus, two distinct nodal structures containing small, pale myocytes within the fibrous tissue matrix were identified around the region of the ablation lesion. Our case appears to be a unique representation of tissue that was associated with the occurrence and maintenance of AT.

KEYWORDS
Atrial tachycardia; Ablation; Histology; Nodal tissue

Case report

A 73-year-old woman with diabetic nephropathy was admitted for radiofrequency (RF) catheter ablation to treat a paroxysmal supraventricular tachycardia, which occurred frequently during dialysis. During electrophysiological study, the tachycardia was easily induced with a single premature extrastimulus and was terminated by rapid intravenous injection of adenosine triphosphate. The earliest atrial potential was recorded 2 cm distal to the orifice of the coronary sinus (CS) (Figure 1A, CS5–6). The atrial activation sequence during the tachycardia was absolutely different from that during ventricular pacing. The diagnosis was adenosine-sensitive atrial tachycardia (AT). The CS potential split into two components after five deliveries of RF energy (50°C) to the earliest site in the CS (Figure 1B). We assumed that the first potential corresponded to the left atrial component and that the second potential corresponded to the CS component. Although AT was temporarily terminated by the delivery of RF energy to the CS, it could be induced by atrial extrastimuli. One week after the procedure, we confirmed that no thrombus remained in the left atrial appendage and undertook a second procedure, when RF energy was delivered to the left atrial endocardium close to the inferior mitral valve annulus. AT was successfully terminated by a single delivery of RF energy (50°C, 60 s) and could not be re-induced by extrastimuli. The patient experienced no AT during dialysis after the second procedure. She died of unrelated sepsis 4 years later. At autopsy, the heart weight was 575 g. Macroscopically, an oval-shaped (15 × 7 mm) sunken lesion was observed adjacent to the inferior mitral valve annulus (Figure 2). Histologically, replacement of atrial myocardium and CS musculature by mature collagenous tissue produced by the RF ablations was observed at the left inferior atrioventricular junction (Figure 3). In serial sections of the left inferior AV junction, including the orifice of the CS, two nodal structures were observed, one in the septal area and the other in the anterior area of the ablation lesion (Figure 2). One was 2 × 1 mm and was located at the epicardial surface of the posterior wall between the openings of the CS and right inferior pulmonary vein 11 mm posterior to the ablation lesion (Figure 4). The other was 0.7 × 0.7 mm and was located on the mitral valve annulus 10 mm anterior to the ablation lesion (Figure 5). Both of the nodal tissues contained abundant fibrous interstitium insulated from the adjacent atrial musculature. Cells were significantly smaller than normal atrial myocardial cells.

Discussion

Focal AT is characterized by rhythmic atrial activation arising from a small area; the AV annulus is one site of...
origin. According to the recent clinical reports, AT arising
from the AV annulus is generally sensitive to verapamil and
adenosine and occurs predominantly in the tricuspid valve
annulus.2,3 AT arising from the left AV annulus is relatively
rare; histological studies have not been reported.4–6
Although the anatomic substrate of adenosine-sensitive AV
annular AT has not been elucidated, participation of acces-
sory AV nodal structures, remnants of the embryological
specialized AV ring tissue, has been suggested.3,7 Despite
results showing a discrepancy between electrophysiological
properties and histological characteristics of the AV ring
tissue,8 histological observations of the tricuspid valve

Figure 1  Surface ECG leads II and V1 and intracardiac electrograms from the high right atrium (HRA), His-bundle region (HIS), CS (CS1, distal; CS10, proximal), and ablation site (ABL) during AT. (A) The earliest atrial potential (CS5–6) was recorded 2 cm distal to the CS ostium (inset). (B) The CS potential showed two components after delivery of RF energy to the earliest site in the CS (inset).

Figure 2  Macroscopic view of the ablation lesion in the left atrial endocardium. An oval-shaped (15 × 7 mm) sunken lesion is seen adjacent to the inferior mitral valve annulus (arrows). Nodal structures are located on either side of the ablation lesion (blue ovals). Ao, aorta; His, His bundle; LA, left atrium; LAA, left atrial appendage; LPV, left pulmonary vein; RA, right atrium; RPV, right pulmonary vein; TV, tricuspid valve; VS, ventricular septum; MV, mitral valve.

Figure 3  Radiofrequency ablation lesion (1 cm from the opening of the CS). Atrial myocardial cells have been completely replaced by mature collagenous fibres indicated by arrows (bar = 1 mm, elastica van Gieson stain). LA, left atrium; LV, left ventricle; MCV, middle cardiac vein; MV, mitral valve; CS, coronary sinus.
annulus support the existence of an ectopic nodal structure.\(^7\)

The three histological criteria for a conduction tract, proposed by Mönckeberg and Aschoff in 1910, appear to remain valid: (i) the cells comprising the proposed tracts should be histologically distinct from their neighbours, (ii) it should be possible to trace the tract through serial sections, and (iii) the cells within the tract should be insulated from the neighbouring myocardium by sheaths with the appearance of fibrous tissue.\(^5,10\) We believe that the present case is the first of multiple ectopic nodal tissues in a patient with adenosine-sensitive AT.

The nodal structure located between the CS and the right inferior pulmonary vein may be a remnant of the left-sided primitive pacemaker tissue of the progenitors of the superior vena cava.\(^11\) The right-sided primitive pacemaker tissue forms the sino-atrial node, and the left-sided tissue projects to an area near the CS, as seen in the present case.

The arrhythmogenic substrate in this case was eliminated by total fibrotic replacement of the ablated tissue, which completely eradicated the AV junctional structure. We had difficulty recognizing the first ablation lesion in the superior CS musculature and the second lesion in the left inferior atrial musculature. Although the mechanism of AT, whether automaticity, triggered activity, or re-entry, was not clear in this case, the induction of AT by a single premature extrastimulus supports re-entry\(^4\) as the mechanism. It is our opinion that the nodal structure of the left AV annulus comprises part of the re-entrant circuit of adenosine-sensitive AT, and a critical part of the AV junction was completely replaced by fibrous tissue in our patient. McGuire et al.\(^8\) noted a sleeve of AV nodal-type tissue, which responded to adenosine, surrounding the tricuspid and mitral annuli. This AV nodal-type tissue may be a substrate for adenosine-sensitive AT arising from the mitral valve annulus.

References