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Pacemaker lead-related macroreentrant atrial tachycardia

Frederico Scuotto[*], Maria Cecilia Xavier Souto, Claudio Cirenza,
Angelo Amato Vincenzo De Paola

Federal University of Sao Paulo, SP, Brazil

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ABSTRACT

Macroreentrant atrial circuits are frequently associated with scarring. Previous reports have shown the possible development of scar tissue that is adjacent to pacemaker (PM) leads. However, reports of PM lead-related reentrant tachycardia are scarce. We report the case of a 63-year-old woman who presented with macroreentrant atrial tachycardia (MAT), related to the atrial trajectory of an old single-lead ventricular PM, that was successfully treated with radiofrequency ablation after a conventional electrophysiological study ruled out isthmus-dependent atrial flutter and provided sufficient data to confirm this diagnosis. This report presents a case of MAT originating around the trajectory of a PM lead, probably because of scar tissue that developed adjacent to the lead. Experimental studies have already shown that interstitial atrial fibrosis may develop adjacent to a ventricular single-lead. This finding suggests that MAT develops in patients with this specific condition. Recognizing this condition is important for managing these arrhythmias and performing safe ablation with the preservation of PM lead integrity.
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1. Introduction

Macroreentrant atrial circuits are frequently associated with scar tissues because they provide two basic conditions for reentry: slow conduction and unidirectional block [1—3]. Previous reports have shown that scar tissue may develop adjacent to pacemaker (PM) leads [4]. However, reports on the occurrence of PM lead-related reentrant tachycardia are scarce.

We report the case of a 63-year-old woman who presented with macroreentrant atrial tachycardia (MAT) related to the atrial trajectory of an old single-lead ventricular PM that was successfully treated using radiofrequency ablation.

2. Case report

A 63-year-old woman was admitted to our hospital because of progressive dyspnea and deterioration of the New York Heart Association functional class. She had a PM implanted 24 years earlier because of a high-grade atrioventricular block. Echocardiography revealed global ventricular dysfunction, with an ejection fraction of

42%. An electrocardiogram (ECG) illustrated the MAT pattern in the atria, with a ventricular paced rhythm of 70 beats per minute (bpm) in the ventricles (Fig. 1). Particularly for F-wave morphology, the ECG illustrated positive F-waves in the inferior leads and iso-diphasic/positive F-waves in lead V1, thereby suggesting an atypical flutter pattern (Fig. 1).

The implanted PM was a single-lead ventricular device (Verity ADx XL VDR, St. Jude Medical, Saint Paul, MN, USA) programmed in the VDD mode, with a basal heart rate of 70 bpm. The stored ECG recordings indicated that the commencement of atrial tachycardia corresponded to the worsening of patient symptoms. Hence, electrophysiological study (EPS) and flutter ablation were indicated for restoring atrioventricular synchrony.

The observed tachycardia cycle length (TCL) was 250 ms during electrophysiological study (EPS). Tachycardia was easily entrained; therefore, we forwent automaticity and triggered activity as possible tachycardia mechanisms. The details of entrainment are described below. Atrial activity accounted for 70% of the TCL (176 ms), implying macroreentry as the mechanism for tachycardia (Fig. 2A).

The atrial activation sequence on the duodecapolar catheter exhibited counterclockwise activation from dipoles 11—12 to 1—2 in the lateral aspect of the tricuspid annulus, exhibiting a break-through from dipoles 11—12 to 13—14. Activation of this last pair of electrodes occurred at the same time as dipoles 9—10 suggesting

Fig. 1. Baseline electrocardiogram showing a macroreentrant atrial tachycardia pattern for the atria, with ventricular pace rhythm for the ventricles. Positive F-wave polarity in the inferior leads and predominant positive polarity in the V1 yields an atypical atrial flutter pattern.

clockwise activation departing from dipoles 11–12 as well. This type of activation refutes the diagnosis of typical CTI-dependent atrial flutter (Fig. 2A).

Low-voltage late diastolic potentials were also observed in the posteroseptal region of the right atrium (RA), confirmed not only after mapping but also after several relocations of the duodecapolar catheter. There were low voltage potentials around the trajectory of the PM lead and adjacent to the septal aspect of the tricuspid annulus, with mid-diastolic potentials occurring in the septal dipoles of the duodecapolar catheter (dipoles 15–16, 17–18, and 19–20) (Fig. 2A).

During mapping, tachycardia spontaneously terminated, and bidirectional CTI block was recorded through differential atrial pacing. Tachycardia was further re-induced and entrained.

Regarding detailed aspects of entrainment, the first prominent feature was the RA origin, since the post-pacing interval (PPI) from the left atrial dipoles of the coronary sinus decapolar catheter was greater than 100 ms, with important changes in the atrial activation sequence. Entrainment of the septal aspect of the CTI was not feasible because of the low-voltage potentials in this region. Subsequent entrainment of the lateral aspect of the duodecapolar catheter (lateral CTI) indicated a PPI of 20 ms with manifest fusion, indicating that the ablation catheter was inside the circuit but not inside the protected isthmus (Fig. 2B). Entrainment from the successful ablation site showed concealed fusion and PPI of 20 ms (Fig. 2C). The radioscopic views are shown in Fig. 3 A and B. A diagram of the probable circuit is shown in Fig. 3C.

Afterwards, careful mapping was performed, and moving the ablation catheter clockwise toward the posterior RA revealed fragmented atrial signal potentials around the PM lead with double potentials along the PM lead trajectory separated by 40 ms (Fig. 2D). To confirm the critical isthmus presence and location, and to improve mapping, we moved the duodecapolar catheter near the ablation catheter, utilizing essentially the distal dipoles to guide careful RF ablation (Fig. 2D, E, and 2F).

Thus, in the beginning of radiofrequency ablation at this site, the double potentials started splitting, and subsequently, tachycardia was terminated (Fig. 2E and F). It is worth noticing that the double potential interval increased to 114 ms with tachycardia ending (Fig. 2F). A single RF application was sufficient to successfully terminate the tachycardia, which demonstrated that the ablation
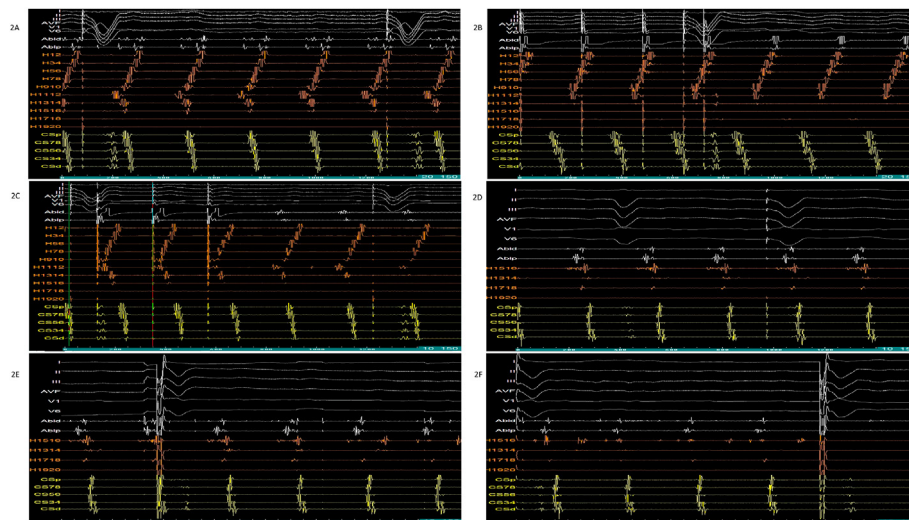
Fig. 2. Electrophysiological Study Recordings
Fig. 2A. Atrial activation sequence with breakthrough at dipoles 11–12 of the duodecapolar catheter.
Fig. 2B. Entrainment from the lateral aspect of CTI shows manifest fusion.
Fig. 2C. Entrainment from the successful ablation site shows concealed fusion. Notice the robust fragmented signal recorded by the ablation catheter on this site.
Fig. 2D. Beginning of RF ablation. Fragmented potentials were recorded by the distal duodecapolar catheter adjacent to the ablation catheter. Notice the early diastolic potential at the distal ablation catheter.
Fig. 2E. Continuation of RF ablation with the initial splitting of the fragmented potentials was recorded by both duodecapolar catheter and ablation catheter.
Fig. 2F. Termination of the tachycardia during RF ablation.
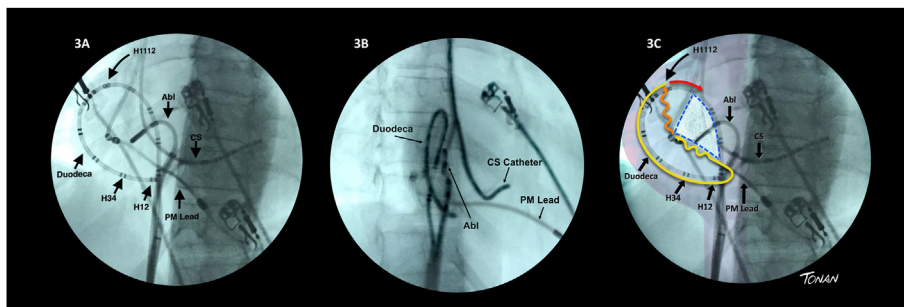
Fig. 3. Fluoroscopic Views and Schematic Drawing of the Tachycardia Circuit
Fig. 3A. Left anterior oblique (LAO) view showing the catheter position during the electrophysiological study.
Fig. 3B. The right anterior oblique (RAO) view shows the ablation catheter's posterior orientation at the successful ablation site.
Fig. 3C. LAO fluoroscopic view showing schematic atrial activation sequence during MAT mapping and ablation. The ablation catheter is positioned along the PM lead trajectory in the posterolateral region of the right atrium. The yellow line shows the atypical flutter circuit, descending rapidly to the lateral aspect of the tricuspid annulus, encountering a fibrotic area (dashed line in blue). The slow conduction zone adjacent to the PM leads returns to the duodecapolar catheter's upper region (dipole 11—12) after running adjacent to the PM lead and fibrotic area (undulating lines).
Abl — Ablation Catheter
CS — Decapolar Catheter positioned in the coronary sinusDuodecapolar
— Duodecapolar catheter positioned around the tricuspid annulus
H12 and H34 — Distal dipoles of duodecapolar catheter
H1112 — Dipoles 11—12 of duodecapolar catheter
PM lead — Pacemaker lead electrode.

catheter was probably inside the protected isthmus of the circuit, possibly showing a narrow circuit surrounded by scar tissue. During RF ablation, the power was reduced to 40 W, aiming at a temperature of 50 °C to preserve lead integrity. Impedance, sensing, and capture thresholds were maintained in the post-ablation evaluation compared with the basal parameters.

After MAT was successfully terminated by RF ablation, significant low voltage signals from the PM lead trajectory toward the septal aspect of the tricuspid annulus were recorded also during sinus rhythm by the same duodecapolar catheter dipoles mentioned above.

Atrial flutter or tachycardia was not induced by any atrial drive train after RF ablation. At the end of the procedure, transesophageal echocardiography was performed to confirm that the PM lead trajectory was within the posterolateral region of the RA.

Postoperatively, the patient showed an improvement in functional status and resolution of dyspnea at rest. A six-month follow-up revealed sinus rhythm and maintenance of dyspnea resolution.

3. Discussion

This report presents the case of a patient with MAT originating around the trajectory of a PM lead, probably due to scar tissue that developed adjacent to this old lead. Entrainment and mapping of the tachycardia suggested a macroreentrant mechanism and its relationship with scar tissue adjacent to the pacemaker lead.

Although previous reports have described stable parameters, such as atrial sensing and the absence of complications in moderate-to-long-term follow-ups for VDD pacemakers [4], data from longer periods are lacking.

Moreover, experimental studies have shown that interstitial fibrosis may develop around a single PM ventricular lead. This finding suggests that MAT may arise in patients with PMs, and especially old leads provide the substrate conditions necessary for its development [5]. In addition, tricuspid valve adhesion of old PM leads has been described in a post-mortem analysis [6].

Electroanatomical mapping systems are unavailable for each EPS in the public health system. However, conventional activation mapping provides important information, and diagnosis as well as ablation is perfectly feasible in this scenario. The conventional EPS

provided sufficient information to rule out typical atrial flutter and aided in diagnosing this atypical new PM lead-related atrial flutter.

Several features observed by the conventional EPS led us to rationally hypothesize that this atypical atrial macroreentry was related to the PM lead: (a) atypical activation sequence with significant breakthroughs in the atrial activation sequence; (b) entrainment performed from the lateral aspect of the CTI with PPI of 20 ms but showing manifest fusion; (c) concealed fusion and PPI of 20 ms shown by entrainment at the successful ablation site; (d) confirmation of CTI block when the tachycardia spontaneously terminated; (e) presence of possible scarring area from the PM lead to the septal aspect of the tricuspid annulus as noticed by the presence of low voltage potentials in that area; (f) distinctly double potentials observed through the PM lead trajectory, and (g) ablation in this region not only interrupted the tachycardia but also separated these double potentials along the line of ablation by more than 70 ms. Consequently, atrial scarring could have developed in this region because of a 24-year-old PM lead, triggering proper electrophysiological conditions for the development of PM-related MAT.

Atypical atrial flutter has been described in patients with normal structural hearts and no prior cardiac surgery,[11–13] and anisotropic conduction through the crista terminalis could be the cause behind this atypical flutter [3,7,8]. However, the presence of breakthroughs in the atrial activation sequence in the duodecapolar catheter, characteristics of entrainment, presence of the previous block in CTI, and pattern of tachycardia termination with a single shot in the protected isthmus of the circuit with double potential splitting at the time of tachycardia end are not characteristics that these authors have previously described regarding the most common atypical right atrial flutter patterns. These patterns include lower and upper loop reentry, low lateral right atrial reentry, and partially dependent CTI short circuits [3,7,8].

In addition to that, Stevenson et al. described that MAT could be demonstrated to rotate around a free wall scar in patients with normal heart structure, describing that atrial scarring was extended from the posterolateral region of the RA (crista terminalis) to the RA free wall [8]. Our study instead described a scarring region from the posterolateral RA toward the septal aspect of the tricuspid annulus, adjacent to the trajectory of the old PM lead, ratifying our

hypothesis of a MAT developed around the trajectory of a PM lead. Therefore, although the presence of a CTI block without previous ablation may indicate that the patient is prone to spontaneous fibrosis and could have developed scarring in the posterior RA, the characteristics mentioned above (specifically related to the septal aspect of fibrotic tissue, atypical atrial activation sequence, ablation catheter orientation, and characteristics of ablation) allowed us to reason that the MAT was related to the PM lead.

Moreover, to exclude double-loop right atrial reentry and focal atrial tachycardia from single-loop atrial macroreentry, we also used the criteria reported from Linton and Haissaguerre et al. [9], comparing the first beat of the tachycardia recorded by two stationary electrodes after entrainment performed by each of them. The result was equal to TCL, thereby ratifying single-loop reentry as a tachycardia mechanism and excluding focal AT and double-loop reentry [9].

Nonetheless, to the best of our knowledge, there have been no reports on MAT and PM leads. The only report describing this type of lead-related complication was published by Abraham et al., who documented ventricular tachycardia related to an implantable cardioverter-defibrillator lead, associated with scar tissue [10]. However, patients with implanted PM for over 20 years are frequently excluded from clinical and observational trials, and these complications might not be properly documented. Although anecdotal, complications during long-term follow-up in these patients must be examined.

4. Conclusion

Macroreentrant arrhythmias may be related to the scar tissue adjacent to device leads. Recognizing this condition is critical for its effective management and safe ablation, with no damage to the PM lead integrity.

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Author contributions

All authors contributed to the case report as well as literature review.

Declaration of competing interest

The authors have no conflicts of interest to disclose.

Acknowledgments

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