

Case Report

Incessant focal right atrial tachycardia causing tachycardia-induced cardiomyopathy reversed by high-density mapping-guided catheter ablation

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Introduction

Atrial tachycardia is an uncommon supraventricular arrhythmia [1] but an important and reversible cause of Tachycardia-Induced Cardiomyopathy (TIC). Persistent tachycardia may lead to left ventricular dysfunction through sustained tachycardia burden, impaired ventricular filling, and adverse remodeling. Importantly, left ventricular dysfunction is often reversible after restoration of sinus rhythm.

We report a case of incessant focal right atrial tachycardia causing severe cardiomyopathy, successfully treated with high-density mapping-guided catheter ablation.

Case presentation

A 52-year-old woman with no prior cardiac history presented with progressive dyspnea and decompensated heart failure. She was tachycardic (120-130 bpm), hypoxemic (SpO₂ 90%), and clinically congested with elevated jugular venous pressure and pulmonary crackles.

Transthoracic echocardiography demonstrated a dilated left ventricle with severely reduced systolic function (LVEF ~25%). Twelve-lead ECG showed a regular narrow-complex long RP tachycardia at 120-130 bpm, with differential diagnosis included sinus tachycardia, focal atrial tachycardia, atypical atrioventricular nodal re-entrant tachycardia, and permanent junctional reciprocating tachycardia [2]. P-wave morphology suggested a high right atrial origin (positive in inferior leads and lead I, negative in AVR) [3].

Guideline-directed medical therapy for heart failure with reduced ejection fraction was initiated. Coronary angiography revealed normal coronary arteries. Despite transient rate control with beta-blockers, recurrent atrial tachycardia persisted, and tachycardia-induced cardiomyopathy secondary to focal AT was suspected.

Electrophysiological study: An electrophysiological study induced a long RP narrow-complex tachycardia with cycle length 450-470 ms and concentric atrial activation.

Abstract

Atrial Tachycardia (AT) is an uncommon [1] but reversible cause of Tachycardia-Induced Cardiomyopathy (TIC). We report a 52-year-old woman presenting with decompensated heart failure and severe Left Ventricular Dysfunction (LVEF ~25%) due to incessant focal right atrial tachycardia. ECG showed a regular narrow-complex long RP tachycardia at 120-130 bpm. Coronary angiography excluded ischemic heart disease. Electrophysiological study confirmed focal AT with ventriculo-atrial dissociation during ventricular overdrive pacing. High-density electro-anatomical mapping identified earliest activation in the posterior superior and superior anterolateral right atrium. Radiofrequency ablation was performed with preservation of sinus node and phrenic nerve function. AT was rendered non-inducible, and sinus rhythm was maintained. At 3 months, the patient demonstrated complete clinical and echocardiographic recovery (LVEF 55%), consistent with reversible TIC.

Keywords: Atrial tachycardia; Tachycardia-induced cardiomyopathy; Catheter ablation; Electrophysiological study; Electro-anatomical mapping.

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Ventricular overdrive pacing demonstrated ventriculo-atrial dissociation, confirming focal atrial tachycardia and excluding atrioventricular re-entrant tachycardia and atypical AVNRT.

Mapping and ablation

High-density electro anatomical mapping (3D mapping system with multipolar catheter) was performed during tachycardia. Earliest atrial activation was identified in two adjacent right atrial regions:

- Posterior superior right atrium (cycle length 450-470 ms)
- Superior anterolateral right atrium (cycle length 500-510 ms)

These findings were consistent with either dual focal atrial tachycardias or a single focus with variable exit pathways.

Given proximity to the sinus node and phrenic nerve, high-output pacing was used to define safe ablation boundaries. Radiofrequency energy (35 W, irrigated contact-force catheter) was delivered, resulting in transient acceleration followed by termination of tachycardia.

Post-ablation programmed stimulation and burst pacing failed to induce atrial tachycardia. Sinus rhythm was maintained, and phrenic nerve capture remained intact throughout.

Outcome: At 3-month follow-up, the patient was asymptomatic (NYHA class I) with no recurrence of atrial tachycardia.

Repeat echocardiography demonstrated complete recovery of left ventricular systolic function (LVEF 55%) with normalization of chamber dimensions, confirming tachycardia-induced cardiomyopathy.

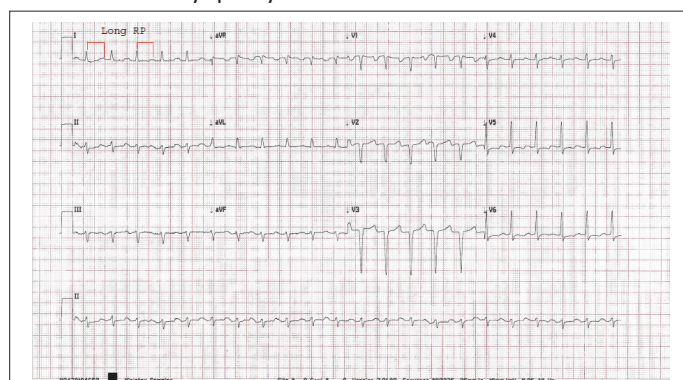


Figure 1: Twelve-lead ECG demonstrating a regular narrow-complex long RP tachycardia (120-130 bpm) with P-wave morphology consistent with high right atrial origin.

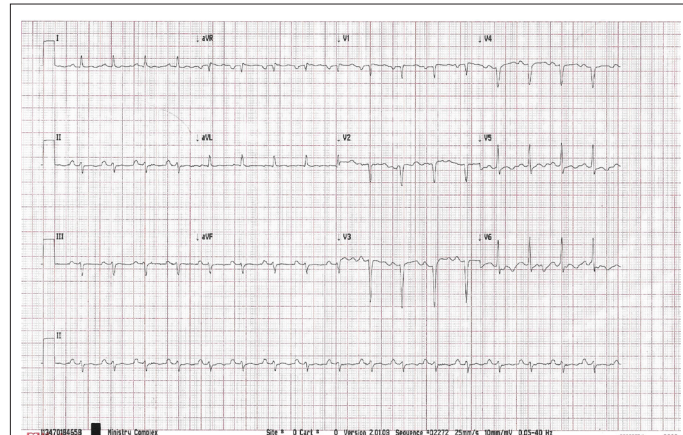


Figure 2: Sinus rhythm ECG following treatment showing resolution of tachycardia and normal atrioventricular conduction.

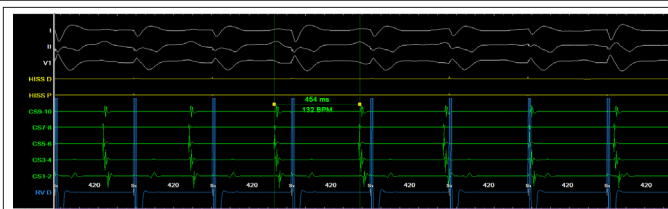


Figure 3: Ventricular overdrive pacing demonstrating ventriculo-atrial dissociation, confirming focal atrial tachycardia.

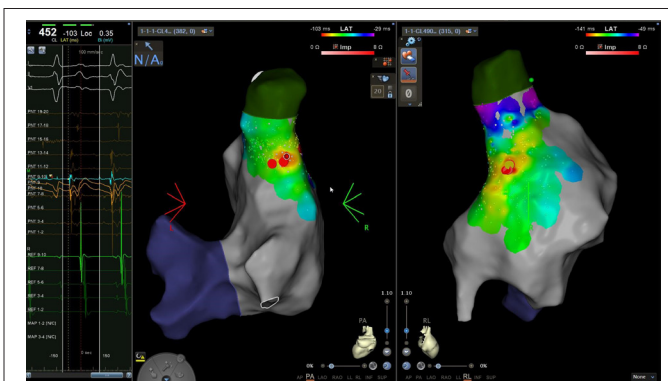


Figure 4: High-density electro anatomical mapping identifying earliest activation in the posterior superior and superior anterolateral right atrium.

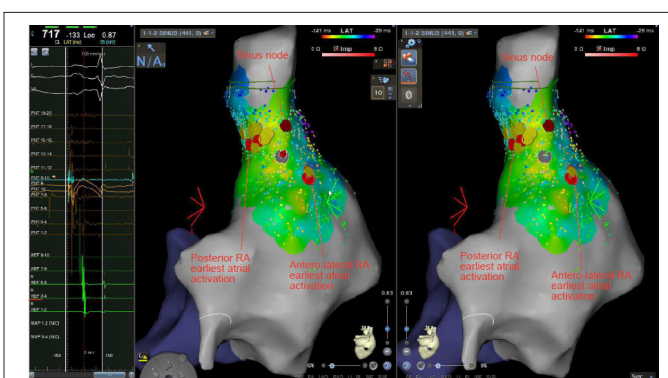


Figure 5: Spatial relationship of earliest activation sites to the sinus node and phrenic nerve with ablation strategy.

Discussion

This case highlights a reversible cause of severe cardiomyopathy due to incessant focal atrial tachycardia. Tachycardia-induced cardiomyopathy results from sustained elevated heart rate leading to myocardial energy depletion, calcium handling abnormalities, neurohormonal activation, and adverse remodeling [4]. Importantly, ventricular dysfunction is frequently reversible following arrhythmia elimination.

The diagnosis of focal AT was supported by long RP tachycardia with characteristic P-wave morphology and confirmed by ventriculo-atrial dissociation during ventricular overdrive pacing, excluding re-entrant supraventricular tachycardias.

High-density electro anatomical mapping was essential for precise localization of closely spaced right atrial foci. This likely represents either dual focal tachycardias or a single focus with variable conduction pathways.

Catheter ablation is first-line therapy for focal atrial tachycardia and is particularly important in tachycardia-induced cardiomyopathy, where elimination of the arrhythmia can result in complete reversal of ventricular dysfunction [5]. Mapping near the sinus node and phrenic nerve is critical to ensure procedural safety.

Timeline

Time	Clinical event
Day 0	Presentation with decompensated heart failure and tachycardia (120-130 bpm).
Day 1	Echocardiography: LVEF 25%.
Day 1	ECG: Long RP narrow-complex tachycardia.
Day 1-3	Initiation of GDMT and stabilization.
During hospitalization	Sinus rhythm intermittently restored with beta-blocker therapy; however, recurrent atrial tachycardia persisted.
During hospitalization	Coronary angiography normal.
Following diagnostic evaluation	Incessant atrial tachycardia-induced cardiomyopathy suspected.
EP study	Focal AT confirmed with VA dissociation.
Mapping	High-density mapping localized earliest activation to the posterior superior and superior anterolateral right atrium.
Ablation	Successful RF ablation with sinus rhythm restoration.
3 months post-ablation	LVEF recovered to 55%, no recurrence of AT.

Key learning points

- Incessant focal atrial tachycardia is a reversible cause of dilated cardiomyopathy.
- Ventriculi-atrial dissociation during ventricular overdrive pacing confirms focal AT.
- High-density electro anatomical mapping is essential in complex right atrial tachycardia.
- Tachycardia-induced cardiomyopathy is often fully reversible after successful ablation.
- Ablation near the sinus node and phrenic nerve requires careful functional mapping.

Conclusion

Incessant focal right atrial tachycardia should be considered in patients presenting with unexplained cardiomyopathy and persistent narrow-complex tachycardia. Early electrophysiological evaluation with high-density mapping enables accurate diagnosis and effective ablation, resulting in complete recovery of ventricular function in tachycardia-induced cardiomyopathy.

Declarations

Patient consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images in accordance with journal requirements.

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Conflict of interest: The authors declare no conflicts of interest related to this manuscript.

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