

Paroxysmal stress-induced atrial tachycardia as an unusual cause of presyncope associated with paroxysmal fatigue and chest pain

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ABSTRACT

A 70 years old man without medical history was referred for pre-syncope associated with paroxysmal chest pain and fatigue after emotional stress and exercise. Symptoms debuted in the last 3 months and were refractory to medication. His rest 12-lead ECG showed only a 1st degree AV block and a borderline QTc of 440 ms. Ecocardiographic evaluation was normal. During exercise ECG testing a relatively rapid atrial tachycardia (cycle length 350 ms) with Wenckebach phenomenon was induced. During this tachycardia patient was almost asymptomatic and there were no ST-segment anomalies, but there was a slight prolongation of QTc to 455 ms. However, in the recovery phase there was a sudden termination of the tachycardia with a 1.4 seconds pause of sinus node activity during which 2 ventricular and 2 atrial escape beats emerged and symptoms were reproduced. During electrophysiologic study both burst pacing and isoproterenol but not programmed stimulation easily induced the tachycardia suggesting a triggered type mechanism. Mapping identified the area with most precocious electrical activity in the superior part of crista terminalis. After 2 radiofrequency applications in this location patient was non-inducible. At 6 months follow-up patient was asymptomatic and with no arrhythmia on Holter monitoring and exercise ECG testing.

Key words: syncope, atrial tachycardia, long QT trait, radiofrequency catheter ablation

INTRODUCTION

Repetitive focal atrial tachycardia (AT) accounts for 5 to 15 percent of arrhythmias in adults undergoing study for paroxysmal supraventricular tachycardia (1). Focal ATs are usually paroxysmal (2-4) and symptoms are lim-

ited to palpitations, but in the presence of structural heart disease symptoms can be more severe. Even in patients with normal hearts persistent AT can produce heart failure (due to various degree of tachycardiomyopathy) (2-4) and it can induce sinus node dysfunction (due to electrical remodeling). Both tachycardiomyopathy

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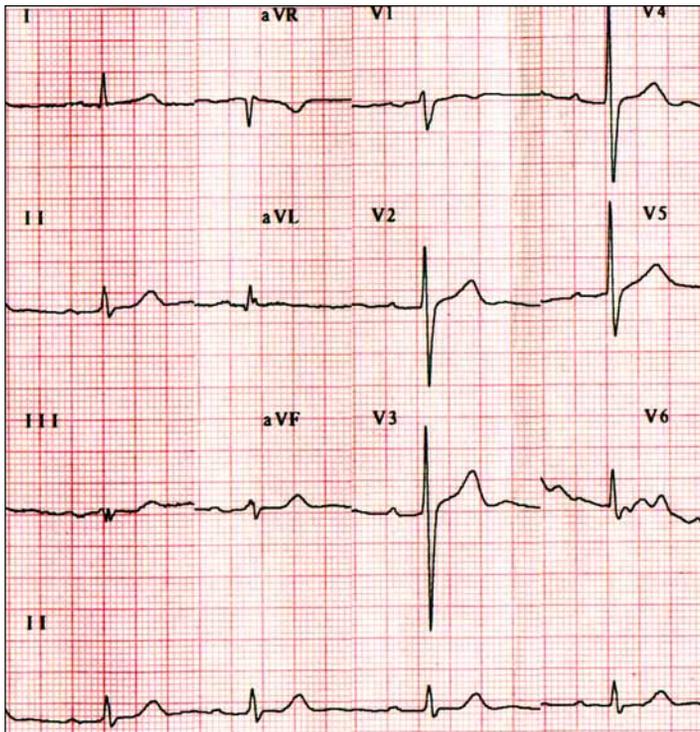


FIGURE 1A. Baseline ECG with first degree AV block

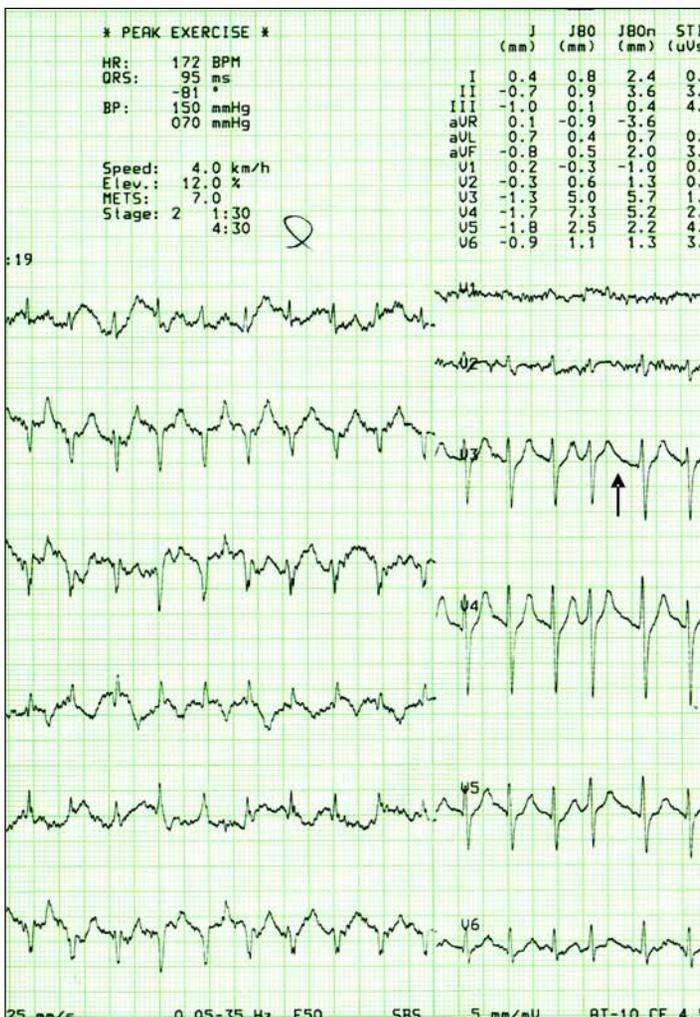


FIGURE 1B. AT with Wenckebach phenomenon (arrow) during exercise ECG test

and sinus node remodeling are potentially reversible after the restoration of sinus rhythm. In patients with structurally normal heart and paroxysmal AT potentially life-threatening symptoms related to paroxysmal secondary bradycardia have never been described. □

CASE REPORT

A 70 years old man without any comorbidities was referred to our hospital because of a 3 months history of pre-syncope, fatigue and chest pain after emotional stress and exercise. Careful anamnesis revealed that chest pain was rather atypical (paroxysmal, short-lived and on a very limited area). Clinical evaluation was normal. His baseline ECG was unremarkable except for a 1st degree AV block (PR interval 0.24 s), a corrected QT (QTc, Framingham formula) interval at the upper limit of normal (QTc=440 ms, FIGURE 1A) and noticeable U waves. Trans-thoracic and trans-esophageal echocardiography showed a structurally normal heart without any disturbances of ventricular wall movement as well as a normal structure of the root, the arch and the descending part of the aorta. Repeated Holter monitoring failed to reveal any arrhythmia or signs of myocardial ischemia. An exercise ECG test was performed during which a rapid supraventricular tachycardia (CL 350 ms) with Wenckebach phenomenon was induced (FIGURE 1B). Despite high ventricular rate during this tachycardia (mean ventricular rate 170 bpm) patient was able to perform a moderate to high level exercise (8MET) and he was almost symptomatic (extremely discrete palpitations) without any ST-segment modifications suggestive of myocardial ischemia. QTc was always at the upper limit of normal (450 ms). During the recovery phase there was a sudden termination of supraventricular tachycardia followed by a 1.4 seconds pause of sinus activity during which 2 ventricular and 2 atrial escape beats appeared (FIGURE 1C). There was exactly this pause when pre-syncope and atypical chest pain were induced but they rapidly disappeared after the activity of sinus node was restored. Moreover, initially sinus rhythm was quite slow (60 bpm,

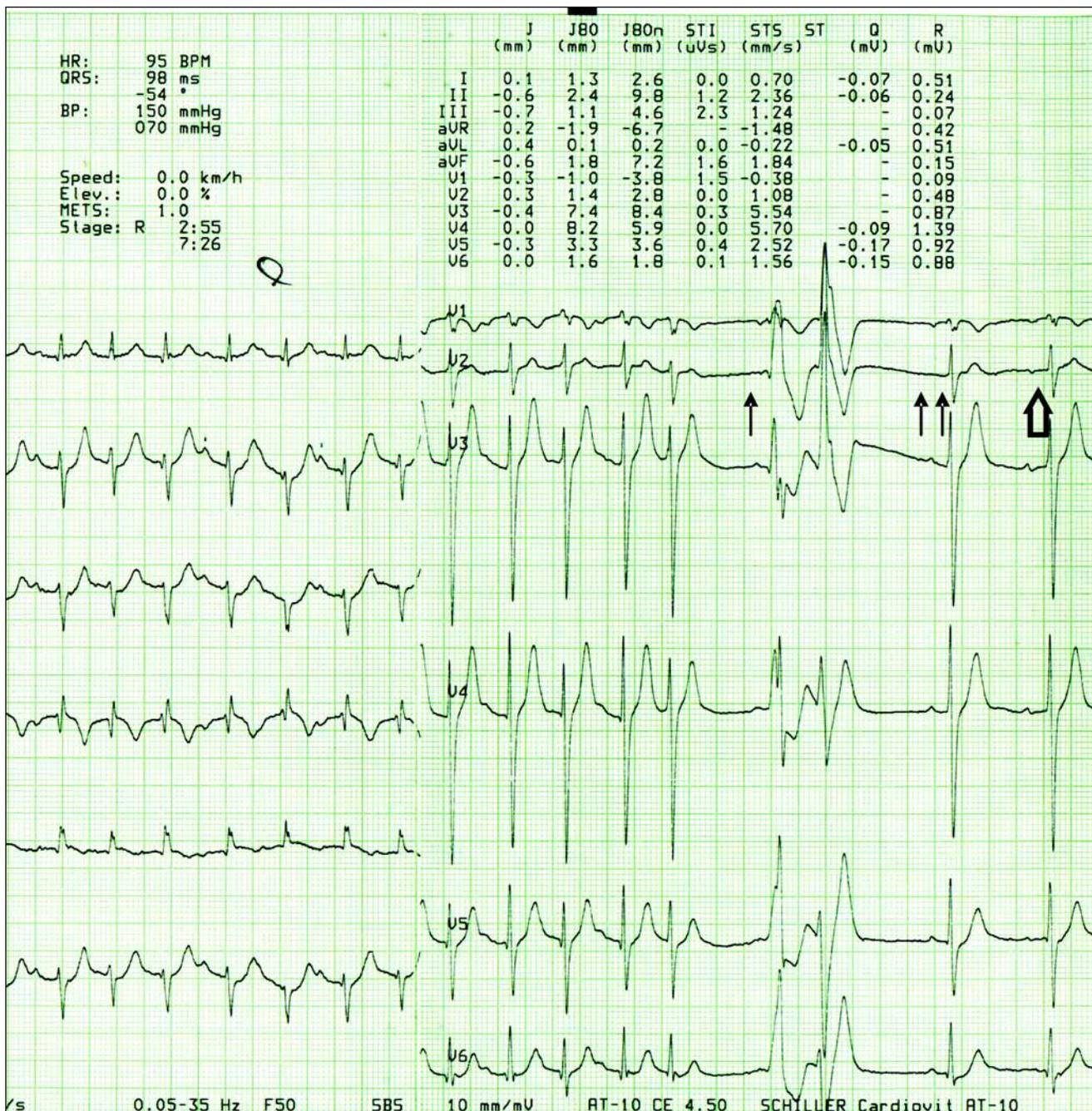


FIGURE 1C. 1400 ms pause till first sinus beat (arrowhead) after sudden termination of the AT during the recovery phase of the exercise ECG testing; first atrial escape beat (arrow) is not conducted and is immediately followed by 2 ventricular escape beats, but the second atrial escape beat (double arrow) is conducted (note different morphology with respect to sinus beats especially in leads V2 to V6).

FIGURE 1C) and it was correlated with fatigue. However, in 20 s sinus rhythm accelerated to 100 bpm (FIGURE 1D) and concomitantly fatigue vanished.

Patient was referred for electrophysiologic study. Under local anesthesia, a decapolar diagnostic coronary sinus catheter and a 4 mm

tip quadripolar mapping/ablation catheter were inserted via right femoral vein. Pacing was performed from proximal pair of the decapolar catheter (at the level of coronary sinus ostium). Burst pacing but not programmed stimulation induced a non-sustained supraventricular tachycardia of 350 ms cycle length (CL). A 4 mm



FIGURE 1D. Sinus activity was accelerating progressively, 20 s latter reaching a peak of 102 bpm (D)

intravenous bolus of isoprenaline induced supraventricular tachycardia and made it sustained. Biatrial activation time (5) was only 105 ms (30% of CL) consistent with a focal AT and activation sequence proved that it originated into the RA. Activation mapping during AT revealed earliest atrial depolarization at the level of superior part of crista terminalis, were a fragmented local electrogram preceded surface P wave by 60 ms (FIGURE 2A). After high output pacing to reveal the possible proximity of the right phrenic nerve (6), an 1 minute RF application was made (55°C, 50W), stopping the tachycardia. However, repeated burst pacing after another 4 mg intravenous bolus of isoprenaline re-induced AT but with a larger CL (375 ms). Re-mapping identified earliest atrial depolarization 1 mm inferior to the previous site, with the fragmented local electrogram preceding surface P wave by 43 ms (FIGURE 2B). Using the same protocol another 1 minute RF application was made, which stopped the tachycardia after a very short initial acceleration (FIGURE 2C). After this RF application patient was no longer inducible. Post-ablation testing showed normal AV conduction (AH=140 ms, HV=50 ms, Wenckebach point 300 ms) and normal sinus node function (SNRTc = 500 ms, SACT = 100 ms). Interestingly QTc during burst pacing at 300 ms CL and after a 4 mg intravenous isoprenaline bolus was 455 ms. At 6 months follow-up patient was completely asymptomatic and with no arrhythmia on repeated Holter monitoring and exercise ECG testing. □

DISCUSSION

Supraventricular arrhythmias in older patients can be challenging. Surface ECG during tachycardia can help to differentiate focal vs macroreentrant arrhythmias (7) and even predict the anatomic site of origin (8), but accurate diagnosis can be impossible during period with high ventricular response when the atrial activity is obscured into the QRS or/and ST-T interval.

Focal AT is a relatively uncommon arrhythmia in adults with the vast majority of patients presenting a non-sustained form (3,4). A large spectrum of symptoms exists, from asymptomatic to complete incapacitation. Symptoms include palpitations, dizziness, chest pain, dyspnea, fatigue, and syncope (4). Paroxysmal AT is



FIGURE 2A. Site of the first RF application at the level of superior crista terminalis, were during AT a fragmented local electrogram preceded by 60 ms the beginning of the surface P wave



FIGURE 2B. The second RF application was 1 mm inferior to the previous, were during AT local electrogram preceded by 43 ms the beginning of the surface P wave

seldom associated with symptoms, especially in patients with structurally normal hearts, but persistent AT can induce tachycardiomyopathy and sinus node dysfunction (9). The severity of symptoms can be difficult to interpret in older patients, in which a higher possibility of coexistent structural or ischemic heart disease exists.

In the present case severity of symptoms (pre-syncope, chest pain and fatigue) after physical and emotional stress in a 70 year old man would suggest a different etiology. However significant coronary artery disease or structural heart disease is highly unlikely considering that at higher heart rates there were no symptoms

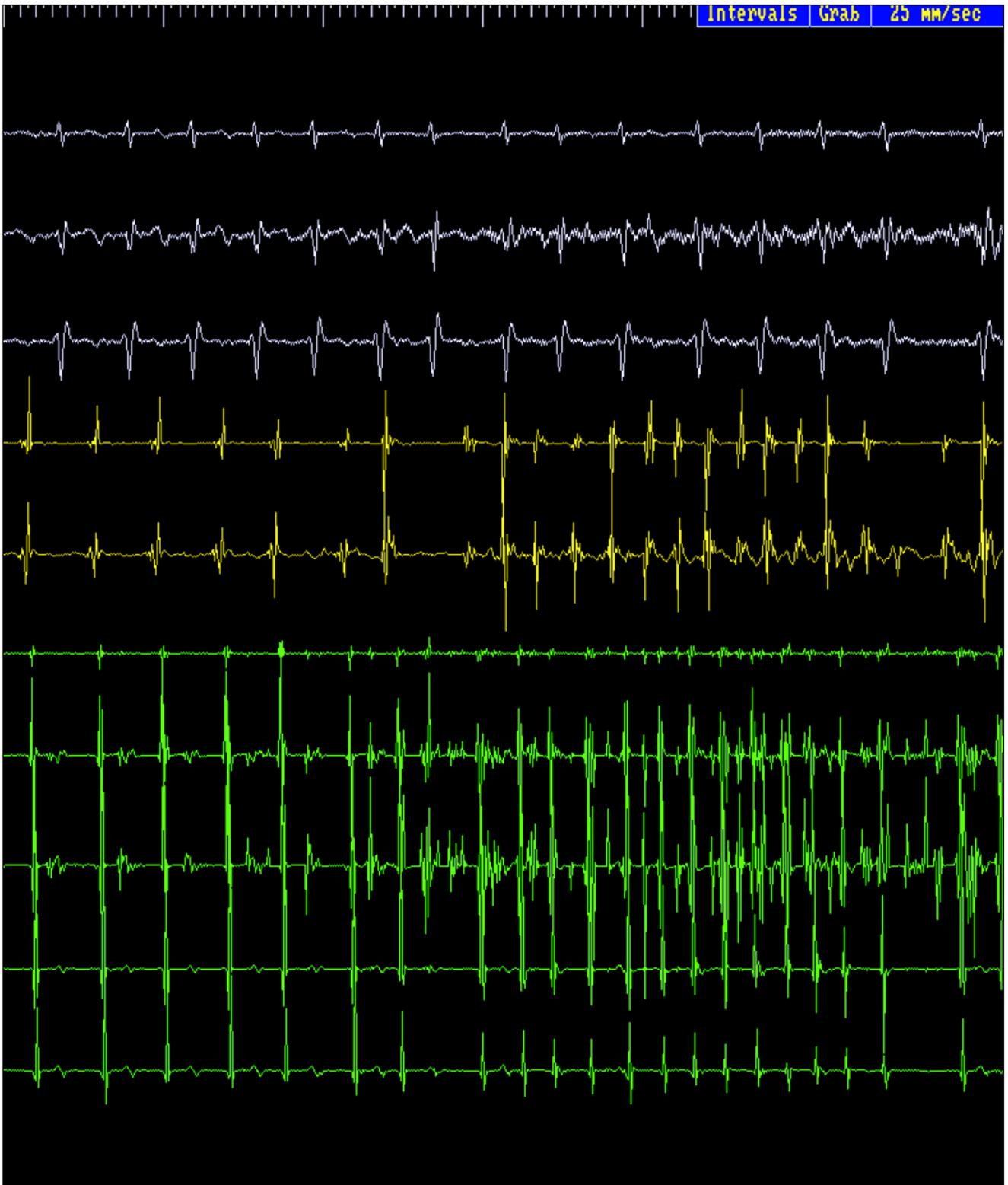


FIGURE 2C. AT termination during RF application was preceded by a short period of acceleration

and no ST-T interval anomalies. Another potential explanation would be that vigorous heart contraction after a sudden large pause especially in a more rigid aorta could trigger vagal reflexes with hypotension and transitory bradycardia. A significant degree of sinus node dysfunction can be reasonably ruled out counting that SNRTc and SACT were normal. Atrio-ventricular dyssynchrony symptoms due to retrograde ventriculo-atrial conduction during PVCs are also unlikely considering that retrograde P waves could not be documented during PVCs. An interesting alternative explanation for the severity of symptoms could be a non-sustained bradycardia-dependent ventricular tachycardia induced by the sudden termination of AT. Although we could not document this, this cannot completely be ruled out especially if we consider the possible presence of a long QT (LQT) trait in this patient (baseline borderline QTc of 440 ms, with slight prolongation at 455 ms during high ventricular rates and isoprenaline

infusion). However we cannot definitively prove the presence of LQT trait because the genetic analysis was not performed. QTc prolongation can be more prominent in conditions known to prolong ventricular repolarization like hypopotasemia or drugs (10).

Catheter ablation of AT has a high efficacy, with a lower than 7% recurrence rate (9), especially for RA tachycardia (11,12). The success rate can be lower in older patients and with multiple foci (11). In this case the success of ablation could be also related to the fact that AT eradication also eliminated the acute bradycardia after sudden termination of AT, abolishing the conditions that can make manifest a LQT trait.

To the best of our knowledge, this is the first reported case of a benign paroxysmal atrial arrhythmia creates a very rare situation with a sum of conditions that can make manifest a LQT trait. □

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