Background

Tachycardia-induced cardiomyopathy (TCM) is characterised by left ventricular dysfunction and heart failure (HF) due to heart rhythm disorder, which are often reversible in the case of successful arrhythmia treatment [1, 2, 3]. It is most often caused by atrial fibrillation (AF) or atrial flutter, sustained supraventricular or ventricular tachycardias, as well as frequent ectopic beats [4, 5, 6]. However, establishing the differential diagnosis for the cause of the TCM can sometimes be difficult. This may happen in the case of a very rare supraventricular arrhythmia such as dual atrioventricular nodal non-reentrant tachycardia (DAVNNT).

The longitudinal dissociation of the atrioventricular (AV) node to slow and fast pathways is usually manifested by AV nodal reentrant tachycardia (AVNRT), which is one of the most frequent supraventricular tachycardias [7, 8]. Dual conduction of sinus impulses to the ventricles via both pathways, due to large differences in their functional properties, is a special feature of DAVNNT. One P wave may be followed by 2 QRS complexes [2, 3, 4, 6]. Part of the sinus impulses may be blocked in the fast or slow AV nodal pathways, with consequent absence of the double conduction which leads to irregular appearance of the tachycardia. Additionally, aberrance of the intraventricular conduction may occur, further complicating the diagnosis. Thus, DAVNNT can mimic a variety of supraventricular and ventricular arrhythmias. Moreover, it is usually resistant to most antiarrhythmic drugs, making radiofrequency (RF) catheter ablation of the slow AV nodal pathway the only effective treatment method [1–6].

Case presentation

A 39-year-old female was admitted to the hospital and presented with symptomatic congestive HF and reported dyspnoea and fatigue. Irregular, narrow QRS complex tachycardia was continuously observed on serial electrocardiograms (ECGs) (Fig. 1, Panel A). The patient had many previous ECGs which showed short and sustained episodes of the tachycardia (Fig. 1, Panel B). There was
no data suggestive of myocarditis or primary cardiomyopathy according to the patient's history or echocardiographic findings.

That patient had a history of paroxysmal supraventricular tachycardia which began at the age of 20. She was treated with nearly all guideline-recommended antiarrhythmic drugs (beta-blockers, flecainide, propafenone, amiodarone), mostly short-term, and without any positive result. Since the narrow QRS complex tachycardia was irregular, we incorrectly considered this arrhythmia to be paroxysmal AF and the patient was referred for RF ablation. Three consecutive attempts at RF catheter pulmonary vein isolation were performed in 2 different clinics since the patient returned to our department. Unfortunately, there was no evident improvement of the tachycardia. The tachycardia gradually transformed into a continuous, drug-resistant, narrow QRS complex tachycardia. The left ventricular ejection fraction (LVEF) decreased to 25%, with signs of worsening congestive HF. Holter ECG monitoring showed recurrent paroxysms of fast, irregular, narrow complex tachycardia (Fig. 2, Panel A) with occasional wide QRS complexes. Wide QRS complexes appeared due to the aberrant ventricular conduction, often after a long previous RR-interval (Ashman phenomenon) (Fig. 2, Panel B). Short episodes of sinus rhythm appeared during sleep (Fig. 2, Panel C). Average heart rate during 24-hour ECG monitoring was 133 bpm, with a maximum heart rate of 179 bpm.

**Patient management and follow-up**

Careful analysis of the 12-lead surface ECG showed that during irregular tachycardia, every P wave was followed by 2 narrow QRS complexes, due to conduction of the same impulse via fast and slow pathways in the AV node. Conduction of the 1st and 2nd P waves via the slow pathway is blocked. The tachycardia gradually transformed into a continuous, drug-resistant, narrow QRS complex tachycardia. The left ventricular ejection fraction (LVEF) decreased to 25%, with signs of worsening congestive HF. Holter ECG monitoring showed recurrent paroxysms of fast, irregular, narrow complex tachycardia (Fig. 2, Panel A) with occasional wide QRS complexes. Wide QRS complexes appeared due to the aberrant ventricular conduction, often after a long previous RR-interval (Ashman phenomenon) (Fig. 2, Panel B). Short episodes of sinus rhythm appeared during sleep (Fig. 2, Panel C). Average heart rate during 24-hour ECG monitoring was 133 bpm, with a maximum heart rate of 179 bpm.

**Figure 1.** 12-lead ECG obtained during hospitalisation. 1 mV = 10 mm, v = 50 mm/sec (Panel A). Fragment of a previous ECG showed irregular tachycardia, which was considered as paroxysmal atrial fibrillation. 1 mV = 10 mm, v = 25 mm/sec (Panel B)

**Figure 2.** Fragments of the Holter ECG recording: narrow QRS irregular tachycardia; middle part of the ECG shows regularly irregular QRS rhythm and P waves, both of which do not meet the criteria for AF diagnosis, which should raise a suspicion of DAVNT (Panel A); episodes of aberrant ventricular conduction (Panel B); sinus rhythm episodes during sleep (Panel C)

**Figure 3.** ECG and laddergram. Every P wave, starting from the 3rd until the 8th, is followed by 2 narrow QRS complexes, due to conduction of the same impulse via fast and slow pathways in the AV node. Conduction of the 1st and 2nd P waves via the slow pathway is blocked.
Tachycardia-induced cardiomyopathy due to dual atrioventricular nodal non-reentrant tachycardia

The main finding was registered through the use of a mapping catheter: every single atrial complex was followed by 2 His and 2 related ventricular complexes (Fig. 5). The diagnosis of DAVNNT was established.

DAVNNT was successfully treated with RF ablation of the slow pathway, with regular sinus rhythm and stable 1:1 AV conduction being restored. Antiarrhythmic treatment was no longer necessary. At the 6-month follow-up visit, the patient did not have signs of HF, LVEF increased to 60%, and no arrhythmias were registered during 48-hour Holter ECG monitoring. Rapid improvement of symptoms and normalisation of LVEF confirmed that the tachycardia was the cause of HF in this patient, thus fulfilling the diagnostic criteria of TCM.

Discussion

DAVNNT, also known as double fire tachycardia, has the same electrophysiologic basis as the more common and well recognized AVNRT. Due to the existence of 2 discrete pathways in the AV node with a large difference in conduction velocity, 1 atrial impulse may result in 2 separate contractions of the ventricles. The most characteristic ECG finding in DAVNNT is a single sinus P-wave followed by 2 QRS complexes. This variant of AV nodal conduction was first reported by Wu et al. in 1975 [9] and there have been several case reports published during the last few years [2, 5, 6, 10]. Due to its very rare occurrence and different ECG patterns with an irregular rhythm, DAVNNT is frequently misdiagnosed as AF [2, 3, 4].

At least 68 cases of DAVNNT were reported in the literature in 2016 and 16 (23.5%) of them were incorrectly diagnosed as AF [2]. Furthermore, pulmonary vein isolation was performed in some of the described patients with DAVNNT, such as in our case. Other arrhythmias which mimic DAVNNT include atrial tachycardia, supraventricular premature beats and atrial bigeminy, atypical AVNRT with retrograde 2:1 block, and junctional extrasystoles and parasystoles [2–6]. Additionally, DAVNNT may be misinterpreted as ventricular extrasystoles or ventricular tachycardia due to aberrant conduction [2, 4, 6].

In recent ESC guidelines on SVT double fire tachycardia is mentioned as an uncommon mechanism of AV nodal tachycardia which may cause TCM and can be successfully treated by slow pathway ablation. DAVNNT is mentioned in ESC guidelines as “an extremely rare tachycardia” and was not included in the algorithm for the differential diagnosis of narrow QRS tachycardia [11].

A reduced LVEF due to TCM has been reported in the majority of patients with DAVNNT who had a long history of palpitations and resistance to antiarrhythmic drugs [1,2,4–6]. In our case, the reduced LVEF returned to normal (from 25% to 60%) after successful slow pathway ablation, which has been widely described as the only effective method for the treatment of double fire tachycardia [2–5, 7, 10].

Using the most recent classification of rare cardiac arrhythmogenic and conduction disorders and rare arrhythmias [12], DAVNNT should be placed into Class VI – cardiac arrhythmogenic disorders and arrhythmias, Group 1 – Primary electrical disorders of the heart, subgroup A – disorders predisposing to supraventricular tachyarrhythmias, number 9 – double fire tachycardia.

Conclusions

Sustained SVT can cause significant worsening of systolic heart function and lead to TCM. A thorough understanding of the arrhythmia’s electrophysiological nature is extremely important for proper treatment. DAVNNT is a rare supraventricular arrhythmia that is often misdiagnosed and can lead to serious consequences for the patient, including unnecessary treatment with anticoagulants or ablation and may be ineffectively treated with antiarrhythmics. RF ablation or cryoablation of the slow pathway in the AV node is the most effective treatment for DAVNNT.
References


Figure 5. DAVNNT during the electrophysiological study. Two QRS complexes follow 1 sinus beat. Intracardiac recording shows 2 His (H) and 2 ventricular (V) signals after 1 atrial impulse (A). Prolongation of the H2V2 interval is explained by aberrant conduction in the ventricles. Abl – mapping catheter, CS – coronary sinus