Clinical Case Report

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Tachycardia-induced cardiomyopathy in a patient with left-sided accessory pathway and left bundle branch block

A case report

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Abstract

Rationale: Tachycardia-induced cardiomyopathy (TIC) is defined as systolic and/or diastolic dysfunction of the left ventricle resulting from prolonged elevated heart rates, completely reversible upon control of the arrhythmia. Atrioventricular reentrant tachycardia (AVRT) is one of the most frequent causes of TIC. In its incessant form, it is unlikely to be controlled by pharmacological treatment, catheter ablation being the principal therapeutic option. The coexistence of left bundle branch block (LBBB) in patients with AVRT may cause difficulties in the early diagnosis and management of tachycardia because of the wide complex morphology, making it harder to localize the accessory pathway (AP).

Patient concerns: A 60-year-old woman, presented incessant episodes of palpitations and shortness of breath due to a LBBB tachycardia leading to hemodynamic instability.

Diagnosis: The patient had a wide QRS tachycardia, with LBBB morphology and a heart rate of 160/minute. Echocardiography showed global hypokinesia with 25% left ventricular ejection fraction (LVEF). Considering the patient's clinical picture, TIC was suspected.

Interventions: The electrophysiological study revealed a left lateral accessory pathway. Catheter ablation was successfully performed at the level of the lateral mitral ring.

Outcomes: One week after the ablation the patient had no signs of heart failure and the LVEF normalized to 55%. During 6-months follow-up the patient presented no more episodes of tachycardia or heart failure and the LVEF remained normal.

Lessons: AVRT is rarely associated with intrinsic LBBB, being a potential cause of TIC. In these patients, it is unlikely to control the arrhythmia pharmacologically, catheter ablation being the best therapeutic option. The variation of QRS complex duration between LBBB pattern in SR and AVRT could be useful for early diagnosis of an ipsilateral AP on surface ECG.

Abbreviations: AP = accessory pathway, AVRT = atrioventricular reentrant tachycardia, CHF = congestive heart failure, LBBB= left bundle branch block, LVEF = left ventricular ejection fraction, SR = sinus rhythm, TIC = tachycardia-induced cardiomyopathy.

Keywords: catheter ablation, left bundle branch block, accessory pathway, mitral ring

1. Introduction

Tachycardia-induced cardiomyopathy (TIC) was first described in 1913 in a young patient who presented with congestive heart failure (CHF) and atrial fibrillation with rapid ventricular response, which resolved by control of arrhythmia.[1,2]

TIC is defined as systolic and/or diastolic ventricular dysfunction resulting from a prolonged elevated heart rate, which is reversible upon control of the arrhythmia or heart rate.[1–4]

Atrioventricular reentrant tachycardia (AVRT) is most frequently paroxysmal and can rarely occur in its incessant form, being one of the most frequent causes of TIC. Once it becomes incessant, it is unlikely to be controlled by pharmacological treatment, catheter ablation being the principal therapeutic option.[1,2]

The coexistence of intrinsic left bundle branch block (LBBB) in patients with AVRT may cause difficulties in the early diagnosis of tachycardia because of the wide complex morphology,[5–7] but most importantly, it can be the cause of the incessant nature of tachycardia.[8]

We present the atypical case of a patient with incessant wide QRS complex tachycardia lasting for 4 days, with hemodynamic instability, drug resistance, which was only resolved by emergency intracardiac overdrive pacing and subsequent catheter ablation.

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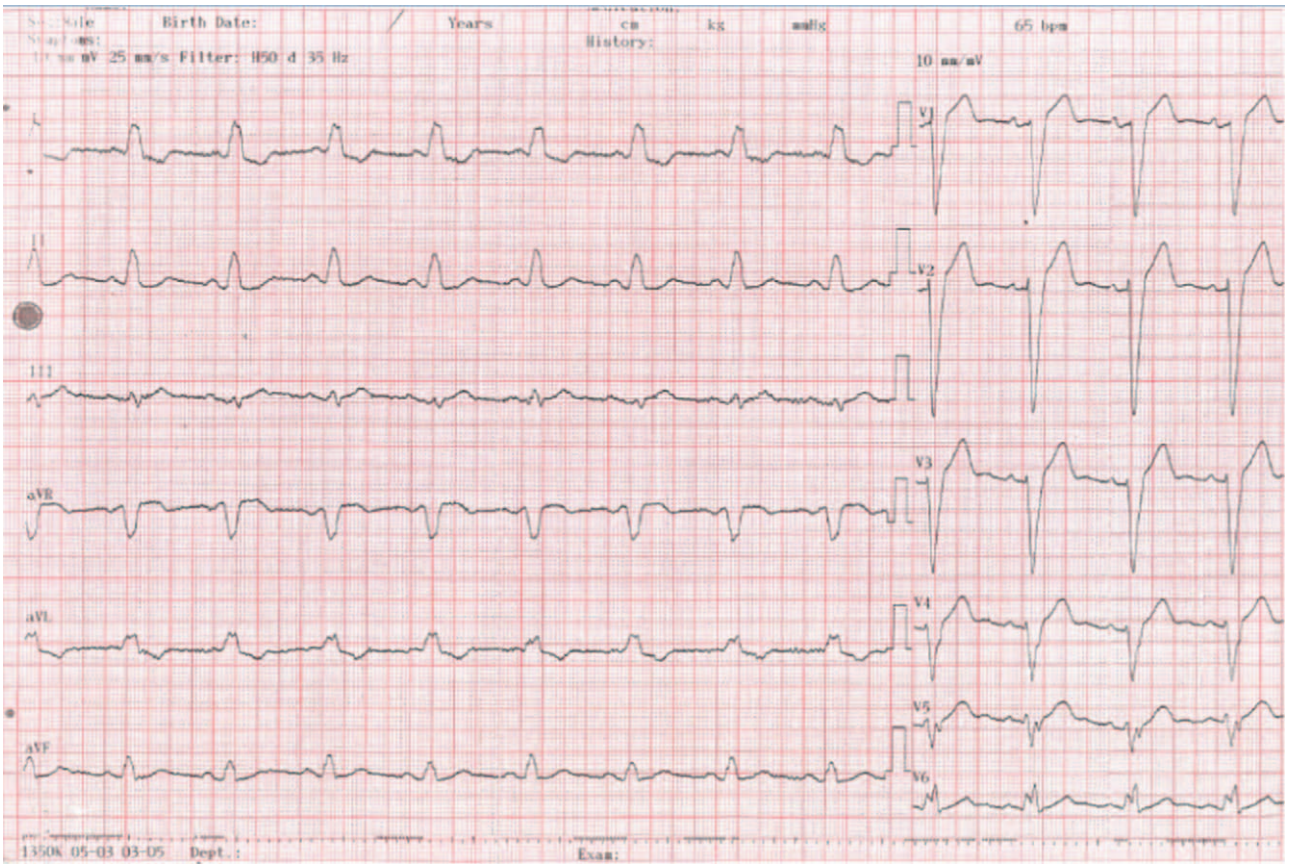

Figure 1. Sinus rhythms with LBBB after electrical cardioversion.

2. Case report

A 52 year-old Caucasian male patient, presented to the Emergency Department of a small town for palpitations. He had no medical history or past interventions. At presentation, ECG showed wide QRS tachycardia with LBBB morphology, 180/minute. The patient was given intravenous amiodarone with no effect on the arrhythmia. At 24 hour after admission, the patient showed CHF signs and symptoms with hemodynamic instability: blood pressure 80/40 mm Hg, pale fingers, dyspnea, transpirations, hepatomegaly, and edema. Electrical cardioversion was performed with conversion to sinus rhythm (SR) – also with LBBB morphology (Fig. 1), but with recurrence of tachycardia in the following minutes. Adenosine and verapamil were administered with no effect. As all therapeutic resources were exhausted without any change in arrhythmia and taking into account the progressive hemodynamic instability, the patient was transferred to a tertiary referral hospital.

The patient underwent an electrophysiology study, which found supraventricular tachycardia with 1:1 atrioventricular conduction that was stopped by overdrive pacing, with spontaneous recurrence after a few seconds through an atrial ectopic beat. During SR, positive delta waves could be seen in leads I, II, III, aVF, with rS complex in lead V1 and QRS transition in leads V4-V6 suggesting the presence of a right accessory pathway (AP) and antidromic AVRT. A temporary stimulation catheter was left in the right ventricle, with repeated conversions through overdrive pacing in the next few hours, with a reduction of tachycardia recurrence and clinical improvement. The patient was transferred to our service for AP catheter ablation ().

On admission to our department, the patient was hemodynamically and respiratorily stable, while ECG showed SR with the same LBBB morphology and preexcitation. We performed an electrophysiology study, with tachycardia recurrence at the beginning of the procedure (Fig. 3A).

During programmed atrial stimulation, a QRS shift from LBBB to right bundle branch block morphology could be seen (Fig. 3B), suggesting the presence of a left lateral AP, which was confirmed by the fusion between atrial and ventricular potentials in the distal coronary sinus catheter (Fig. 3C). We successfully performed AP ablation (Fig. 3D). During the procedure, we noticed a wider QRS complex during AVRT compared to SR, which made us suspicious about a second, right-sided AP, with anterograde conduction and antidromic AVRT. This possibility was excluded because of the constancy in AH and HV intervals during programmed atrial pacing, at the end of the procedure. Furthermore, we observed that the QRS complex during preablation SR was narrower compared to post-ablation SR (Fig. 3D). In both cases, the narrower QRS complex during preablation SR could be explained by the fusion between atrioventricular node with LBBB conduction and AP conduction (Fig. 4). After the procedure, the patient maintained SR with LBBB morphology and no preexcitation on surface ECG, while clinically and echocardiographically, 1 week after ablation, there were no signs of CHF, with the normalization of LVEF (Fig. 5).

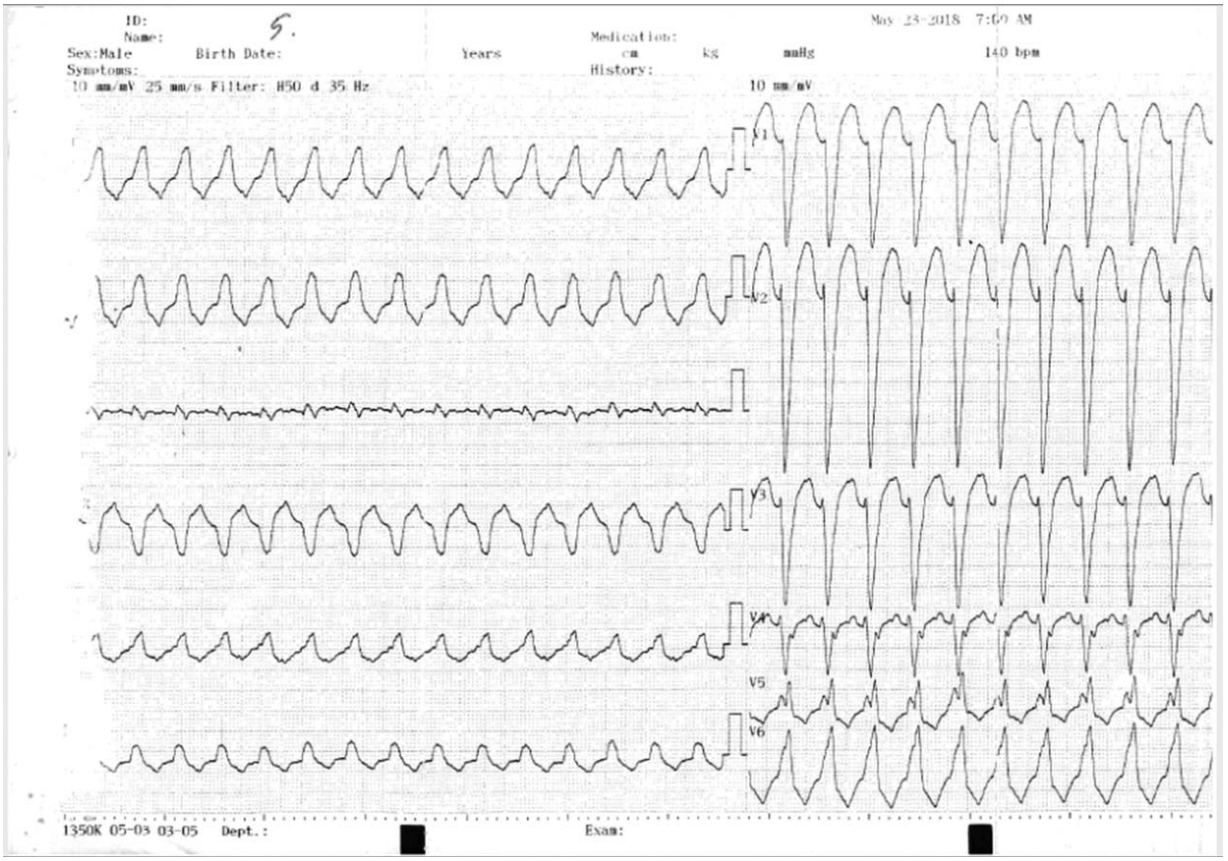

Figure 2. Wide complex tachycardia with LBBB morphology on admission to the regional hospital.

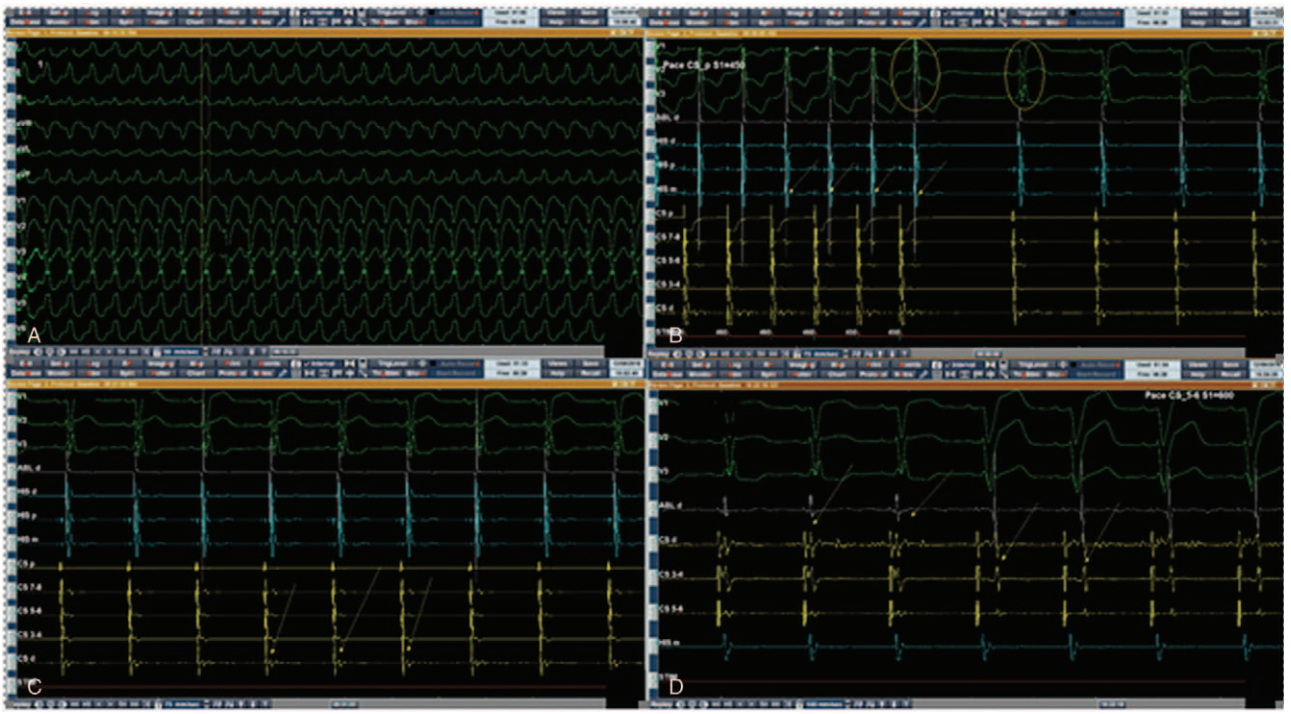

Figure 3. (A) Wide complex tachycardia at the beginning of the EP study. (B) During programmed atrial stimulation, a shift in QRS morphology from LBBB (second circle) to RBBB (first circle) can be observed. (C) Fusion between A and V potentials in the distal coronary sinus suggesting a left lateral AP. (D) The first two arrows show the presence of the AP – fusion between A and V potentials; the last two arrows show the disappearance of the fusion – successful ablation of the AP. Also, a wider QRS complex after ablation can be observed on surface ECG (V1-3).

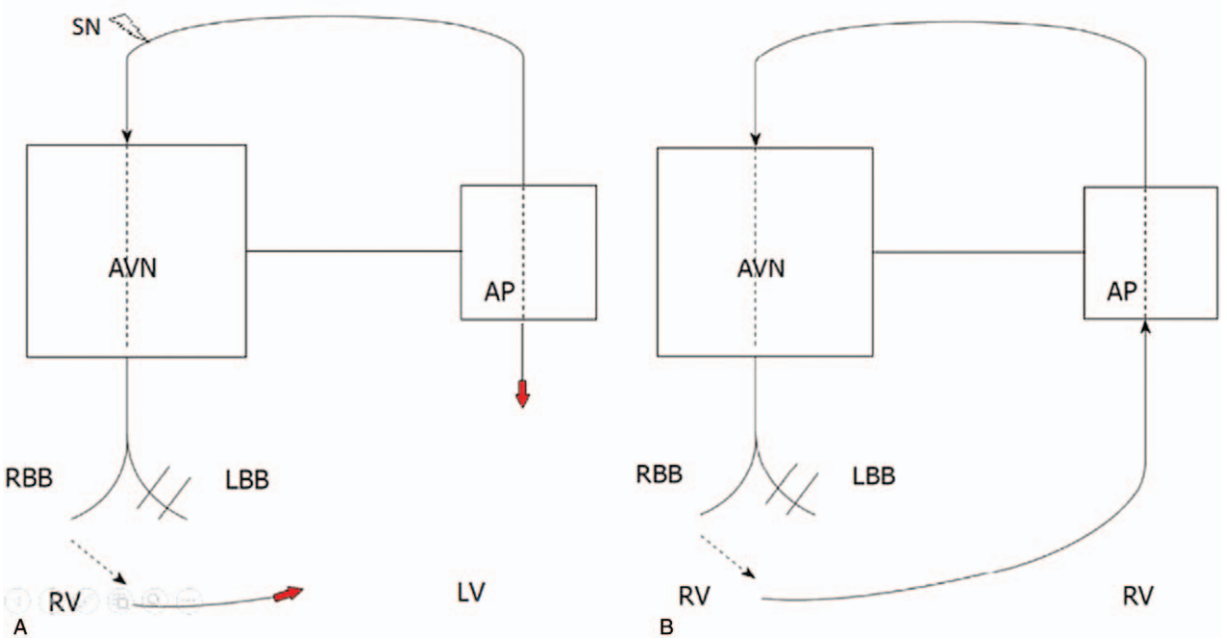

Figure 4. (A) The QRS complex in SR results from a fusion between AVN + LBBB conduction and left lateral AP conduction, which makes it narrower than in SR after ablation of the AP, and also narrower than in tachycardia. (B) Orthodromic AVRT with LBBB – the left lateral AP conducts retrogradely, so the QRS will be wider than in SR. (Adapted from[5]).

3. Discussion and conclusion

The fast clinical and echocardiographic recovery after arrhythmia control in a patient without a history of structural cardiac disease confirms the diagnosis of TIC. Taking into account the reversibility of heart failure after TIC, early arrhythmia control is the cornerstone of complete recovery in these patients. The fast normalization of LVEF in our patient, 1 week after arrhythmia control, is in accordance with the current literature data[1] and could be explained by the short overall time that our patient spent in tachycardia.

Even though AVRT is one of the main causes leading to TIC, the number of case reports in the literature is scarce. Catheter ablation, as performed by us in our patient, is the best therapeutic option once AVRT becomes incessant[1].

There are many algorithms for localizing APs in patients with LBBB, but most of them refer to patients with functional, not intrinsic BBB. Also, it is demonstrated that the presence of LBBB, by causing a delay in intraventricular conduction, facilitates the induction of orthodromic AVRT when a left-sided AP is present, which may be an explanation for the incessant nature of our tachycardia[8–10].

The coexistence of a left lateral AP and intrinsic LBBB is a rare finding in patients with AVRT and could be easily mistaken for antidromic AVRT with anterograde conduction via a right AP. The variation in our patient's QRS complex, narrower during pre-ablation SR compared to both tachycardia and post-ablation SR, could be explained by the fusion between LBBB pattern and left lateral AP conduction. In 1 study, the authors found that a QRS duration closer to normal during SR compared to tachycardia could be useful in diagnosing LBBB + ipsilateral AP on surface ECG. They also investigated whether fusion of LBBB pattern and AP conduction could provide physiological resynchronization in these patients. The conclusion was that even though some of them may benefit from left-sided AP conduction, the risks are higher than the advantages (i.e., malignant arrhythmias), catheter ablation being the best therapeutic option whenever possible.[11]

Finally, surface ECG may still play an important role in the diagnosis of wide complex tachycardia, even in patients with intrinsic LBBB. Concomitant LBBB and left lateral AP diagnosis on surface ECG could be challenging, "but not always unattainable" if some criteria are met: rapid conduction through the AV node and/or slow conduction through the AP in SR. In the absence of either of these 2 criteria, premature conduction through the AP will tend to conceal the LBBB pattern, resulting in a normal QRS duration on surface ECG.[12] However, if either of these is present, a narrower QRS complex during LBBB pattern SR compared to orthodromic tachycardia may be useful in unfolding an ipsilateral AP.

3.1. Consent

Informed written consent was obtained from the patient for the publication of this case report and any accompanying medical images.

Author contributions

Conceptualization: Ioan Alexandru Minciuna, Gabriel Cismaru.
Data curation: Gabriel Cismaru.
Methodology: Ioan Alexandru Minciuna.
Supervision: Gabriel Cismaru, Gabriel Gusetu, Horatiu Comsa, Dumitru Zdrenghea, Dana Pop, Rosu Radu.
Validation: Mihai Puiu, Gabriel Cismaru, Gabriel Gusetu, Bogdan Caloian, Dumitru Zdrenghea, Dana Pop.
Visualization: Gabriel Cismaru.

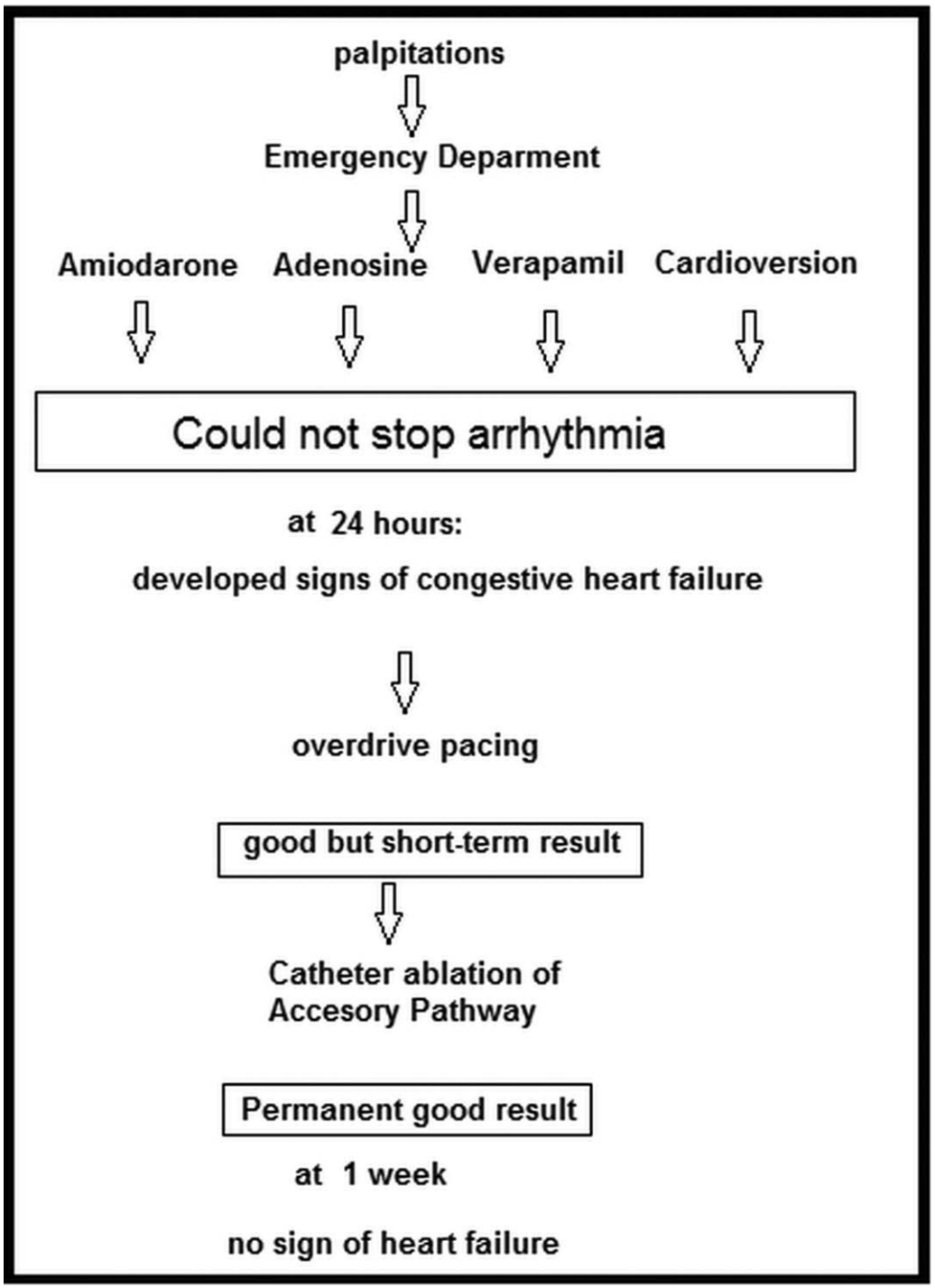

Figure 5. Flow chart with the evolution of the patient before and after catheter ablation of the accessory pathway.

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