Recovery of cardiac function after ablation of atrial tachycardia arising from the tricuspid annulus

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Tachycardia-induced cardiomyopathy is a reversible form of heart failure. An early diagnosis and an effective cure of the underlying tachycardia are crucial for a favorable outcome. Different kinds of atrial and ventricular arrhythmias may induce tachycardiomyopathy. Focal atrial tachycardia may be easily suppressed by means of transcatheter ablation. Relationships between focal atrial tachycardia and tachycardiomyopathy have not been deeply analyzed. In the present paper we report a case of a 76-year-old man with tachycardia-induced cardiomyopathy caused by recurrences of focal atrial tachycardia arising from the tricuspid annulus. The arrhythmia was successfully treated with transcatheter ablation. In the follow-up no recurrences of the arrhythmia occurred and a significant improvement in myocardial function was observed.

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Introduction

It is well known that a chronic tachycardia may induce a reversible form of systolic dysfunction: the so-called tachycardiomyopathy. Such a myocardial dysfunction may be partially or totally reversed by successful transcatheter ablation of the underlying persistent or permanent arrhythmia[1,2]. An early diagnosis of this condition is mandatory for an effective treatment.

Focal atrial tachycardia can be successfully suppressed by transcatheter ablation in the majority of cases with low incidence of complications and low recurrence rate[3-6]. Nevertheless, there is paucity of data in the literature about the relationship between focal atrial tachycardia and tachycardiomyopathy. In the present paper we describe a case of a man with episodes of focal atrial tachycardia inducing tachycardiomyopathy.

Case report

A 76-year-old man was referred to our hospital for ablation of supraventricular tachycardia. In the last years he had been admitted twice in other hospitals for dyspnea associated with supraventricular tachycardia. He had been symptomatic for almost 3 years for exertional dyspnea (NYHA class III) and for episodes of palpitations associated with dyspnea at rest. The patient had no history of systemic hypertension, myocarditis, and ischemic heart disease. Amiodarone (200 mg/day for almost 1 year) had been tested for the prevention of arrhythmia recurrences, but it revealed to be ineffective. No other antiarrhythmic drug except from amiodarone had been tested in the other hospitals. A 24-hour ECG Holter performed in one of the previous hospitalizations showed a mild sinus bradycardia (under beta-blockers and digitalis) at 56 b/min and three episodes of prolonged sustained supraventricular tachycardia (Fig. 1A) with a variable cycle length (ranging from 410 to 660 ms), a PR interval during tachycardia ranging from 140 to 160 ms and a long RP interval (Fig. 1B); the tachycardia stopped with a QRS complex not followed by a P wave (Fig. 1B); the P morphology of the three episodes of tachycardia in the three Holter leads was very similar. Not all the arrhythmic episodes were recognized by the patient. Therapy at the time of admission to our hospital was: carvedilol, digitalis, angiotensin-converting enzyme inhibitor, furosemide, and aspirin. Significant findings at physical examination were: a third heart sound; a 3/6 holosystolic blowing

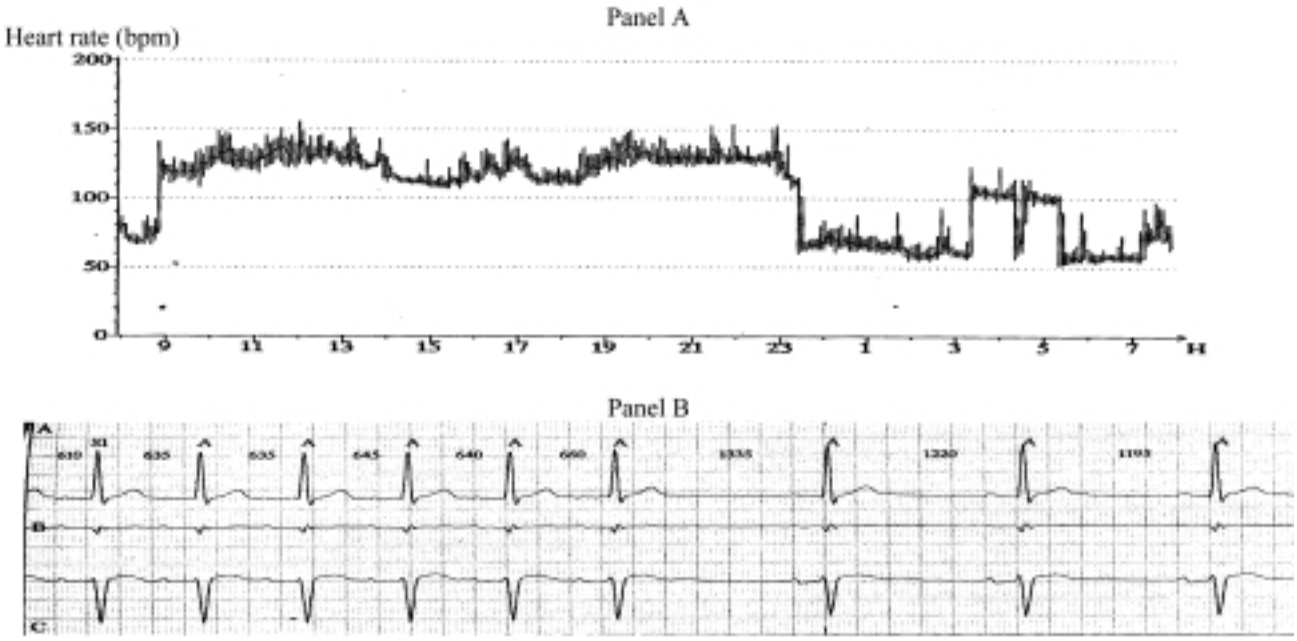

Figure 1. *Twenty-four-hour Holter ECG.* Panel A: *analysis of the heart rate (y axis) during the 24 hours (x axis). Three episodes of tachycardia are recorded.* Panel B: *example of tachycardia termination. During tachycardia a long RP > PR interval is observable. At tachycardia interruption a QRS complex not followed by P wave is observed. A, B and C = Holter ECG leads.*

murmur at the apex; no signs of pulmonary and/or systemic congestion were present. The ECG at admission showed atrial tachycardia at a heart rate of 115 b/min with atypical alterations of the recovery phase (Fig. 2). The P morphology was: positive in D1-aVL, biphasic in D2-D3-aVF and V_1, flat in aVR and in all the other precordial leads. It was possible to stop the tachycardia by means of carotid sinus compression. During carotid sinus compression, an increasing of the cycle length preceded tachycardia interruption, with prolongation of the PR and PP intervals (Fig. 3A). A single P wave during tachycardia not followed by the QRS complex was observed as well (Fig. 3B). Echocardiography in sinus rhythm showed a dilated left ventricular chamber (left ventricular end-diastolic diameter 62 mm), a severe depression of the systolic function with a left ventricular ejection fraction (LVEF) of 30%, without any abnormalities of the regional wall motion, and a moderate to severe mitral regurgitation; the diastolic transmitral flow pattern at Doppler examination was consistent with a good atrial contractility. The tachycardia revealed to be iterative and a repeated ECG showed atrial tachycardia at a rate of 155 b/min; the P morphology was very similar to the previous ECG. Laboratory examinations (including thyroid hormones) were in the normal range.

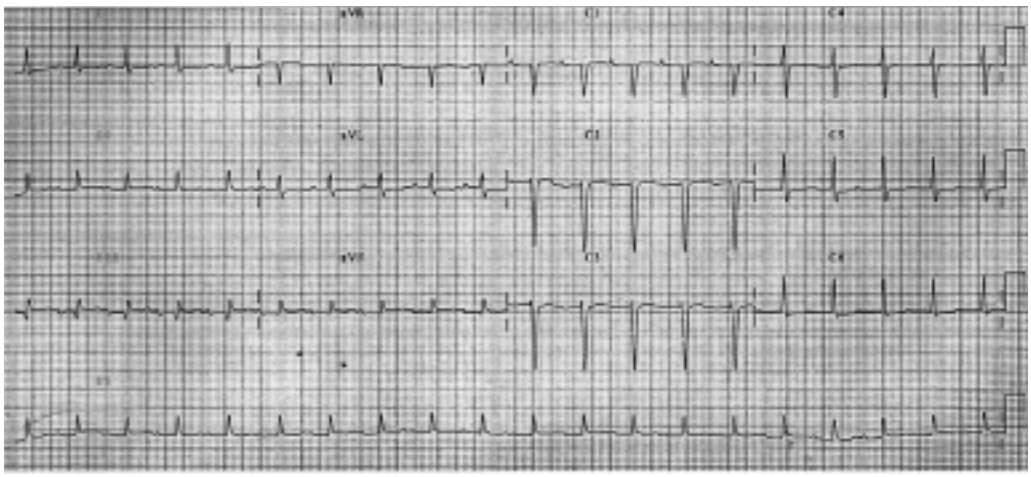

Figure 2. *Twelve-lead ECG during one episode of tachycardia. Supraventricular tachycardia with a long RP interval is observable. The tachycardia cycle length is 530 ms. For the description of the P wave morphology see the text.*

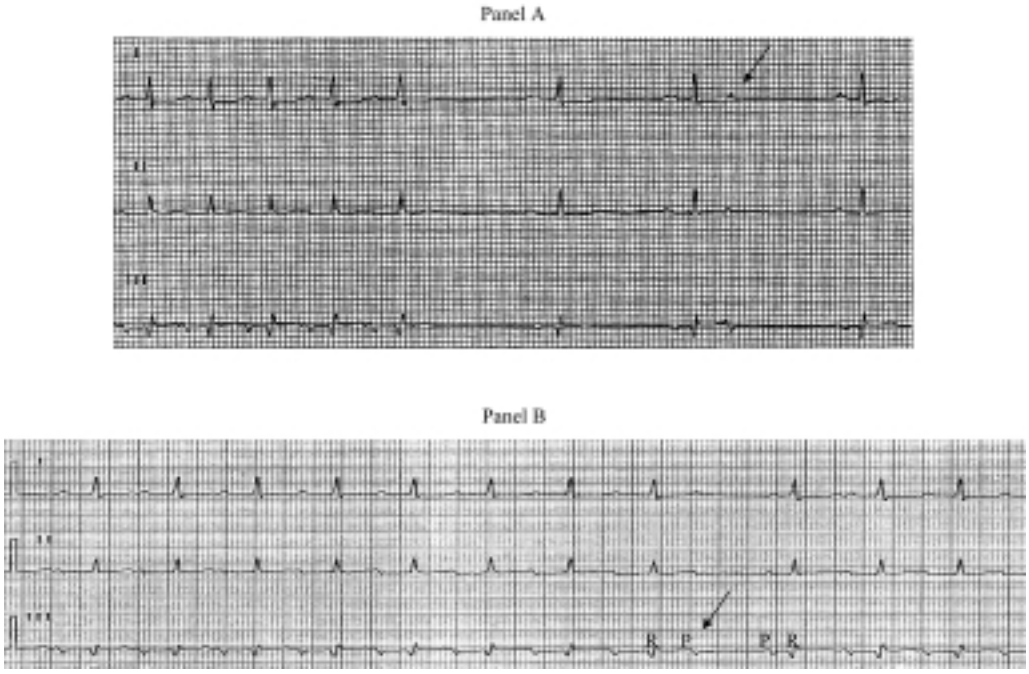

Figure 3. *ECG recording during carotid sinus compression. In* panel A *tachycardia interruption is observable. After two sinus beats an early premature atrial beat* (arrow) *not conducted to the ventricle (with the same morphology of the P wave during tachycardia) is recorded.* Panel B: *during carotid sinus compression a sequence of RPPR* (arrow) *can be observed; such a sequence is very suggestive of atrial tachycardia.*

An electrophysiological study was performed during spontaneous atrial tachycardia. A decapolar catheter was inserted into the coronary sinus; a quadripolar catheter was placed in the His bundle region and a Cordis Navistar 4 mm tip ablative catheter for electroanatomic CARTO mapping was placed in the right atrium. The tachycardia cycle length was 420 ms. A 3 mg infusion of intravenous adenosine immediately terminated the tachycardia without development of atrioventricular block. The tachycardia was reinducible both spontaneously and with a single atrial extrastimulus. During tachycardia, in the anterior aspect of the tricuspid annulus, the bipolar recording showed a low voltage double atrial potential preceding the onset of the P wave morphology at the surface ECG by 30 ms (Fig. 4). The unipolar potential of the P wave was totally negative in this site. The position was confirmed to be on the atrioventricular annulus according to the atrio-

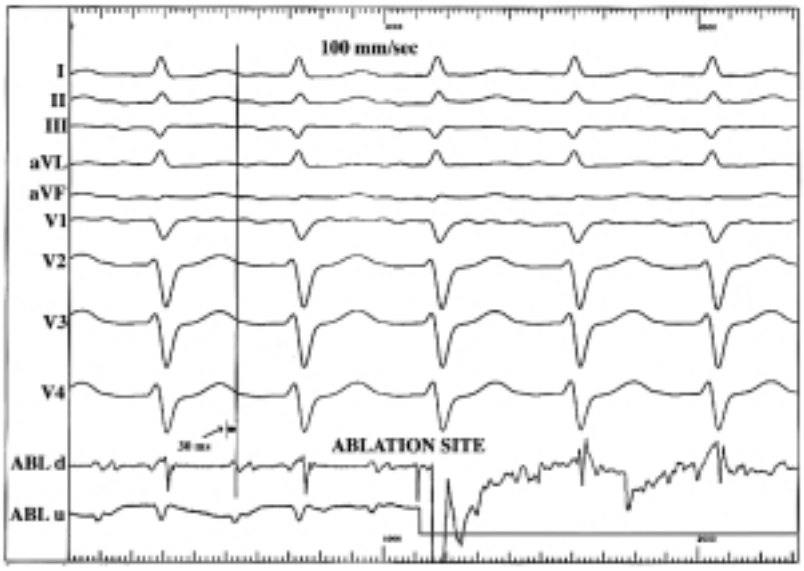

Figure 4. *Intracavitary recording during transcatheter ablation procedure. The ablator catheter is placed in the anterior aspect of the tricuspid annulus. During tachycardia a low-voltage double potential preceding the onset of the P wave at surface ECG by 30 ms* (arrow) *was recorded in this site. The unipolar potential corresponding to the P wave was totally negative.*

ventricular ratio between the local atrial electrogram and the ventricular electrogram, as previously described by Haissaguerre et al.[7], in addition to the anatomic findings at biplane fluoroscopy and to electroanatomic findings at CARTO mapping (Fig. 5). In this site a single radiofrequency application was effective to suppress the tachycardia (Fig. 6). After radiofrequency application the tachycardia was not inducible neither under isoproterenol infusion.

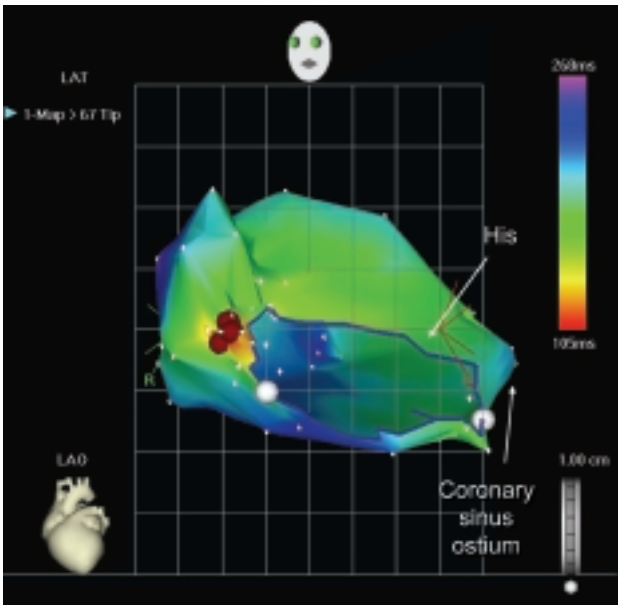

Figure 5. *Electroanatomical mapping (CARTO system) of the right atrium during tachycardia. In left anterior oblique (LAO) view, a small area of premature activation* (red and yellow) *is located in the anterior tricuspid annulus* (blue circle line). Red circles *are placed in the sites where radiofrequency energy was delivered. The sites of His bundle and coronary sinus ostium are indicated.*

At 8-month follow-up the patient was completely asymptomatic. The therapy was unchanged. ECG at 1, 3, 6, and 8 months showed sinus rhythm at a normal rate. Holter recording at 3 and 6 months did not show any recurrence of the arrhythmia. Echocardiography performed 6 months after ablation showed a left ventricular end-diastolic diameter of 55 mm, an LVEF of 48%, and mild to moderate mitral regurgitation.

Discussion

It is well known that myocardial dysfunction associated with chronic arrhythmias, the so-called tachycardiomyopathy, may be partially or totally reversed by eliminating the arrhythmia or by controlling the heart rate[1,2,8]. Two different forms of the disease can be recognized: a pure tachycardiomyopathy when the tachycardia arises in a normal heart and is the only cause of myocardial dysfunction, and an impure form when systolic dysfunction is caused by the tachycardia in association with other factors[9]. Although the precise pathophysiological mechanisms of systolic dysfunction are not completely understood, it seems reasonable that the duration of arrhythmia plays an important role as a determinant in the recovery of myocardial function following cure of tachycardia[1]. Thus, an early diagnosis is crucial for an effective therapy such as successful catheter ablation. Moreover, recurrent tachycardia after apparent resolution of tachycardiomyopathy has been described to be associated with a rapid decline in left ventricular function and development of heart failure, with a not negligible risk of sudden death[10]. For this reason, an effective treatment with a very low incidence of recurrences, such as ablation in comparison to drugs, would be preferable.

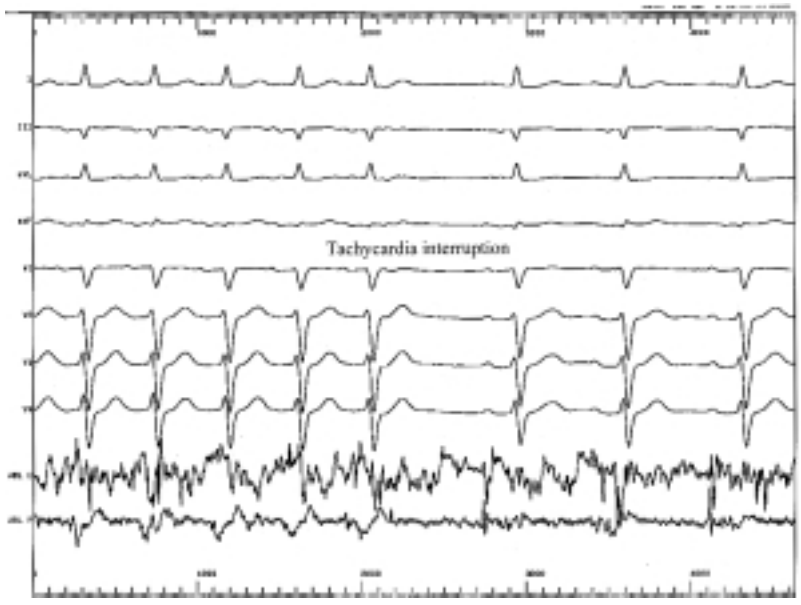

Figure 6. *Arrhythmia interruption during radiofrequency application (see the artifact in the recording of the ablator catheter); sinus rhythm is restored.*

In our patient, arrhythmia suppression by means of transcatheter ablation was correlated with an improvement in symptoms and LVEF. These results are in accordance with other observations in the literature[11-15]. However, LVEF in our patient resulted to be significantly improved, but not completely normal at 8-month follow-up. A possible explanation could be found in the clinical history of the patient. He had been admitted twice in other hospitals for dyspnea associated with supraventricular tachycardia. Unfortunately, the antiarrhythmic therapy (amiodarone) administered to the patient resulted to be unsuccessful in preventing tachycardia recurrences. Moreover, asymptomatic episodes of atrial tachycardia had been documented. So, it can be hypothesized that the deleterious effects of the rapid heart rate have been present for a significant long time. It can be argued if early arrhythmia ablation would have been associated with complete normalization of LVEF. Another possible hypothesis to explain such a clinical evolution can be drawn: by suppressing the tachycardia we probably eliminated only one of the hypothetical factors that were responsible for the cardiomyopathy. Even if an idiopathic dilated cardiomyopathy deteriorated by the tachycardia cannot obviously be excluded, no other factors known to precipitate heart failure were present in our patient.

Another interesting aspect of this case report concerns the electrophysiological features of the arrhythmia. The ECG morphology, the Holter recording, and the observation of QRS-P-P-QRS sequence during carotid sinus compression were very suggestive of atrial tachycardia. From the analysis of the findings at the electrophysiological study and from the electroanatomic reconstruction of the arrhythmia, it is clear that it was a focal atrial tachycardia arising from the anterior aspect of the tricuspid annulus. This anatomic location is in accordance with the anatomical definition of different sectors of the tricuspid annulus proposed in previously published guidelines[16]. The P wave morphology at the surface ECG during tachycardia was consistent with this location. Focal atrial tachycardia arising from the tricuspid annulus has been studied by Morton et al.[17]. These authors reviewed the data of 9 patients with tricuspid annulus focal tachycardia: the focus was identified to be infero-anterior in 7 and superior in 2 patients[17]. Also Matsuoka et al.[18] studied 5 patients with six different atrial tachycardias arising from the atrioventricular annulus. In such a population two sites of origin were on the mitral annulus, one right midseptal, two right postero-septal, and one classified as posterolateral[18], that corresponds to the infero-anterior definition of the paper of Morton. However, no site was identified to be in the anterior section of the tricuspid annulus as in our patient. The presence of a double potential in the effective ablation site during tachycardia in our patient confirms what was previously observed in the literature[18].

Moreover, it is interesting to underline that the response of the tachycardia to adenosine may reflect a typical behavior of atrial tachycardias arising from the atrioventricular annulus. McGuire et al.[19-21] investigated the electrophysiological characteristics of the cells around the atrioventricular annuli. These authors observed that atrioventricular cells around both annuli are histologically similar to atrial cells but resemble nodal cells in their electrophysiology, response to adenosine and lack of connexin-43[19]. In the paper of Matsuoka et al.[18] adenosine was effective to terminate atrial tachycardia in 5 out of 6 patients (in 1 patient adenosine infusion was not done). In our patient a very low dose of adenosine terminated the tachycardia. These observations support the hypothesis that tachycardias arising from the atrioventricular annulus may be sustained by such junctional cells with properties similar to nodal cells.

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