CASE PRESENTATION

The Challenges of Tachycardia Induced Cardiomyopathy - a Case Report

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ABSTRACT

Tachycardia-induced cardiomyopathy (TIC) is characterized by reversible left ventricular dysfunction caused by long-standing tachycardia. Treatment options for tachyarrhythmias causing TIC have evolved, especially the rhythm control strategies, ensuring a better and more sustainable control of the arrhythmia.

We report the case of a 46-year-old male presenting with acute heart failure, atrial fibrillation (AF) of unknown duration and severe left ventricular dysfunction, as well as left ventricular dilation. His medical history was relevant for atrial flutter treated with catheter ablation, hypertension, and frequent atrial ectopy for which he had been prescribed amiodarone. Coronary artery disease and other potential causes for left ventricular dysfunction were excluded with coronary angiography and cardiac magnetic resonance. Thus, the patient had a high suspicion of TIC. We opted for a rhythm control strategy, however, after a successful initial electrical cardioversion, he had AF recurrence a few days later, under classic heart failure medication and antiarrhythmics. Pulmonary vein isolation was then performed, with no complications. At the one-month follow-up visit the patient was arrhythmia-free and had a normal left ventricular ejection fraction, with a slightly enlarged left ventricle. We opted to continue the heart failure medication.

This case illustrates a typical case of AF induced TIC and the limited pharmacological options that exist for rhythm control, as well as the high efficacy of catheter ablation and value of imaging.

Keywords: tachycardia induced cardiomyopathy, atrial fibrillation, catheter ablation, rhythm control.

REZUMAT

Cardiomiopatia indusă de tahicardie (TIC) se caracterizează prin disfuncție reversibilă a ventriculului stâng cauzată de tahicardie persistentă. Opțiunile de tratament pentru tahiaritmii care cauzează TIC au evoluat, în special strategiile de control al ritmului, asigurând un control mai bun și mai durabil al aritmiei.

Prezentăm cazul unui bărbat în vârstă de 46 de ani care vine cu fenomene de insuficiență cardiacă acută, fibrilație atrială (FA) de durată necunoscută și disfuncție ventriculară stângă severă, precum și dilatare de cavități cardiace stângi. Din istoric său medical reținem flutter atrial tratat prin ablatie, hipertensiune arterială și ectopie atrială pentru care a trimit tratament cu amiodaronă. Boala coronariană și alte cauze potențiale ale disfuncției ventriculare stângi au fost excluse prin angiografie coronariană și rezonanță cardiacă. Astfel, pacientul avea o mare suspiciune de TIC. Am optat pentru o strategie de control al ritmului, și cu toate acestea, după o cardioversie electrică inițială reușită, FA a recidivat câteva zile mai târziu, sub medicamente clasice pentru insuficiență cardiacă și antiaritmice. A fost efectuată izolarea venei pulmonare, fără complicații. La controlul de o lună, pacientul nu avea aritmie și avea o fracție de ejecție a ventriculului stâng normală, cu ventricul stâng ușor dilatat. Am optat pentru continuarea medicamentului pentru insuficiența cardiacă.

Acest caz ilustrează un caz tipic de TIC indusă de AF și opțiunile farmacologice limitate care există pentru controlul ritmului, precum și eficacitatea ridicată a ablației și valoarea imagisticii cardiace.

Cuvinte cheie: cardiomiopatie indusă de tahicardie, fibrilație atrială, ablație, control al ritmului.

INTRODUCTION

Tachycardia induced cardiomyopathy (TIC) is an established cause of heart failure, characterized by left ventricular (LV) dysfunction in the setting of a long-standing tachycardia. The particularity of this pathology is its reversibility once the causative tachycardia has been treated.

We present the case of a patient with persistent atrial fibrillation (AF) who develops TIC and was successfully treated with catheter ablation (CA).

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CASE REPORT

We present the case of a 46-year-old man, who was admitted for progressive worsening of dyspnea in the past month, accompanied by heart palpitations and atypical chest pain.

The patient had no relevant family history, denied alcohol consumption and his personal medical history included a cavotricuspid isthmus (CTI) ablation for typical atrial flutter 5 years prior, hypertension and symptomatic atrial ectopy for which he had been previously started on amiodarone. His medication also included Nebivolol 5 mg per day and Perindopril 5 mg per day.

On clinical examination, blood pressure was 160/80 mmHg, heart rate was 120 beats per minute, respiratory rate was 30 breaths per minute and the oxygen saturation 90% while breathing ambient air. Furthermore, his cardiovascular examination showed signs of congestive heart failure: laterally displaced point of maximal impulse, irregular heart sounds, as well as a third heart sound, jugular distension (15 cm of H_2O), hepatomegaly with hepatojugular reflux, bilateral basal inspiratory crackles on pulmonary auscultation and marked bilateral edema of the lower limbs. The electrocardiogram showed AF (120 beats per minute), normal QRS duration and electric criteria for left ventricular hypertrophy with a strain pattern. The laboratory tests were remarkable for high levels of NT-proBNP (3000 ng/dl). Conversely, there was no increase in cardiac troponins, no inflammatory syndrome or dyselectrolytemia. The blood cell count, hepatic and renal functions were normal, as well as the thyroid hormones levels. Transthoracic echocardiography was performed, revealing a dilated LV (LV end diastolic volume of 200 ml measured by disk summation method), with concentric hypertrophy (wall thickness of 15 mm), global hypokinesia and a left ventricular ejection fraction (LVEF) of 30% calculated by Simpson biplane method. A mitral annulus dilation was also noted (mitral annulus anteroposterior diameter of 38 mm), as well as left atrial enlargement (45 ml/m^2 calculated by area-length method) and a grade II mitral regurgitation, secondary to the annular dilation.

The patient was shortly thereafter started on intravenous loop diuretics and digitalis, as well as low molecular weight heparin (LMWH) adjusted to weight. His symptoms improved slightly under medication. The treating physician then opted for rhythm control, therefore the patient underwent a transesophageal echocardiogram, which excluded left auricular thrombosis. Cardioversion was later performed under sedation (synchronous external electric shock with 200 J) and sinus rhythm was restored. Given the severe LV dysfunction with unknown etiology, several diagnostic tests were scheduled during the hospital stay. Firstly, given the chest pain and multiple risk factors for atherosclerosis, coronary angiography was performed documenting normal coronary arteries. Secondly, a cardiac MRI was performed, which showed severe LV dysfunction (LVEF 28%) with global hypokinesia, a dilated LV (LV end diastolic volume of 120 ml/m^2), concentric hypertrophy and no evidence of late gadolinium enhancement/ fibrosis.

The patient's embolic risk was assessed according to the current guidelines (CHA_2DS_2VASc = 2) and an indication for long term oral anticoagulation was established. The patient was started on an antivitamin K agent, superimposed with LMWH until a therapeutic INR was obtained. Oral amiodarone 200 mg per day was continued. Additionally, he received medication for heart failure: Furosemide 80 mg per day, Ramipril 10 mg per day, Nebivolol 5 mg per day and Spironolactone 50 mg per day.

During the seventh day of hospitalization, the patient developed a new episode of AF, which was poorly tolerated. Initial pharmacological cardioversion with intravenous amiodarone was attempted, but then abandoned in the favor of electrical cardioversion, which was performed successfully.

As the main causes of heart failure had been excluded, the patient had a high suspicion of TIC. He was presented with the option of pulmonary vein isolation using radiofrequency ablation (Figure 1), which was performed under therapeutic INR on the 9th day of admission. During the procedure, bidirectional block in the CTI was also confirmed. There were no post procedural complications. The patient was discharged in sinus rhythm, with the previously mentioned treatment.

On one-month follow-up, the patient was free of heart failure symptoms. His ECG showed sinus rhythm and his echocardiography showed significant improvement in LVEF (55% calculated by Simpson biplane), however a degree of LV dilation persisted (end diastolic volume 85 ml/m^2). The diuretics were stopped after the follow-up visit, amiodarone was stopped after three months, however the prognosis-modifying heart failure medication was continued.

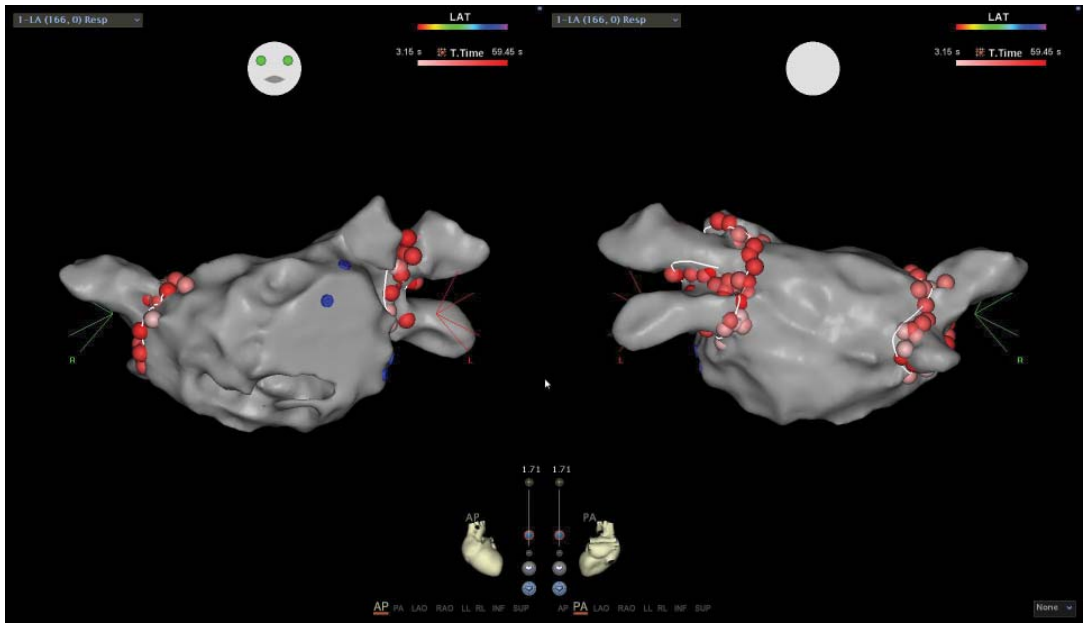

Figure 1. Three-dimensional reconstruction of the left atrium and pulmonary veins using an electroanatomic mapping system; circumferential isolation of the pulmonary veins.

DISCUSSION

Tachycardia-induced cardiomyopathy is a reversible cause of heart failure with reduced ejection fraction. The diagnosis is usually established when demonstrating the improvement in LV function after successfully treating the tachyarrhythmia, in the absence of other identifiable etiologies. In conclusion, TIC is essentially an exclusion diagnosis. Tachycardia-induced cardiomyopathy is described in various settings, such as AF, incessant supraventricular tachycardia, as well as ventricular tachycardia[1]; frequent arrhythmia may be the only cause for heart failure, or it may worsen a pre-existing condition. Treatment options are rate control or rhythm control therapy, pharmacological or interventional.

Our case illustrates a typical scenario of TIC related to AF, in a patient in whom other causes for LV dysfunction had been excluded through various imaging techniques. Imaging played an important role in the patient management, from the initial diagnosis of LV dysfunction using echocardiography, to the exclusion of primary cardiomyopathy using CMR. Echocardiography is ideal for patient follow-up in TIC, given its cost-efficacy. Through echocardiographic assessment after CA, we also identified persistent LV dilation, after resolution of LVEF. There are various reports in the literature of persistently increased LV volumes in patients with TIC in whom LVEF had normalized after adequate treatment[2]. The underlying mechanism of TIC caused by FA is not fully known, but some triggers have been described, such as the loss of atrial contraction and irregularity in contraction[3]. Echocardiography can identify changes associated with TIC in AF such as increased filling pressures and diastolic dysfunction, impaired LV contractile function and LV dilation, functional mitral regurgitation, which in turn can contribute to further LV dysfunction and dilation[2]. When eliminating the trigger, most of the changes previously described resolve, but LV dilation might persist. This could be explained by the persistent changes found at a cellular level, according to Muller et al.[4]. This information might support the continuation of heart failure medication even after the normalization of the LVEF. The significance of persistent morphologic changes of the LV and the possible impact on prognosis are yet to be clarified.

Furthermore, this case underlines the limited long term pharmacological rhythm control options for patients with TIC and AF, as well as the low likelihood of that medication to prevent recurrences. Given the failure of antiarrhythmic medication (amiodarone), our patient had a guideline-based indication[5] for CA, which successfully prevented AF episodes and allowed for almost total recovery of the LVEF. Catheter ablation has been shown to reduce AF recurrence (around 80% freedom from AF after multiple ablations)[6-9].

Landmark AF trials have initially compared a rhythm control strategy using CA and rate control strategy in patients with heart failure, concluding that CA is superior in improving LVEF in selected patients with heart failure[7,8]. The studies initially followed soft endpoints such as improvement in LVEF and freedom from AF recurrence, but later trials, such as CASTLE-AF, showed improvement in mortality and heart failure hospitalization in patients treated with CA[9]. The CASTLE-AF trial compared CA to medical management, that included both rate control and medical rhythm control with antiarrhythmics and showed the net superiority of CA. The only trial comparing rhythm control strategies (CA vs. amiodarone) in patients with AF and heart failure was AATAC-AF6. The results from this trial went on to confirm the superiority of CA in preventing AF recurrence, reducing heart failure admissions and mortality.

Lastly, this case is a reminder of the likely association between atrial flutter and AF[10] and the importance of periodic assessment for thrombotic risk, even in those successfully treated with catheter ablation of the CTI.

CONCLUSION

The diagnosis of TIC is difficult and requires a high degree of clinical suspicion. However, its recognition is of great importance, as it is one of the few potentially reversible cause of heart failure, with appropriate treatment. An early rhythm control strategy might significantly improve the chances of complete LVEF recovery. Interventional treatment of tachyarrhythmias causing TIC has gained ground in recent years and is proving to be superior to medical treatment.

Further studies are necessary in order to determine the ideal duration of heart failure medication in this patient population after the resolution of the LVEF, as well as the implications of persistent LV dilation on the prognosis of the disease.

Compliance with ethics requirements:

The authors declare no conflict of interest regarding this article. The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from all the patients included in the study.

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