

Persistent Atrial Fibrillation And Atrial Flutter Complicated By Tachycardiomyopathy Because Of Intermittent Conduction Through Accessory Pathway

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Abstract

The term tachycardiomyopathy refers to a specific form of tachycardia-related cardiomyopathy caused by supraventricular or ventricular tachyarrhythmias that are both associated with ventricular rates higher than 120 bpm. The arrhythmias which are most frequently associated with these forms of heart disease are atrial fibrillation and atrial flutter, particularly found in the elderly population. The most frequent clinical manifestation is heart failure. In this case we are reporting a clinical case of a patient that came to our attention because of an episode of heart failure associated with atrial fibrillation and atrial flutter. The patient had also prolonged and repetitive strips of rapid conduction with wide QRS morphology. We don't know if the cause is pre excitation or ectopia. We showed that those strips of tachycardia with wide QRS, particularly when they were associated with atrial flutter, were so fast and consistent to determine the left ventricular contractile dysfunction; we showed also that those strips of wide complex tachycardia were caused by pre-excitation through an accessory right posteroseptal pathway and supported by the reentry circuit of common atrial flutter. The block of conduction through the accessory pathway and the elimination of atrial arrhythmia allowed the regression of left ventricular contractile dysfunction. We believe that this case is interesting because it shows that there is a strict continuity between sophisticated electrophysiological mechanisms and clinical manifestation.

Introduction

The term tachycardiomyopathy refers to a specific form of tachycardia-related cardiomyopathy caused by supraventricular or ventricular tachyarrhythmias that are both associated with ventricular rates higher than 120 bpm. The arrhythmias which are most frequently associated with these forms of heart disease are atrial fibrillation and atrial flutter, particularly found in the elderly population. The most frequent clinical manifestation is heart failure.¹ Previous experiments with animals showed that the electrophysiological stimulation by atrial pacing leads to hemodynamic alterations, which appear early in the beginning of the 24 hours. These hemodynamic alterations are: the reduction of cardiac output and of ejection fraction, the increase of end diastolic intracavitary pressure and of peripheral vascular resistance. Furthermore these hemodynamic alterations appear to be linked to two factors: the duration/chronicity of the tachycardia

and the mean ventricular frequency.² The diagnosis of these forms of heart disease can be not immediate. In fact, especially in the elderly population, the opposite is more frequent: heart failure, with the electromechanical feedback and the neurohumoral activation is responsible of atrial fibrillation.³ Here we report the clinical case of a patient that came to our attention because of episode of heart failure associated with prolonged and repetitive strips of rapid conduction through an accessory pathway during atrial fibrillation and atrial flutter.

Case Report

G.C. is a 72 years old male. In 1991 he underwent a left pneumonectomy because of lung adenoma; seven years later a bilateral inguinal lymphadenectomy. In 2008, he had the first episode of atrial fibrillation, which was treated with electrical cardioversion; in 2011 he had his second arrhythmia treated with electrical cardioversion and antiarrhythmic prophylaxis therapy with dronedarone until another relapse in 2012. At this time neither clinical or echocardiographic signs of left ventricular dysfunction were present. A baseline 24 hours ECG recording showed some short stretches of wide-QRS tachycardia interpreted as non-sustained ventricular tachycardia. In 2012 the patient underwent a radio frequency pulmonary veins ablation with sinus rhythm restoration. He was assigned to antiarrhythmic therapy with amiodarone, digoxin and bisoprolol. In 2013 the baseline 24 hours ECG recording proved the persistence of

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Disclosures:
None.

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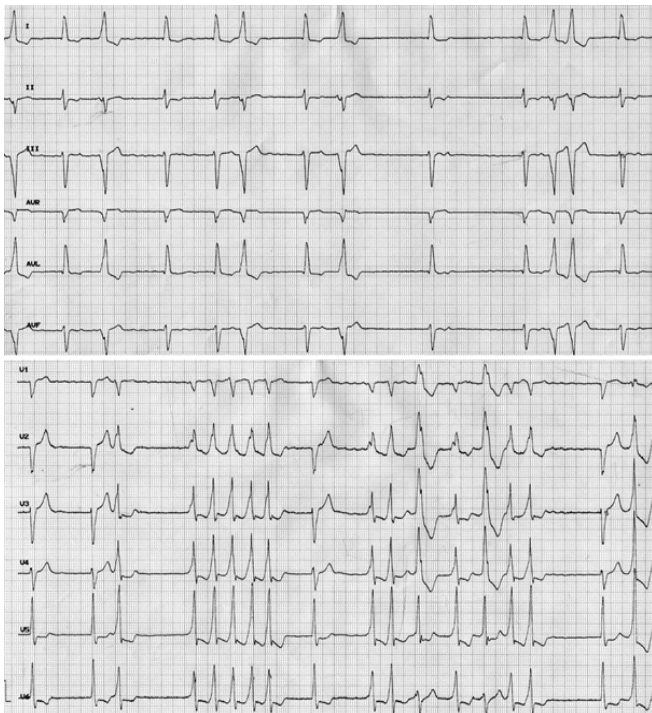


Figure 1:

Atrial fibrillation with heart rate < 100 bpm alternating with strips of tachycardia with a wide QRS morphology, iterative, 3-10 sec in duration, with variable heart rate (ranging from 90 to 160 bpm), not showing critical coupling and Ashman Phenomenon

sinus rhythm and the disappearance of the strips of non-sustained ventricular tachycardia. In february 2015 he experienced another atrial fibrillation that was treated effectively with electrical cardioversion. A few weeks later the patient arrived in the Emergency Department because of dyspnoea and palpitations. The clinical examination showed the presence of rales > 50 % of the two lung fields and high jugular venous pressure. The brachial blood pressure was 120/80 mmHg, the body temperature was 36.7 C, the hourly diuresis was 20 cc/h, the heart rate was 98 bpm; the peripheral pulse oximetry was 88 %. The chest X ray showed alveolar oedema. The main laboratory parameters were: creatinine 1.9 mg/dl; hemoglobin: 12 mg/dL, Na: 134 mEq/L, K: 4.5 mEq/L, pH: 7.42, pO₂: 86%, pCO₂: 38, bicarbonates: 24, lactates: 0.9. The ECG examination showed the presence of atrial fibrillation with heart rate < 100 bpm. This arrhythmia was alternating with strips of tachycardia with a wide QRS morphology having suspected delta wave with negative polarity in limb leads and positive polarity in leads I, aVL, and V3 to V6. The strips were: iterative, appeared after the cycle lengthening, lasted 3-5 sec with variable heart rate ranging 90 - 160 bpm and without critical coupling (fig. 1). The echocardiogram showed a not dilated left ventricular with severe contractile dysfunction (EF 30 %), septal-to-posterior wall motion delay as a measure for LV dyssynchrony and moderate functional mitral regurgitation. The patient was initially treated with furosemide and oxygen with good clinical response and resolution of fluids overload. A few days later we planned to perform a coronary angiography that excluded the presence of coronary artery disease. Then we restored the sinus rhythm through the electrical cardioversion and we planned to perform the electrophysiological study for detailed diagnosis and treatment of the arrhythmia. The question was: is this arrhythmia a ventricular tachycardia or a pre-excited supra ventricular tachycardia? However, the next day there



Figure 2:

Atrial flutter steadily conducted to the ventricles with a 2.1 ratio and wide QRS totally pre excited, showing delta wave with negative polarity in limb leads and positive polarity in leads I, aVL and V3-V6 suspected for conduction through a posteroseptal pathway

was a recurrence of typical atrial flutter that are conducted to the ventricles with a 2.1 ratio and wide QRS totally pre excited, identical in morphology to those present during atrial fibrillation and suspected of conduction through a posteroseptal pathway (fig. 2). With typical atrial flutter, the wide QRS conduction was constant, ranging 110 - 130 bpm in rate. This longtime high rate was responsible of cardiomyopathy. The patient was symptomatic for palpitations and mild exertional dyspnoea. The electrophysiological study, performed during atrial flutter and wide QRS showed a short HV interval in confirmation of the initial activation of the ventricle by an accessory pathway (fig. 3). We wanted to perform the ablation of the accessory pathway or of the atrial flutter but this was not possible because it was very difficult to access the left subclavian and femoral veins: in fact, in the past, the patient underwent a left pneumonectomy and bilateral inguinal lymphadenectomy. Therefore we performed the electrical cardioversion and we assigned anti arrhythmic therapy with amiodarone. The patient was discharged from hospital with sinus rhythm. One month later he was asymptomatic; sinus rhythm persisted and echocardiographic data had significantly improved with decrease of septal to posterior wall motion delay, decrease of mitral regurgitation and improvement in systolic function.⁴

Discussions

This case captured our interest for three reasons in particular. The first concerns the diagnosis of tachycardiomyopathy that was very difficult to make because of two factors. The most important: the phases of fast ventricular conduction with wide QRS were not constant during atrial fibrillation, which was the first arrhythmia detected at the time of admission in the hospital, but the strips with fast and wide QRS were developed in the patient during atrial flutter, a few days later, after the restoration of sinus rhythm. At first it seemed unlikely that fast and wide QRS runs, when they were in the context of atrial fibrillation, iterative, mostly conducted with narrow QRS and intermediate ventricular rate, could sustain a ventricular contractile dysfunction. But when the patient developed atrial flutter he had totally wide, fast and constant QRS strips. At this point, the correlation with the left ventricular contractile dysfunction appeared to be more plausible.⁵ The second factor that initially discouraged the diagnosis of tachycardiomyopathy was the knowledge that, most frequently in the elderly population, the atrial fibrillation and the atrial flutter are a result of heart failure and not the cause.³

The second aspect that captured our interest was the diagnosis of

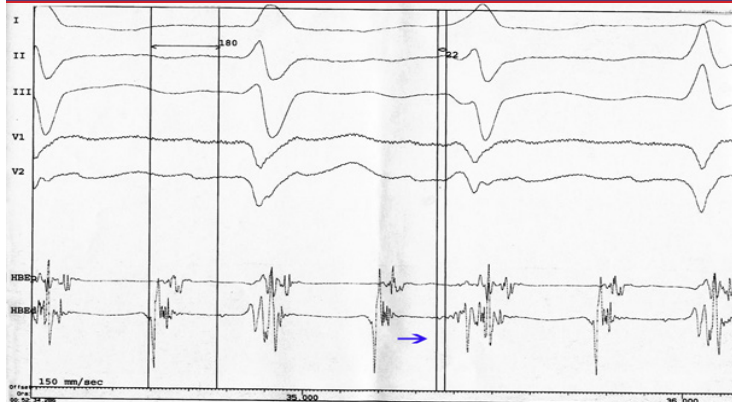


Figure 3: The electrophysiological study, performed during atrial flutter and wide QRS pre excited showing a short HV interval in confirmation of the initial activation of the ventricle by accessory pathway

runs of wide complex tachycardia. Indeed this clinical case shows us the hard dilemma of wide QRS during atrial fibrillation: ventricular ectopy, preexcitation or aberrant ventricular conduction?⁶ We immediately ruled out the third hypothesis: the QRS morphology was not aberrant and there was no Ashman phenomenon. However, on the basis of the ECG only, it was much more difficult to distinguish between ventricular ectopy and pre-excitation even if the morphology of the QRS and the absence of fixed coupling, clearly suggested a ventricular pre-excitation through a posteroseptal right accessory pathway.⁷ Objections to the diagnosis of pre-excitation came from the presence of certain elements in favor of ventricular ectopy such as the tendency to grouping, the iterative nature and the compensatory pauses. Furthermore, the patient had never presented ventricular preexcitation unless an error occurs in the interpretation of non sustained ventricular tachycardia that was observed in 2012 during 24 hours ECG recording. Therefore, on the basis of all these arguments it seemed highly probable that the runs of wide QRS were caused by ventricular pre-excitation through a posteroseptal right accessory pathway which was manifest only when the atrial activation was non originated in the sinus node but in the circuit of atrial fibrillation and, particularly, of atrial flutter. This pathway was easily depressed by various antiarrhythmic drugs such as amiodarone.

The third aspect that captured our interest was the most intriguing: to explain why pre-excitation was constant during atrial flutter. Probably this depends on the reentry circuit of the common flutter in which the wave front rotates around this counterclockwise. In this way, as the activation of coronary sinus and ostium precede the activation of AV node and bundle His, the wave front can activate the posteroseptal pathway in advance and make the preexcitation manifest.

The electrophysiological study, although limited by the inability to perform the ablation, has demonstrated, during typical atrial flutter conducted with wide complexes, a virtual HV interval. The study therefore confirmed the diagnostic hypothesis that, during atrial flutter, the conduction to the ventricles occurs mainly through the accessory pathway.

Conclusions

This case report shows that more sophisticated mechanism than those initially evident can be hidden behind an episode of a heart failure. The conduction to the ventricles through an accessory pathway can be supported by the presence of atrial arrhythmia and therefore not be evident during sinus rhythm. If the pre excited complexes are

rapid and prolonged they can be responsible of tachycardiomyopathy and heart failure. As other cases of tachycardiomyopathy demonstrate, the signs of left ventricular contractile dysfunction may regress with the control of the heart rate. In this specific case, this meant the blocking of the conduction through the accessory pathway and the elimination of atrial arrhythmia. The latter supports the conduction through the accessory pathway. It remains to prove the long-term effectiveness of the applied treatment, that is the pharmacological therapy with amiodarone plus atrial electrical cardioversion. We are aware of the limitation of not having been able to subject the patient to ablative treatment because of lack of vascular access.

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