

November 2016

# Mahaim Tachycardia Induced Cardiomyopathy

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CASE REPORT

# Mahaim Tachycardia Induced Cardiomyopathy

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## ABSTRACT

We present the case report of a 22-year man, with incessant palpitations, chest pain, shortness of breath, and pulsations in his neck for the past 7 months. He was referred to the cardiology unit for workup of wide complex tachycardia (WCT). His echocardiography, 6 months earlier, had demonstrated severe left ventricular (LV) systolic dysfunction, severe global hypokinesia, mild tricuspid regurgitation (TR), and mild mitral regurgitation (MR) which resolved with medical therapy including beta-blockers. He underwent electrophysiological study, which revealed a decremental right sided atriofascicular pathway causing a WCT with left bundle branch block (LBBB) morphology and left axis deviation (LAD, Mahaim tachycardia). This was successfully ablated by radiofrequency ablation (RF) with abolition of the tachycardia. This case report highlights Mahaim tachycardia induced cardiomyopathy, a rare but curable cause of cardiomyopathy.

**Key Words:** *Mahaim tachycardia. Wide complex tachycardia. Decremental atriofascicular pathway. Cardiomyopathy. Atrioventricular. Radiofrequency ablation. Atrioventricular reentrant tachycardia (AVRT). Extrastimulation. Incremental pacing. Entrainment. Tachycardia induced cardiomyopathy.*

## INTRODUCTION

Tachycardia induced cardiomyopathy (TIC) is caused by persistent supraventricular (SVT) or ventricular tachyarrhythmia (VT), characterized by LV systolic dysfunction, dilatation and clinical manifestations of heart failure which are reversible with normalisation of the heart rate and may otherwise have a less favourable prognosis.[1,2] Mahaim tachycardia is an unusual form of SVT of the atrioventricular reentrant tachycardia (AVRT) variety, dependent on a decremental accessory pathway that conducts only in anterograde direction.[3] TIC dependent on this unusual AVRT is a rare presentation.

We present a rare case of Mahaim TIC which resolved with the control of heart rate with beta blockers.

## CASE REPORT

A 22-year man with no addiction or other comorbidities, came to our clinic with history of recurrent palpitations, left side chest pain with associated sweating, shortness of breath, and pulsations in his neck for the past 7 months. There was no significant family history and no history of upper or lower respiratory tract infection. His baseline ECG is shown in Figure 1. The ECG of the tachycardia revealed a wide complex tachycardia (WCT) with a left bundle branch block (LBBB) morphology and left axis deviation (LAD, Figure 2). His previous laboratory workup revealed normal thyroid profile and echocardiography done 6 months back revealed a visually estimated ejection fraction (EF) of 30% with severe global hypokinesia, left ventricular end systolic dimension (LVESD) of 47 mm, left ventricular end diastolic dimension (LVEDD) of 55 mm, left atrial size of 32 mm, mild mitral regurgitation (MR), mild tricuspid regurgitation (TR) and pulmonary artery systolic pressure (PASP) of 40 mmHg. No other structural abnormalities were identified. He was taking acetylsalicylic acid, beta-blockers (bisoprolol) and ACE inhibitors (ramipril) with which his tachycardia had been relatively controlled and his EF had improved to a mild dysfunction with a visually estimated EF of 50% along with normalization of the cardiac chambers. However, the frequency of episodes of tachycardia had recently increased despite taking medications. His last episode was 6 days prior to visiting our clinic. He was booked for an electrophysiologic study with a view to ablation. His laboratory workup showed normal thyroid function, normal electrolytes and renal function. On the day of the procedure, he was in incessant tachycardia. The morphology of the tachycardia was consistent with prior tachycardia episodes. It was terminated using 12 mg of intravenous adenosine.

Electrophysiological (EP) study showed baseline HISS to ventricle (HV) interval of 30 ms. The WCT was easily inducible with atrial extrastimulation from the high right atrium (HRA). Straight pacing from the HRA caused full pre-excitation at lower rates than pacing from the proximal coronary sinus (CS). The ventriculo-atrial (VA) conduction was present. The retrograde atrial activation was concentric and decremental. The tachycardia was entrained from the right ventricular (RV) apex and the HRA, ventricular-atrial-ventricular (VAV) and atrial-ventricular-atrial (AVA) responses, respectively, were noted to the cessation of entrainment, ruling out atrial tachycardia and ventricular tachycardia as the possible

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*Received: June 25, 2015; Accepted: June 06, 2016.*

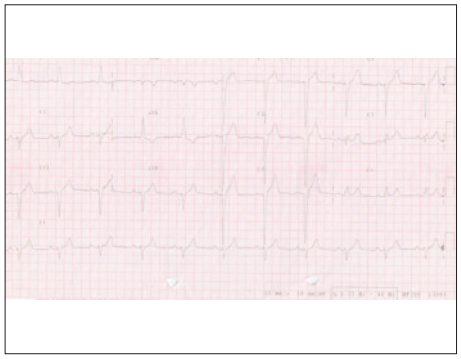

**Figure 1:** 12 lead ECG showing normal sinus rhythm, left axis deviation, T wave inversion in lead aVL, partial left bundle branch block and poor R wave progression in anterior chest leads.

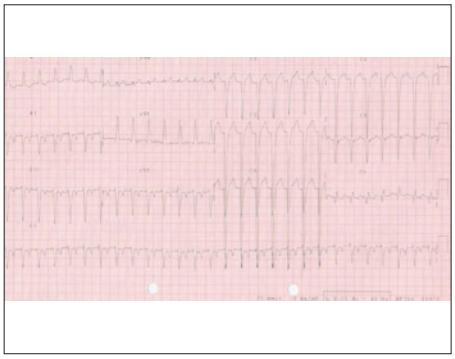

**Figure 2:** ECG showing wide complex tachycardia, left bundle branch block and left axis deviation.

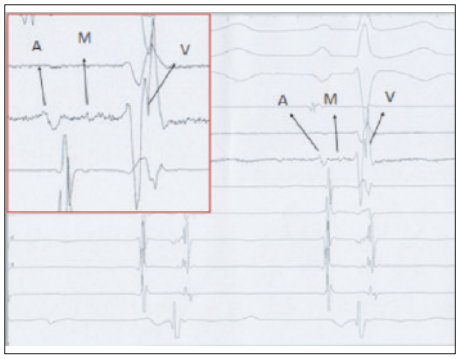


**Figure 3:** EP trace showing M-Potential, **A:** Atrial activation, **M:** M-Potential, **V:** Ventricular activation. In the inset, a magnified form of the EP trace has been shown.

causes. The remaining possibility was an atrio-ventricular reentrant tachycardia (AVRT) or an atrio-ventricular nodal reentry tachycardia (AVNRT) with aberrant conduction. The post-pacing interval (PPI) minus tachycardia cycle length was 70 ms with a VAV response. The VA time was 95 ms. In view of an LBBB morphology tachycardia with pre-excitation at baseline and diagnostic EP maneuvers, the diagnosis of atrio-fascicular tachycardia (Mahaim tachycardia) was made.

An eight French (8F) Mullins sheath was used for catheter stability prior to ablation. The tricuspid annulus was mapped during sinus and atrial paced rhythm. A Mahaim “M” potential (Figure 3) was noted at 9 O' clock position (lateral tricuspid annulus, left anterior oblique (LAO) 30° projection). Radiofrequency (RF) energy was applied using power at 50 Watts and temperature of 60°C, for one minute. Thereafter no pre-excitation was noted on incremental pacing or extra-stimulation. No tachycardia was inducible. VA conduction remained intact and unchanged and no evidence of another accessory pathway was found. The patient left the laboratory in stable condition and was discharged the same day.

He was followed up in the clinic at 6 weeks and 6 months and was doing well without recurrence of tachycardia. A repeat echocardiogram at 6 weeks of ablation, revealed a normal LV systolic function with visually estimated EF of 55%, LVESD of 30 mm, LVEDD of 50 mm, trace TR, trace MR and no pulmonary hypertension.

## DISCUSSION

TIC is a relatively rare entity, but is readily treatable with a good prognosis in many patients.[2,4] While the exact incidence of TIC remains unclear, an association between tachycardia and cardiomyopathy has been recognized for some time.[5] Virtually, every form of supraventricular tachyarrhythmia, including ectopic atrial tachycardia, non-paroxysmal junctional tachycardia, and atrial fibrillation, has been associated with reversible LV systolic dysfunction or cardiomyopathy.[6,7] The development of a cardiomyopathy has also been documented with ventricular tachyarrhythmias and frequent ectopic beats.[8] Even incessant short bursts of tachycardia have been known to cause TIC.

Mahaim atriofascicular or atrioventricular accessory pathways with decremental, anterograde only conduction are uncommon. The most common clinical manifestation related to these pathways is antidromic reentrant tachycardia.[9] However, the progression of this kind of tachycardia to dilated cardiomyopathy is a very rare presentation.

In this case, an unusual cause of AVRT (a right sided atriofascicular pathway with only anterograde decremental conduction) led to TIC due to increasing frequency of both sustained and non-sustained tachycardia. It was successfully ablated with RF ablation, resulting in resolution of patient's symptoms.

A similar case was reported recently from India, where automatic parasystole was reported to arise from the atriofascicular pathway causing not only tachycardia but also contributing to TIC due to its frequency. Both were settled after the successful ablation of the pathway.[10]

This case is a rare combination of two unusual entities (Mahaim tachycardia and TIC). Mahaim pathways are amenable to ablation which is curative for the AVRT. TIC is a serious but reversible disease with a favourable outlook, provided a timely diagnosis is made and the underlying cause of tachycardia is treated.

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