

Tachycardia-induced cardiomyopathy caused by a special pacemaker algorithm

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Abstract

An elderly lady presented with breathlessness, fever and palpitations. She was treated for community-acquired pneumonia with antibiotics. Subsequently, she went into acute pulmonary edema. She was managed with standard heart failure medications and a trial of non-invasive ventilation. ECG showed A-V sequential pacing at a rate of 150/min with broad QRS. It was thought to be a pacemaker-related tachycardia. On pacemaker check, a special algorithm (atrial fibrillation suppression algorithm) was switched off. She subsequently improved. We here review the cause of this tachycardia-induced cardiomyopathy. (Cardiol J 2011; 18, 5: 573–576)

Key words: pacemaker, tachycardia-induced cardiomyopathy

Introduction

Tachycardia-induced cardiomyopathy is a well-established cardiac condition with a good prognosis. Herein we describe a case of tachycardia-induced cardiomyopathy caused by a special algorithm mode in a dual chamber pacemaker.

Case report

An 80 year-old woman presented to the ED with a two-day history of breathlessness, cough and fever. She mentioned experiencing palpitations but denied any chest pain or syncope. Her past medical history included sick sinus syndrome for which a dual chamber pacemaker (Identity, SJM, Sylmar, CA, USA) had been implanted four months earlier. An echocardiogram prior to implantation showed normal left ventricular systolic function with preserved ejection fraction. She was independent and mobile and lived alone. On examination she was mildly dyspneic. Her pulse rate was 140 bpm (regular), blood pressure 110/70 mm Hg and temperature 39.9°C. Chest auscultation revealed bibasal crepitations. Cardiovascular examination revealed

normal heart sounds with no added sounds/murmurs. There was no pedal edema. Blood tests showed a raised WCC at $20 \times 10^9/L$ with normal U&E. Chest X-ray showed cardiomegaly with bibasal shadowing. She was treated for community-acquired pneumonia with antibiotics. ECG showed sequential atrio-ventricular (AV) paced rhythm with a rate of 140/min as shown in Figure 1.

She was transferred to a general medical ward, but her dyspnea increased. On further assessment, she was found to be in acute pulmonary edema. ECG showed A-V sequential pacing at a rate of 150/min but the QRS was broader than in the admission ECG. She was transferred to the coronary care unit and managed with diuretics, standard heart failure medications and a trial of non-invasive ventilation. Her ECG rhythm was thought to be a pacemaker-mediated tachycardia. Hence a magnet was applied on the pacemaker site, but this revealed an underlying narrow complex tachycardia interspersed with paced complexes and pacing spikes with functional non-capture. On removal of the magnet (to prevent the risk of R-on-T phenomenon), the rhythm was a narrow complex tachycardia (intrinsic sinus tachycardia) at a rate of 145 bpm, which changed quickly

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Figure 1. Sequential atrio-ventricular paced rhythm with a rate of 140/min (admission ECG).

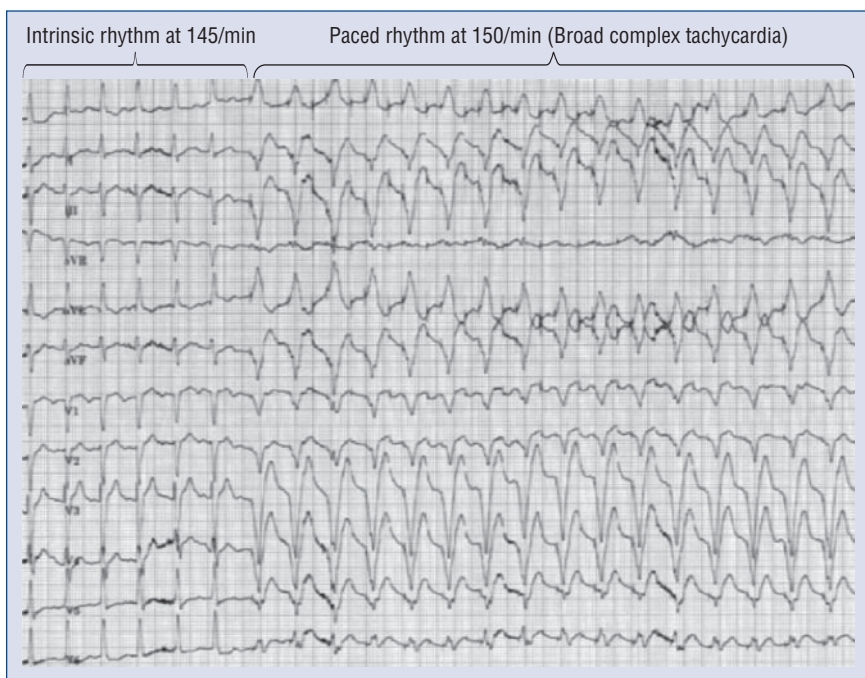


Figure 2. ECG on removal of magnet.

to a broad complex tachycardia (A-V sequential pacing) with a rate of 150 bpm within 2 s, as shown in Figure 2.

An echocardiogram at this time showed a dilated left ventricular (LV) with severely impaired LV systolic function and an ejection fraction of 20%. Right ventricular (RV) size and function were normal. On pacemaker interrogation next morning, the following parameters were detected:

- Identity, St Jude Medical pacemaker (with special atrial fibrillation [AF] suppression algorithm).
- The atrial overdrive-pacing algorithm i.e. the AF suppression algorithm was switched on.
- Atrial tracking upper rate was 150/min.
- Sensor-driven upper rate was 150/min.
- Automatic mode switching rate was 160/min.
- AV delay was rate responsive.

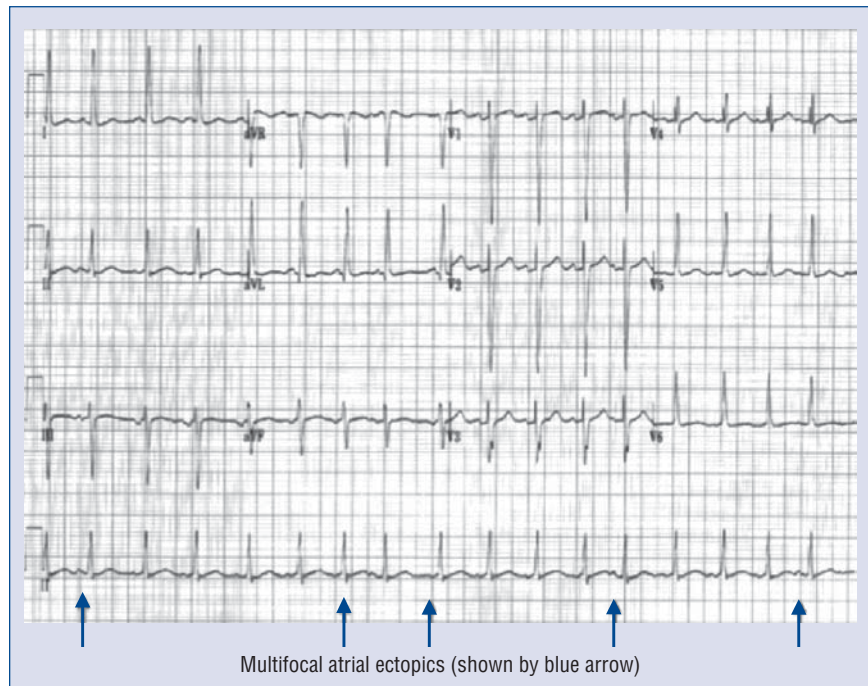


Figure 3. ECG after atrial fibrillation suppression algorithm was switched of.

The AF suppression algorithm was switched off. Following this, the rhythm reverted to a sinus rhythm with multifocal atrial ectopics (as shown in Fig. 3).

She subsequently improved and was discharged home a week later. Four months later, her echocardiogram showed mild to moderate left ventricular systolic function with an ejection fraction of 45%, and more importantly she was NYHA class I with regards to symptoms.

In summary, her pneumonia was causing an atrial tachycardia, which the pacemaker was overdriving due to this special AF suppression algorithm setting. This led to tachycardia-induced cardiomyopathy.

Discussion

This is a unique case of cardiomyopathy caused by the tachycardia (tachycardia-induced cardiomyopathy) from the atrial overdrive pacemaker.

The AF suppression algorithm is a special program designed to suppress AF by overdrive atrial pacing (i.e. at a rate just above the intrinsic sinus rate), while it continually monitors intrinsic atrial rate, increasing the stimulation rate when the intrinsic rate increases. When the AF suppression is programmed 'on' the paced rate will be higher than the patient's intrinsic sinus rate. If it senses a P-wave (or atrial ectopics), it begins a 16-cycle window,

looking for further sensed P-waves. This window expires with no further P-wave activity. If it senses the second P-wave within this window, the device responds by overdriving the rate (AF suppression will increase the rate on a sliding scale, from 10 bpm for paced rates below 60, to 5 bpm for paced rates above 150). The mechanism in frequent atrial ectopics can be explained by the following example (Fig. 4).

Initially, the ECG demonstrates dual-chamber pacing at a rate of 70 bpm. So the intrinsic atrial rate is less than the paced rate. The device senses five consecutive P-waves at a rate of 90 bpm (marked P in the Fig. 4) and responds by overdriving the rate in a stepwise manner. P-1 and P-2 are two P-waves sensed in a 16-cycle window. Hence it immediately starts overdriving with the second P-wave, increasing the rate from 70 bpm to 80 bpm. It resets the 'P' counter to zero, ends the window and begins looking for P-waves. P-3 starts another 16-cycle window, and P-4 is the second P-wave sensed in the window. This causes a further increase in the overdrive rate (from 80 bpm to 87 bpm), again resetting the 'P' counter to zero and ending the window. P-5 is another intrinsic P, which starts another 16-cycle window. This is similar to our patient's ECG taken just after removal of the magnet. Application of a magnet switches off this algorithm (the pacemaker loses its sensing power).

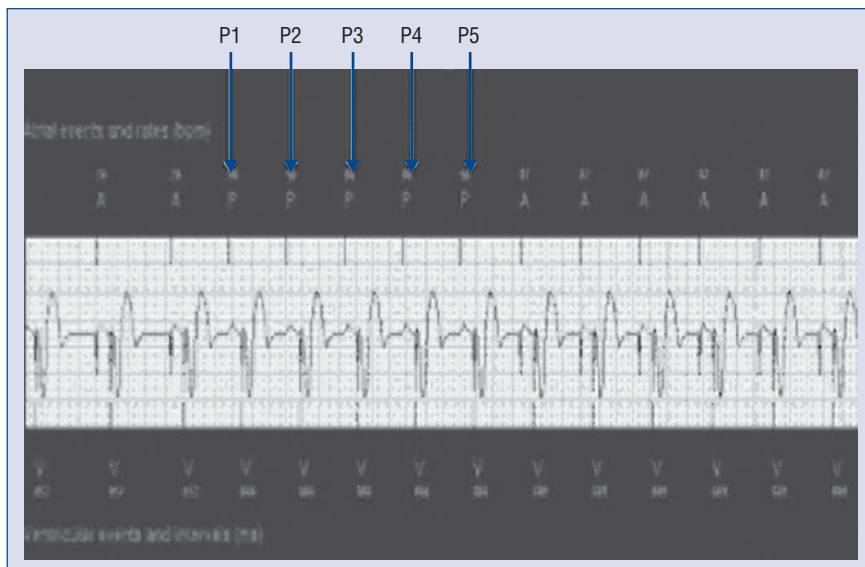


Figure 4. Rhythm strip showing the mechanism in frequent atrial ectopics.

Subsequent removal of the magnet turned the algorithm back on. At this point, the pacemaker started tracking the intrinsic rate (which was sinus tachycardia at a rate of 145 bpm), thereby increasing the paced atrial rate in a sliding manner until it was overdriving the intrinsic rhythm (at a paced rate of 150 bpm) as evident in the ECG 2. As her intrinsic atrial rate was not exceeding the maximum programmed atrial tachycardia detection rate (ATDR), the device did not switch mode i.e. did not turn the algorithm off. The mode switch may have helped her during the acute left ventricular failure event.

The initial narrow complex rhythm (140/min on Fig. 1) was due to fusion beats (ventricular beats superimposed on the paced beats). But the rhythm changed to broad complex (paced beats) due to the increase in the rate (150/min), which was preventing the fusion beats from occurring [1].

Learning points

1. While managing arrhythmia with an underlying pacemaker, always check the pacemaker

details from the notes and arrange for pacemaker interrogation.

2. As the AF suppression algorithm is not tolerated by every patient [2, 3], a baseline echocardiogram is recommended before this programmed mode is switched on.
3. Our case highlights the importance of a magnet as a therapeutic, as well as a diagnostic, tool.

Acknowledgements

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