

Tachycardia-induced cardiomyopathy long after a pacemaker implantation for the treatment of unusual 2:1 atrioventricular block: What is the mechanism?



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Introduction

Device interrogation sometime provides useful information about previous arrhythmic events. We describe a patient with atrioventricular (AV) block in whom a pacemaker interrogation found the unpredictable cause of pleural effusion.

Case summary

A 49-year-old man experienced syncope while he crossed a street, and he was transferred to our hospital. An electrocardiogram (ECG) showed a second-degree 2:1 atrioventricular block with narrow QRS complexes (Figure 1A). Curiously, the RR intervals were not constant while the PP intervals remained steady during the 2:1 AV block (Figure 1B). The echocardiogram revealed a normal left ventricular systolic function and no morphologic abnormalities. No significant coronary stenosis was observed on the coronary angiogram. No electrolyte abnormalities were noted. He did not have any relevant medical history and did not take any medications. Because the 2:1 AV block did not improve, a dual-chamber pacemaker (Advisa DR MRI A3DR01, Medtronic, Minneapolis, MN) was implanted.

The alternating RR intervals during the 2:1 AV block seen in the present case were uncommon. A quite similar ECG finding was however previously reported.¹ The authors offered the concealed conduction concept as the causal mechanism.² The term “concealed conduction” is used when a proximal atrial impulse penetrates the AV node but fails to

traverse it completely. The concealed penetration into the AV node is inferred by its aftereffects on the propagation of succeeding impulses. The authors specifically described the phenomenon as follows. When blocked atrial impulses penetrate deeper into the AV junction, the subsequent PR intervals are long, while the impulses blocked at its shallow level are followed by shorter PR intervals. Importantly, they stated that when 2 levels of concealment are postulated in an ECG showing normal QRS complexes, the conduction disturbance is most probably confined within the AV node. Thus, the interesting phenomenon we observed suggested that our patient may have had intranodal block rather than infranodal block. The former is generally considered more benign than the latter.³ The current guidelines,³ however, clearly state that permanent pacemaker implantation is indicated for second-degree AV block with associated symptomatic bradycardia, regardless of the site of the block. His pacemaker implantation was based on this guideline.

One year later, the patient saw his family physician for dyspnea on effort. A chest radiograph demonstrated a pleural effusion. He was prescribed 20 mg of furosemide. He had a quick improvement in his symptom, and the pleural effusion was no longer seen 2 weeks after his initial visit. The furosemide was then withdrawn. He never experienced the recurrence of the symptom. Another 8 months later, he was seen in our outpatient clinic for a regular pacemaker check.

There are a wide variety of diseases that can result in pleural effusion.⁴ However, given that only a brief administration of the oral diuretic cured his pleural effusion without recurrence, any chronic disease or disease resulting in irreversible damage was unlikely. Anyway, he had never presented with the symptom until he was implanted with the pacemaker. Therefore, it was essential to check for any pacemaker-related defects, such as pacemaker failure, lead perforations, or heart failure due to chronic right ventricular pacing.

The echocardiogram findings and lead positions remained the same, and no pacemaker failure was noted. Instead, a number of atrial fibrillation (AF) or atrial tachycardia (AT) events were recorded by the pacemaker (Figure 2A). During

KEYWORDS Atrioventricular block; Autonomic tone; Tachycardia-induced cardiomyopathy

ABBREVIATIONS AF = atrial fibrillation; AT = atrial tachycardia; AV = atrioventricular; ECG = electrocardiogram (Heart Rhythm Case Reports 2016;2:107–110)

Conflicts of interest: The authors report no conflict of interest. Funding: None declared. **Address reprint requests and correspondence:** Dr Akinori Sairaku, Department of Cardiovascular Medicine, Hiroshima University Graduate School of Biomedical and Health Sciences, 1-2-3 Kasumi, Minami-ku, Hiroshima 734-8551, Japan. E-mail address: rjrgw059@ybb.ne.jp.

KEY TEACHING POINTS

- Alternations of the ventricular cycle length could be observed even during 2:1 second-degree atrioventricular (AV) block, and this observation may be explained by different depths at which blocked impulses penetrate into the AV junction.
- Tachycardia-induced cardiomyopathy could be one of the causes of decompensated heart failure, even in patients with AV block needing a pacemaker.
- Interrogation of pacemakers often provides some clues about the cause of previous cardiac events.

the AT/AF, the pacemaker mode was automatically switched from DDD to DDIR. An extremely rapid ventricular response was noted during some AT/AF events, in particular soon after the beginning of the AT/AF events (Figure 2B). A cardiac compass report (Figure 2C) showed that the AT/AF persisted for about 6½ months and terminated spontaneously. A medical interview with the patient proved that his dyspnea coincided with the first occurrences of the AT/AF events. According to the records of the thoracic impedance in his pacemaker, it markedly decreased also soon after the beginning of the AT/AF events (Figure 2D).

A double-chamber tachycardia was carefully ruled out via device interrogation. It was therefore assumed that the patient developed tachycardia-induced cardiomyopathy secondary to a rapid AT/AF, resulting in acute decompensated heart failure.⁵ He obviously had symptomatic second-degree AV block requiring a pacemaker 20 months before the last clinical visit. Thus, the rapid ventricular response during the AT/AF was very unlikely if his AV conductivity remained severely impaired. Prior to the pacemaker implantation, we carefully excluded any potential reversible causes of AV block, such as drug toxicity, electrolyte abnormalities, diseases with periaortioventricular node inflammation, transient injury or ischemia of the conduction system, or hyper-vagotonia.³ It is known that AV block could be reversed transiently in patients with cardiac sarcoidosis.³ However, the findings from his multiple echocardiographic examinations, ECGs, and device interrogations never suggested cardiac sarcoidosis.⁶ Did his AV block then heal spontaneously?

The patient presented with sinus rhythm at 68 beats per minute and a prolonged PQ interval of 270 milliseconds when the sensed AV delay of the pacemaker was changed from 150 to 300 milliseconds. The Wenckebach cycle length was determined to be 720 milliseconds (Figure 3), and the AV nodal effective refractory period was found to be 620 milliseconds. He was prescribed 5 mg of bisoprolol.

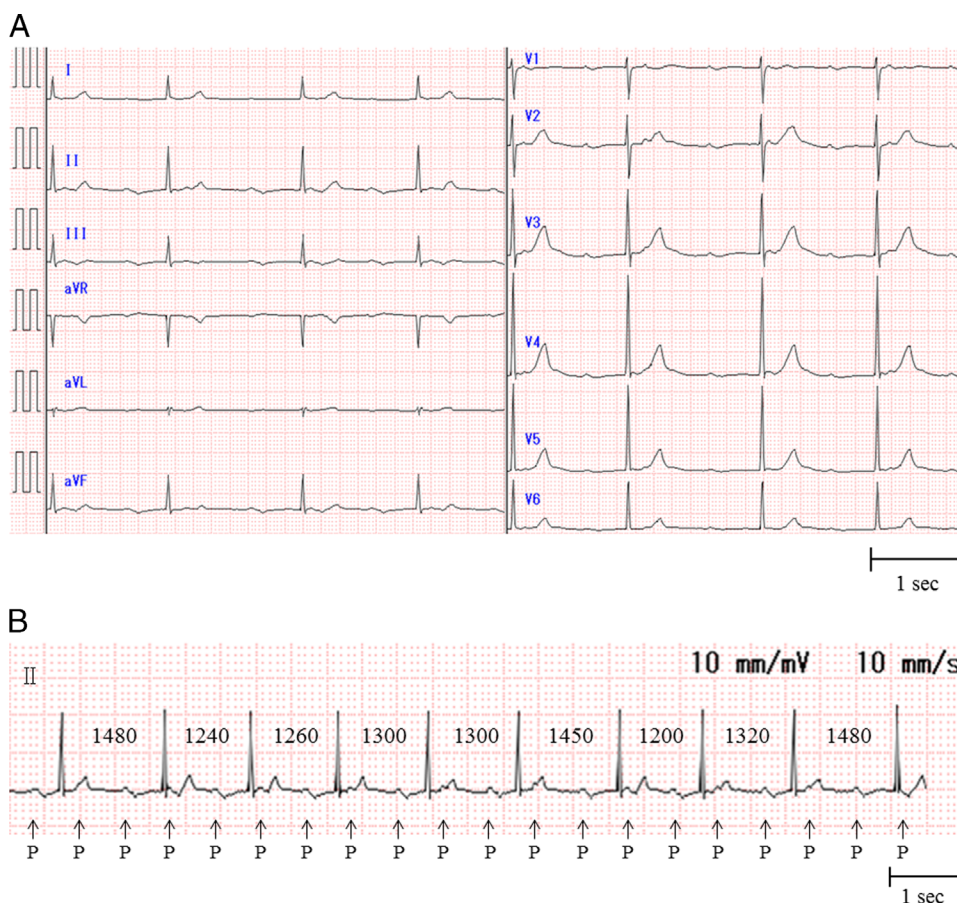


Figure 1 A: An electrocardiogram recorded at the patient's initial clinical visit. A 2:1 atrioventricular block was noted. B: A long-duration recording of an electrocardiogram at the initial clinical visit. The RR intervals were irregular during 2:1 atrioventricular block.

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