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Case Report

Pacemaker Mode Switching may Cause Potential Ventricular Tachycardia

LiBing Zhang[1,2], Zhi Zhao[1,2], Qi Jin[1,2], Wei Fang[1,2], Yuan Yang[3], YongNan Fu[1,2*]

[1]Department of Cardiology, Nanchang University, Nanchang, China; [2]Department of Cardiology, Jiangxi Hypertension Research Institute, Nanchang, China; [3]Department of Pharmacy, Nanchang University, Nanchang, China

ABSTRACT

Patients with SSS are usually treated with a permanent pacemaker to control and improve symptoms. Here, we report a case of an implanted dual-chamber pacemaker with an automatic switching mode function due to Sick Sinus Syndrome (SSS) 8 years ago. The patient was admitted again because of Heart Failure (HF) symptoms. AT and hypokalemia occurred after admission, followed by Ventricular Tachycardia (VT). The prolongation of the QT interval caused by the change of pacemaker mode might be involved in the occurrence of VT. After emergency cardioversion, increasing ventricular pacing frequency, and correction of the hypokalemia, VT was well controlled and the patient was discharged from the hospital.

Keywords: Ventricular tachycardia; Pacemaker mode switching

Abbrevations: ECG: Electrocardiograph; VT: Ventricular Tachycardia; LVEF: Left Ventricular Ejection Fraction; AT: Atrial Tachycardia; HF: Heart Failure; SSS: Sick Sinus Syndrome

INTRODUCTION

The Automatic Switching Mode (AMS) of the programmable function chambers of most biventricular pacemakers nowadays. Atrial tachyarrhythmias are detected when the sensed atrial rate exceeds a "rate-cutoff," "running average," "sensor-based physiological" rate, or using "complex" detection algorithms [1]. The results of the AMS algorithm can vary depending on sensitivity and specificity. As a result, responses to atrial tachycardia differ in terms of the speed of AMS onset, the rate stability of the response, and the speed of resynchronization with sinus rhythm. Because the ventricular response of DDD pacemakers is dependent on the atrial rate, rapid ventricular pacing can occur with DDD pacemakers during episodes of Atrial Tachycardias (ATs), especially during Atrial Fibrillation (AF). Therefore, AMS changes the pacemaker mode based on an algorithm, and pacemaker syndrome occurs when there is a loss of atrial synchronization to ventricular pacing and intolerance to ventricular pacing [2]. We report a case of VT due to pacemaker mode switching.

CASE PRESENTATION

Case report headings

Pacemaker mode switching may cause potential ventricular tachycardia.

History of presentation

A 74-year-old female patient was admitted to the hospital 8 years ago due to chest tightness and syncope. And implanted a permanent dual-chamber pacemaker with an automatic switching mode function (the pacing mode is DDD) (Figure 1). The patient started showing symptoms of pacemaker-mediated heart failure 2 years ago. Recently, the patient was admitted to the hospital due to worsening symptoms of heart failure.

Correspondence to: YongNan Fu, Department of Cardiology, Nanchang University, Nanchang, China, E-mail: fyn1028@126.com

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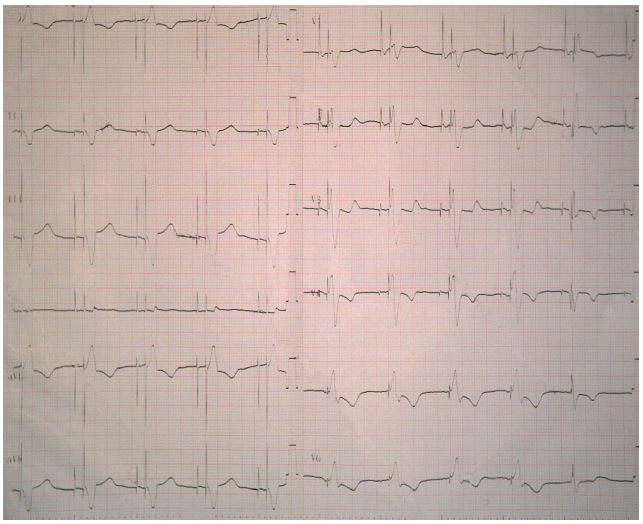

Figure 1: Baseline 12-Lead ECG after pacemaker surgery.

Past Medical History: Medical history of hypertension, and diabetes.

Differential diagnosis: 1) Rheumatic heart disease; 2) Dilated heart disease.

Investigations: Complete ECG examination after admission to our hospital showed that: Sinus tachycardia, pacemaker in VAT mode, QT/QTc interval 342/458 ms, and then AT led to pacemaker mode switching, changed to VVI pacing mode, QT interval prolongation, QT/QTc interval 542/546 ms (Figure 2) and ventricular tachycardia (Figure 3). Laboratory tests suggest hypokalemia.

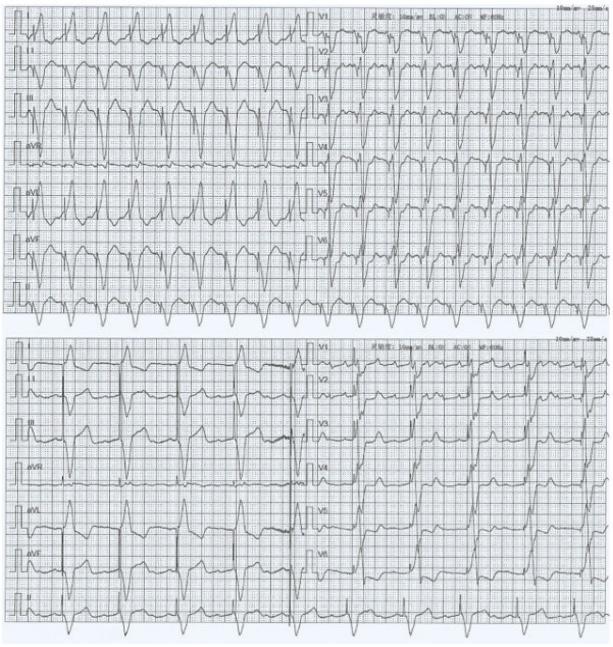

Figure 2: 12-Lead ECG before and after pacemaker mode switching. **Note:** Before pacemaker mode switching, 12-lead ECG showed: Sinus tachycardia, pacemaker in VAT mode, atrial sensing, ventricular pacing mode, QT/QTc interval 342/458 ms.

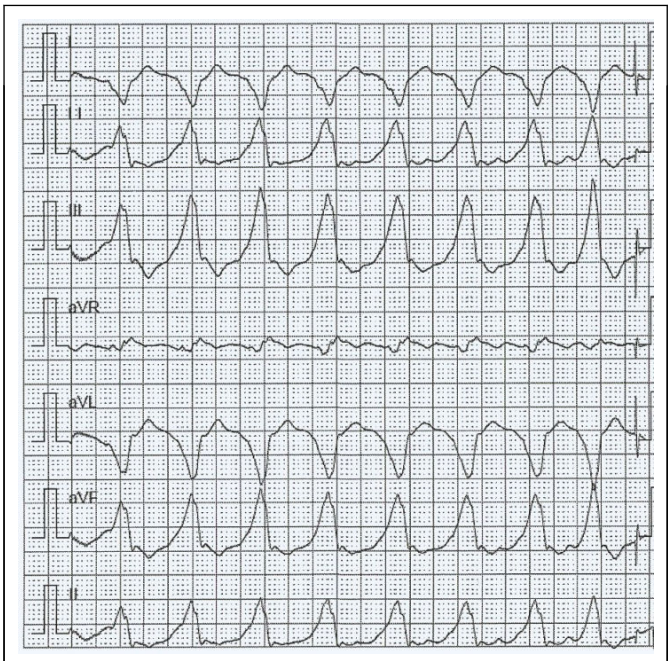

Figure 3: Electrocardiogram record depicting ventricular tachycardia.

Management (medical/interventions): During hospitalization, After emergency cardioversion, the patient was supplemented with potassium and magnesium, and the pacemaker frequency was increased to 110 beats/min, VT was well controlled. Echocardiography showed an LVEF of 45%.

RESULTS AND DISCUSSION

Potential VT from dual-chamber pacemaker mode switching is extremely rare. The automatic switching of the working mode of the pacemaker means that when the atrial rate is higher than the frequency set by the atrial sensing electrode of the pacemaker, the pacemaker automatically turns off the atrial sensing and switches to VVI mode, thereby avoiding the occurrence of potentially dangerous rapid ventricular pacing [1]. Currently, electrodes used for ventricular pacing are typically placed at the right ventricular apex or right ventricular septum. However, both right ventricular apical pacing and right ventricular septal pacing differ from the normal ventricular activation sequence. Studies find that right ventricular pacing is associated with higher rates of atrial fibrillation, HF, and mortality [3-7]. Results from the MOST trial showed a 2.6-fold increased risk of HF hospitalization in patients with normal baseline QRS duration when the right ventricular pacing rhythm accounted for more than 40% of total heart rate [5]. Meanwhile, the Dual Chamber and VVI Implantable Defibrillator (DAVID) trial showed that the risk of hospitalization or death was significantly increased when the right ventricular pacing rhythm accounted for more than 40% of the total heart rate in patients with left ventricular systolic dysfunction [7]. In this case, the patient was implanted with a dual-chamber pacemaker due to sick sinus syndrome with normal cardiac function, normal pre-implantation QRS complexes, and a postoperative ventricular pacing rate of 80%-100%. After 6 years, the patient began to develop pacemaker-mediated HF with symptoms and signs of HF, such

as chest tightness, shortness of breath, and edema of both lower extremities. Patients with increased atrial pressure and atrial enlargement are susceptible to AT due to pacemaker-mediated HF, leading to dual-chamber pacemaker mode switching [8-11]. During the course of symptoms of HF in the above case, the patient developed underlying electrolyte disturbances and hypokalemia with continued diuretics. Coupled with AT, the dual-chamber pacemaker mode is switched from DDD to VVI, resulting in slow heart rate and QT prolongation. These two factors simultaneously lead to VT.

Follow-up

After the patient's condition was stable, the patient underwent an upgrade from a dual chamber pacemaker to CRT-D. The patient's heart failure symptoms improved, and there was no VT During the follow-up.

CONCLUSION

Pacemaker syndrome is defined as intolerance to ventricular-based (VVIR) pacing due to loss of Atrioventricular (AV) synchrony. Although there is no universal agreement on the definition or diagnostic criteria of pacemaker syndrome, symptoms include dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, orthostatic hypotension, and even syncope.In the new pacing mode, the ventricular pacing electrodes are placed in the region of the His bundle or the left bundle branch, as opposed to the traditional ventricular pacing electrodes placed in the right ventricular apex or right ventricular septum. Due to the high technical requirements of His bundle pacing, the low technical difficulty of pacing in the left bundle branch region, and the stable pacing threshold, it may become the future development direction of His-Purkinje system pacing. Patients with a high proportion of ventricular pacing often lead to pacemaker-mediated heart failure, severe pacemaker syndrome developed in nearly 20% of VVIR-paced patients and improved with reprogramming to the dual-chamber pacing mode. Because prediction of pacemaker syndrome is difficult, the only way to prevent pacemaker syndrome is to implant atrial-based pacemakers in all patients. Most notably, quality of life, as assessed by a variety of metrics, decreased at the time of diagnosis of pacemaker syndrome and improved after the pacemaker was reprogrammed to a physiologic mode. So physiological pacing is best. Patients with dual-chamber pacemakers, especially those with cardiac insufficiency with apical pacemakers, should pay close attention to the pacing mode of the patient, follow up closely, and detect pacemaker mode conversion as soon as possible to prevent possible complications caused by pacemaker mode conversion. Damage to cardiac function and prevent the occurrence of VT. When this syndrome is suspected, careful evaluation is mandatory because atrial-based, physiologic pacing may markedly improve physical function and quality of life. In this csae, the only deficiency was the patient's low potassium. Low blood potassium may be a cause of VT, but the pacemaker pattern switch was higher than before potassium supplementation, so this is also interesting in this case.

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