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Case Report

Right ventricular perforation in a patient with recurrent right coronary artery occlusions

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Abstract: Ventricular perforation is a rare complication of pacemaker implantation. A thinner muscle wall after myocardial infarction is one of the risky factors for ventricular perforation. This report will introduce a patient who has been suffered three myocardial infarctions during the past five years and right ventricular perforation, which was successfully managed with percutaneous lead removal. This case illustrates the importance of anti-platelet therapy in patients with recurrent coronary artery occlusions and the necessity for us to be alert to the possibility of right ventricular perforations in patients with isocheimal right ventricular cardiomyopathy.

Keywords: Pacemaker, myocardial perforation, myocardial infraction, active fixation lead

Introduction

Ventricular perforation, a rare complication of permanent pacemaker implantation, for which a thinner muscle wall after myocardial infarction is one of the risky factors for ventricular perforation [1]. A single-center study reported results on timing of delayed perforation with the St. Jude Riata lead. There were 8 cases of lead perforation of a total of 416 implanted Riata leads. Seven patients underwent successful lead revision in the electrophysiology laboratory; one of the patients developed an effusion that required pericardiocentesis when the perforated lead was pulled out of the pericardium and repositioned [2]. This article will take a rare case of a male patient presents with three myocardial infarctions and right ventricular (RV) lead perforation as an example.

Case report

A 71-year-old male patient, who had suffered from chest pain 6 hours, presented to our department on November 30, 2012. He has had hypertension and cerebral embolism for one year. Vital signs showed heart rate of 46 bpm, BP of 94/62 mmHg, and respiratory rate of 22 breaths per minute. Aside from that, his lungs were clear; heart sounds were irregular without any murmurs. Electrocardiography (ECG) showed complete aterioventricular block with inferior wall myocardial infraction (IWMI) and right ventricular myocardial infraction (RVMI) (**Figure 1**). The patient was given a loading dose of 300 mg aspirin and 600 mg clopidogrel. A temporary pacemaker was implanted in the right ventricle because of the minimal heart rate was 34 bpm. Angioplasty and aspiration thrombectomy were performed (**Figure 2A**, **2B**). In addition, the laboratory results later revealed an initial serum troponin I of 8.6 ng/ml (normal < 0.03 ng/ml) and activated partial thromboplastin time of 41.0 s (normal 31~43 s). On the seventh day, coronary angiography revealed no evidence of coronary stenosis in the right coronary artery (RCA) (**Figure 2C**, **2D** and Video S1). The sinus rhythm was restored and the temporary pacemaker electrode was removed. The patient was discharged with prescriptions for aspirin 100 mg daily, clopidogrel 75 mg daily and simvastatin 10 mg daily.

On April 11, 2014, the patient returned to our department with sudden onset of severe back pain. Due to the lack of insurance, he has run out of medication for 3 months. By that time his blood pressure was 120/80 mmHg, heart rate was 80 bpm, and cardiac auscultation was normal. The bedside ECG indicated sinus rhythm

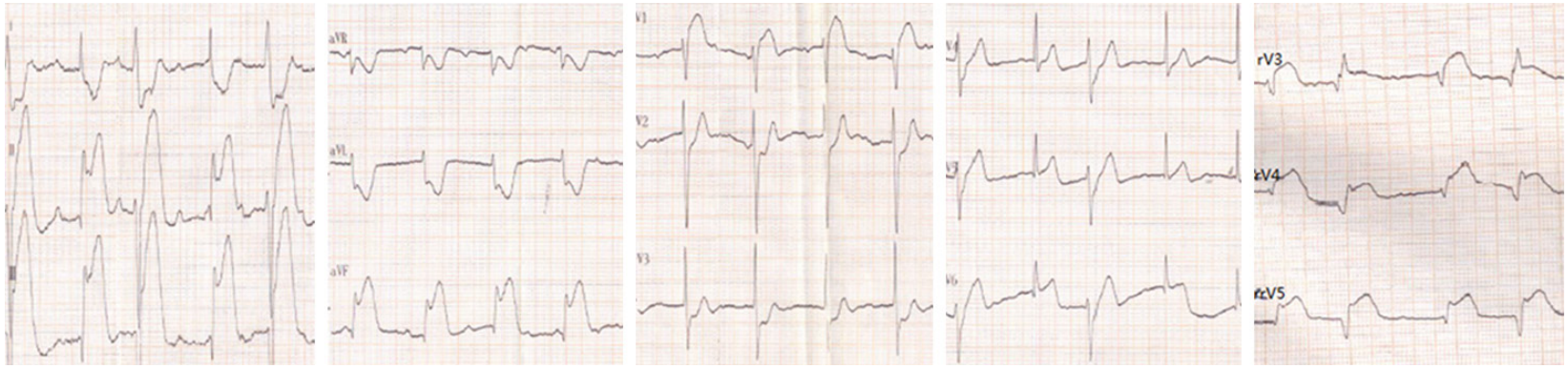

Figure 1. ECG showed complete aterioventricular block with IWMI and RVMI for the first time in the hospital.

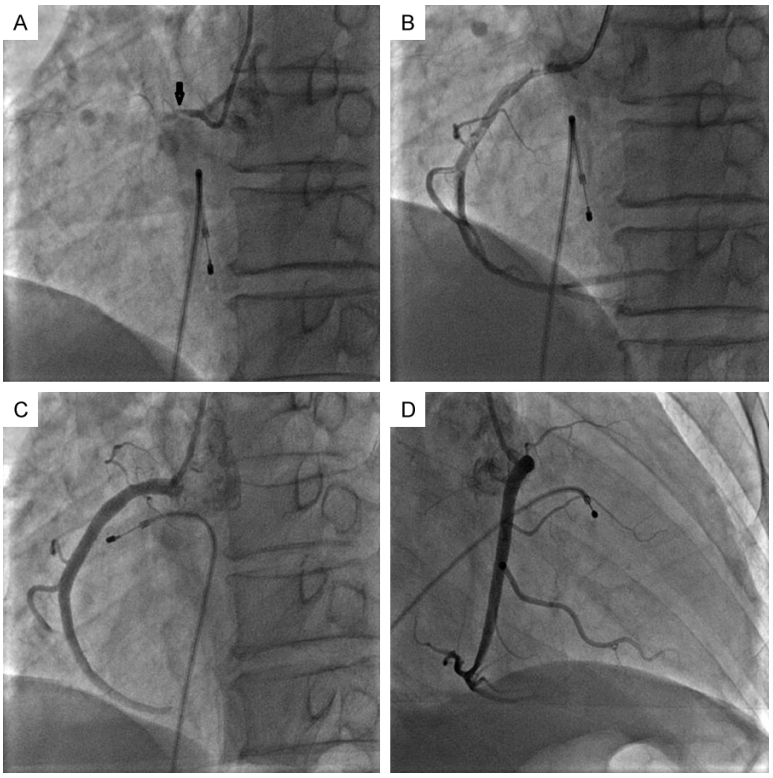

Figure 2. The results of coronary angiography for the first time in the hospital. A. Coronary angiography revealed an acute total occlusion (arrow) of the proximal right coronary artery. B. Aspirate thrombus was performed in the RCA. C and D. After antithrombotic therapy there was no evidence of coronary stenosis.

with IWMI and RVMI (**Figure 3**). To rule out the possibility of aortic dissection, CT was performed. The result showed a large thrombus in the aortic root, and a long thrombus from the distal right iliac artery to the proximal right internal iliac artery. The laboratory results revealed serum troponin I of 6.9 ng/ml and activated partial thromboplastin time of 42.7 s. The treatment started with aspirin 300 mg, clopidogrel 600 mg, and low molecular weight heparin calcium 6000 u. Ten days later, there was no thrombus in the aortic root, the distal right iliac artery or the proximal right internal iliac artery. However, reocclusion of the RCA was noted in the catheterization laboratory (**Figure 4A**). Under the support of a temporary RV pacing catheter, aspiration thrombectomy was performed (**Figure 4B**). Despite both coagulation tests results were in the normal range, hypercoagulable state of prethrombotic state was diagnosed, and lifelong anticoagulation was recommended.

On July 8, 2017, the patient was transferred to our department for pacemaker implantation for sick sinus syndrome. Two months before, the patient had syncope and low heart rate. For the past 6 months, the patient had been noncompliant with all of his medications. Holter monitor underwent in a local hospital showed slow heart (HR_{min} 24 bpm, average 50 bpm, RR_{max} 4S) with normal AV conduction. Physical examination was unremarkable only for enlargement of heart. Full blood and coagulation screens were normal. A tranthoracic echocardiogram revealed enlargement of the RV (diameter 40 mm) and dysfunction of RV. The patient received a dual chamber pacemaker (ZEPHYR™ XL DR 5826, St. Jude Medical, St. Paul, MN, USA) with two active fixation leads (TENDRIL™ STS 2088TC-52, and TENDRIL™STS 2088TC-65, St. Jude Medical, St. Paul, MN, USA). A cardiac catheterization, which documented chronic total occlusion of RCA (**Figure 5A**), was also performed. Unfortunately, the patient refused to accept percutaneous coronary intervention. On the second day, the patient pointed out severe pain above the left rib arch. The chest X-ray revealed pacemaker leads in proper position (**Figure 5B**) and there was no pericardial effusion on echocardiography. His chest pain reduced gradually by diclofenac sodium. However, on the fourth day the patient complained of left chest pain and chest tightness, especially when he was lying down. The CT showed the development of a large left pleural effusion and displacement of RV lead, but the tip position could not be confirmed because of artifacts (**Figure 5C**, **5D**). A left chest tube was placed, with a return of 1000 ml of sanguineous fluid, and RV perforation was confirmed by the CT (**Figure 6A**). Hence a relative small thoracotomy was performed, which showed the ventricular pacemaker lead had perforated the RV free wall (**Figure 6B**). The RV lead was removed and the ventriculotomy was repaired. Due to normal AV conduction, the pacemaker was programmed to AAI mode. The patient's postoperative course was good

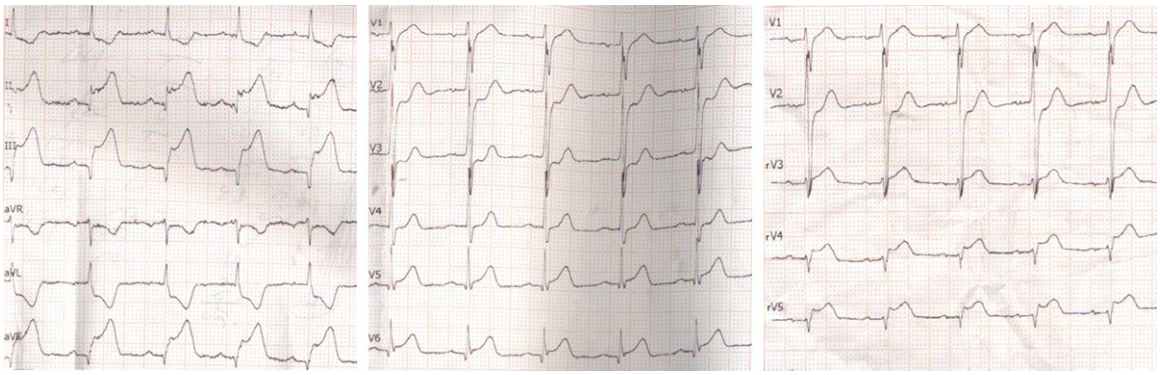

Figure 3. The bedside ECG indicated sinus rhythm with IWMI and RVMI for the second time in the hospital.

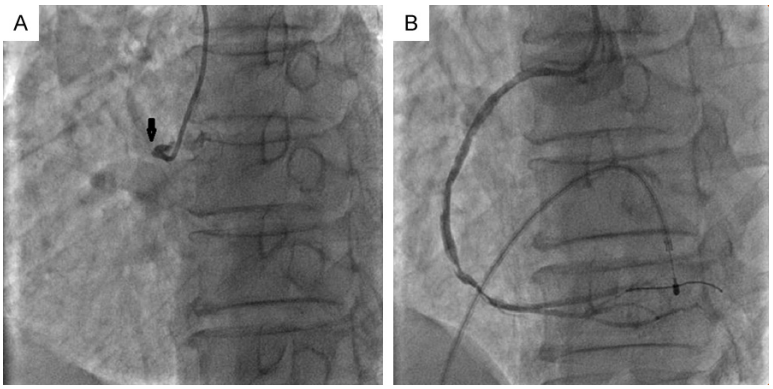

Figure 4. The results of coronary angiography for the second time in the hospital. A. Reocclusion of the right coronary artery (arrow) was again noted in the catheterization laboratory after the patient ran out of medication for 3 months. B. Aspirate thrombus was performed similar to the first hospitalization.

and he was discharged on the 9th postoperative day.

Discussion

Although most acute coronary syndromes are caused by atherothrombosis, they may still occur in patients with coronary arteries that appear normal in angiography [3]. Coronary artery spasm, coronary embolism, and hypercoagulating states have been described as inducement of myocardial infarction (MI) in patients without evidence of coronary stenosis [4]. Recurrent MI and angiographically normal coronary arteries are always a challenge due to unclear pathophysiology, management and prognosis. In this case, the initial diagnosis was MI, and the coronary angiography showed no significant coronary stenosis. During the first hospitalization, the patient received aspiration and antithrombotic therapy, and thromboses in RCA were completely removed. The results showed that thrombus was the cause of acute myocardial infarction. The patient has been suffered recurrent attacks of ST-segment elevation myocardial infarction due to withdrawal of antithrombotic therapy for three months. Thrombus were not only found in the coronary arteries, but also in central artery. With the treatment of dual antithrombotic and aspiration thrombectomy, thrombus disappeared completely. The results further prove the above conclusion. In addition, the patient has a history of cerebral embolism. Despite both coagulation tests results were in the normal range, we speculate that the patient may be hypercoagulable state of prethrombotic state, lifelong anticoagulation was recommended.

The major risky factors for cardiac perforation are age, female sex, body mass index below 20, use of anticoagulants or steroids, and use of leads with an extendable fixation lead [5]. In this case, another risk is thin myocardial wall secondary to isocheimal RV. Because of the enlargement of RV, echocardiography can't be an accurate diagnostic procedure in suspicious perforation. As in this case, repeated CT can be useful and efficient diagnostic procedure in suspicious perforation. It provides accurate information on electrode position and its relation to other tissues and organs, which can be useful for further therapeutically approach [6]. The treatment options and replacement of electrodes are similar in many medical centers, but for the safety reasons these procedures should

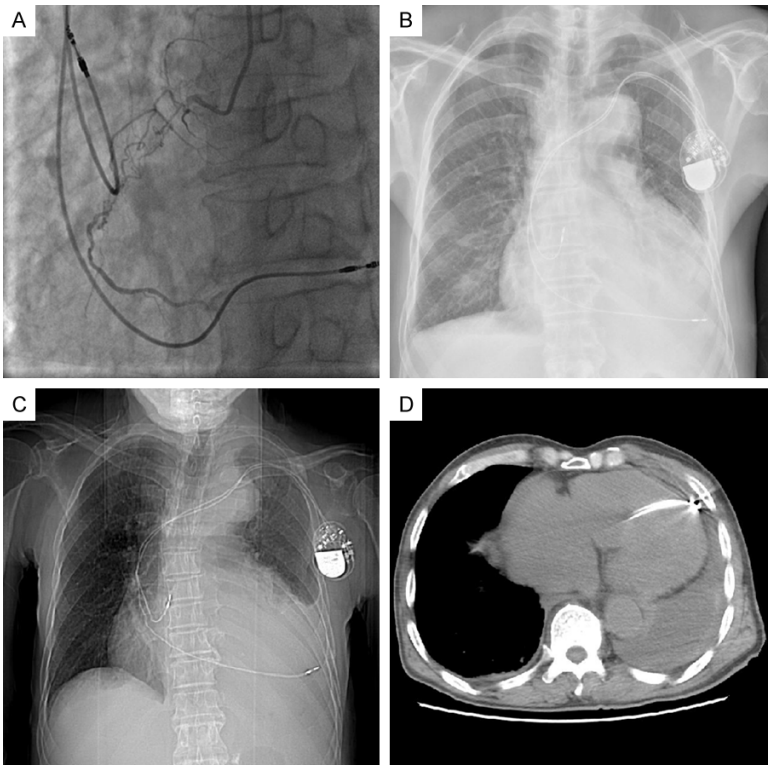

Figure 5. The results of imaging examination for the third time in the hospital. A. A cardiac catheterization documented chronic total occlusion of RCA after the pacemaker implantation. B. Chest X-ray revealed pacemaker leads in proper position on the second day after the implantation. C and D. On the seventh day after the implantation, a chest computed tomography showed the development of a large left pleural effusion and displacement of right ventricular lead.

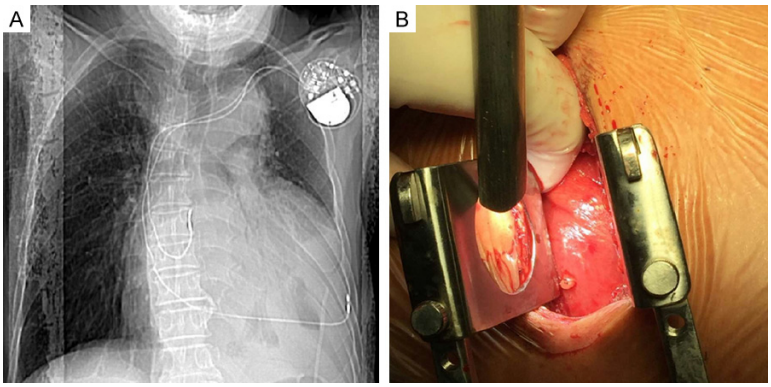

Figure 6. Perforation of the right ventricle was confirmed by the chest computed tomography and left anterolateral thoracotomy. A. The CT showed the right ventricle lead tip extending beyond the cardiac silhouette. B. The ventricular pacemaker lead had perforated the right ventricular free wall.

be done at equipped centers with cardiovascular surgery units [7].

In summary, this case illustrates the importance of anti-platelet therapy in patients with recurrent coronary artery occlusions and the necessity for us to be alert to the possibility of right ventricular perforations in patients with isocheimal right ventricular cardiomyopathy.

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Disclosure of conflict of interest

None.

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