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Journal of Cardiology Cases

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Case Report

Pacemaker malfunction after acute myocardial infarction in a patient with wrap-around left anterior descending artery supplying the right ventricular apex

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ARTICLE INFO

Article history:

Received 26 September 2017
Received in revised form 13 February 2018
Accepted 3 March 2018

Keywords:

Myocardial infarction
Wrap-around left anterior descending artery
Pacemaker malfunction
Myocardial scintigraphy

ABSTRACT

A 50-year-old man with a dual-chamber pacemaker was admitted to our hospital complaining of chest pain. Anterior ST segment elevation myocardial infarction (STEMI) was diagnosed. Emergency coronary angiography revealed total occlusion of the proximal left anterior descending artery (LAD), and primary percutaneous coronary intervention was performed. Angiograms showed that the LAD was wrapped around the apex of both ventricles. On day 8, ventricular fibrillation and cardiopulmonary arrest occurred due to elevation of the pacing threshold because of pacemaker malfunction. The pacemaker was upgraded to an implantable cardioverter-defibrillator and the lead was inserted into the right ventricular septum. Myocardial scintigraphy with thallium-201 and technetium-99m pyrophosphate located the infarct zone around the apex of both ventricles.

We conclude that pacing failure of the right ventricular lead occurred in this case of LAD occlusion due to a LAD supplying the right ventricular apex. Clinicians should be aware of the possibility of pacemaker failure in patients presenting with anterior STEMI due to a wrap-around LAD.

<Learning objective: Pacemaker failure due to right ventricular lead malfunction can also occur in patients presenting with myocardial infarction of the left coronary arteries. In our case the culprit lesion was located in the left anterior descending artery, which wrapped around the apex to supply parts of the right ventricle.>

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Introduction

The left anterior descending coronary artery (LAD) does not usually supply the apex of the right ventricle [1,2], which generally receives blood from the right coronary artery (RCA). Myocardial infarction is one of the causes of pacemaker malfunction [3], and there have been several reports of pacemaker malfunction after infarction involving the RCA [4–6]. We report a rare case of pacing failure after acute myocardial infarction (AMI) in a patient with a wrap-around LAD that supplied the right ventricular (RV) apex.

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<https://doi.org/10.1016/j.jccase.2018.03.001>

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Case report

A 50-year-old man was admitted to our hospital complaining of chest pain. His past history included third-degree atrioventricular block, chronic atrial fibrillation, hypertension, diabetes, and chronic kidney disease. A dual-chamber pacemaker (Zephyr™XL DR; St. Jude Medical, St. Paul, MN, USA) had been implanted five years earlier and had been programmed for VVI mode (because of chronic atrial fibrillation after) at a basal rate of 80 beats per minute (bpm) and hysteresis of 50 bpm.

On admission, his heart rate was 64 bpm and blood pressure was 160/90 mmHg. The electrocardiogram showed ST segment elevation in leads V2–4 and ST segment depression in leads II, III, and aV_F. Transthoracic echocardiography revealed hypokinesia of the anterior wall and apical region. Laboratory tests showed elevation of creatinine kinase (CK) to 457 IU/L and troponin I was positive.

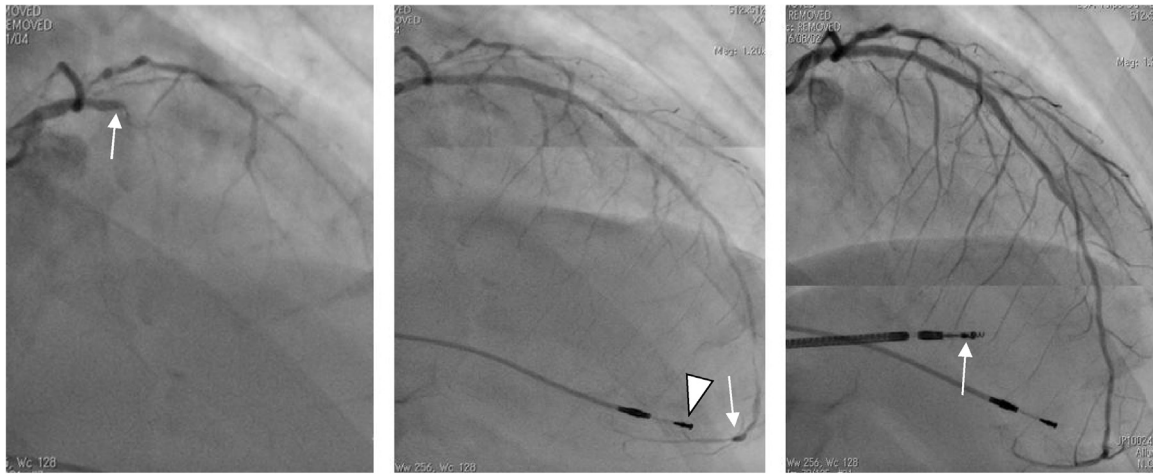


Fig. 1.

Coronary angiography and pacing leads. (A) Coronary angiography demonstrated total occlusion of the proximal left anterior descending artery (LAD) (arrow) and 75% stenosis of the left circumflex artery (LCX). (B) Percutaneous coronary intervention including drug-eluting stent placement in the proximal LAD was performed. The LAD wrapped around the apex of the heart (arrow). The pacemaker lead had been inserted at the right ventricular (RV) apex (arrowhead). (C) The pacemaker was upgraded to an implantable cardioverter-defibrillator (ICD) and the ICD lead was inserted in the RV septum (arrow) because of the high pacing threshold at the RV apex.

Emergency coronary angiography showed total occlusion of the proximal LAD and 75% stenosis of the left circumflex artery (LCX) (Fig. 1A), with grade 3 collaterals from the RCA. The patient underwent primary percutaneous coronary intervention with deployment of a drug-eluting stent across the culprit lesion in the LAD. Angiograms showed that the LAD wrapped around the apex of the heart (Fig. 1B). After admission to the intensive care unit, we confirmed that his pacemaker was functioning correctly (pacing threshold of 1.0 V @ 0.4 ms, sensing threshold of 8.0–9.2 mV, and lead resistance of 581 Ω). In addition CK showed the highest value (1736 IU/L) on day 2.

Initially, the patient's clinical course was stable, however, on day 7 a nonsustained ventricular tachycardia of 30 s was recorded. On the following day, ventricular fibrillation (VF) and subsequent cardiopulmonary arrest occurred. Cardiopulmonary resuscitation (CPR) was performed immediately, including multiple defibrillations. After return of spontaneous circulation (ROSC), the patient was placed under general anesthesia in case of recurrent VF. An electrocardiogram showed bradycardia and bigeminal premature

ventricular contractions with a tendency for progression to VF (Fig. 2). Pacemaker malfunction was suspected and confirmed by a pacemaker check showing an increase in pacing threshold to 5.5 V, despite a normal sensing threshold (11.6–12.0 mV) and lead resistance (724 Ω). Premature ventricular contractions subsided after the pacing rate was increased to 100 bpm and the output to 7.5 V. On day 9, the patient underwent secondary percutaneous intervention with deployment of a drug-eluting stent in the LCX. Insertion of a temporary pacemaker was attempted, but failed because of a high voltage threshold at the RV apex.

Considering the high risk of recurrent VF, the pacemaker was upgraded to an implantable cardioverter-defibrillator (ICD) (Ellipse™DR, St. Jude Medical) on day 18. The ICD lead was placed at the RV septum (Fig. 1C) (pacing threshold of 0.5 V @ 0.5 ms, sensing threshold of 0.5 mV, and lead resistance of 390 Ω). No further complications occurred and the patient could be discharged on day 35.

After discharge, myocardial scintigraphy was performed with thallium-201 (^{201}Tl) and technetium-99m pyrophosphate

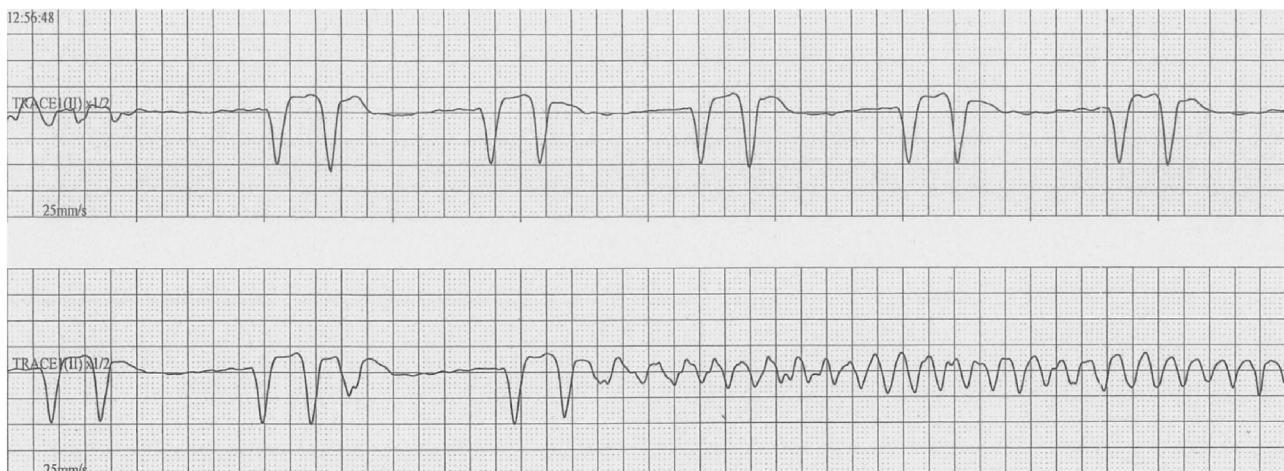


Fig. 2. Electrocardiogram on day 8. Electrocardiogram showed the onset of ventricular fibrillation following bradycardia and bigeminal premature ventricular contractions.

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