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CASE REPORT

INTERMEDIATE

CLINICAL CASE: SURGERY AND INTERVENTIONS

Loss of Right Atrial Pacing Lead Capture Due to Myocardial Infarction Obscuring Diagnosis on Electrocardiogram

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ABSTRACT

Right coronary artery occlusion can lead to failure to capture from the right atrial pacing lead. In this case, acute infarction resulted in failure of the right atrial lead to capture and thus increased right ventricular pacing. The new ventricular pacing masked the diagnosis of acute myocardial infarction. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:890–894) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Atrial infarction is an uncommon and often unrecognized consequence of acute coronary artery occlusion whose clinical significance is unclear.[1] The blood supply to the right and left atria arises from the right coronary artery (RCA) and left circumflex artery with significant variability and redundancy in distribution.[2-4] Right atrial (RA) lead failure after proximal RCA myocardial infarction (MI) has been described, but data are limited.[5,6]

We present a case of RA lead failure after late-presentation MI caused by complete occlusion of the RCA where the diagnosis was obscured owing to new ventricular pacing. Despite revascularization, loss of RA capture persisted, and the patient underwent implantation of a new RA lead. During lead revision, the RA septum was the only viable location for implantation of the new lead.

LEARNING OBJECTIVES

- To recognize pacemaker malfunction as a possible sequela of myocardial infarction.
- To recognize that ventricular pacing can mask electrocardiographic changes indicating myocardial infarction.
- To gain appreciation for the distribution of the atrial coronary artery blood supply.

CASE PRESENTATION

A 73-year-old man with a medical history of sinus node dysfunction with a dual-chamber pacemaker, paroxysmal atrial fibrillation on oral anticoagulation, cerebrovascular accident, hypertension, dyslipidemia, and type II diabetes mellitus presented to our hospital with persistent chest pain, diaphoresis,

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nausea, and shortness of breath, which had begun 3 days before admission.

In the emergency department the patient's presenting rhythm was right ventricular (RV) paced with retrograde atrial activation and capture failure from the atrial lead, although at the time of initial evaluation the lack of atrial capture was not appreciated (Figure 1). In comparison with his previous electrocardiogram (ECG), the RV pacing was new. Initial blood studies revealed a troponin T of 5.620 ng/mL (reference range 0.000-0.030 ng/mL). A chest x-ray revealed stable pacemaker lead positions. A limited echocardiogram showed preserved left ventricular systolic function with no appreciable wall motion abnormality and RV hypokinesis.

The patient's pacemaker had been implanted 7 years previously for symptomatic sinus bradycardia, after which those symptoms resolved with atrial rate-responsive pacing. At his most recent routine follow-up evaluation 5 months before his MI, he was pacing 97% in the RA and minimally in the RV, with stable device lead sensing, impedances, and thresholds.

As a result of his ongoing symptoms and concern for acute RV infarction, the patient was taken urgently for coronary angiography, where he was found to have complete occlusion of the proximal RCA (Figure 2) and moderate stenosis in the mid left anterior descending artery. A drug-eluting stent was placed to the proximal RCA with immediate restoration of flow and excellent angiographic results. After restoration of blood flow, an atrial branch arising off the proximal RCA was observed (Figure 3). The patient's symptoms improved after coronary revascularization, but capture failure of the RA lead still persisted.

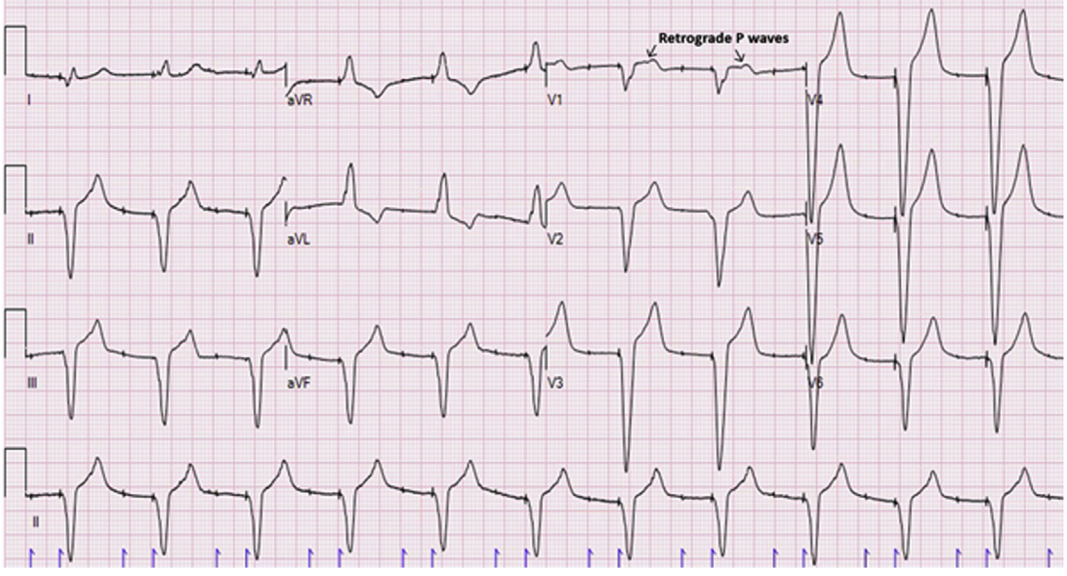
After revascularization, ventricular pacing persisted, and interrogation of the device revealed atrial non-capture despite maximal output pacing (5 V @ 2.0 ms). Pacing inhibition revealed an underlying low atrial or junctional escape rhythm with no intrinsic atrial signal sensed on the RA lead. Impedance measurement of the RA lead was stable compared with sustained measurements at 616 ohms. The RV lead parameters were normal and unchanged.

In light of the new drug-eluting stent with dual antiplatelet therapy in addition to sustained oral anticoagulation, monitoring for recovery was initially planned; however, he manifested significant dyspnea with RV pacing or intrinsic junctional

ABBREVIATIONS AND ACRONYMS

MI = myocardial infarction
RA = right atrium
RCA = right coronary artery
RV = right ventricle

FIGURE 1 Electrocardiogram at Presentation



Electrocardiogram showing right ventricular apex pacing, retrograde atrial activation, and capture failure from the right atrial pacing lead.

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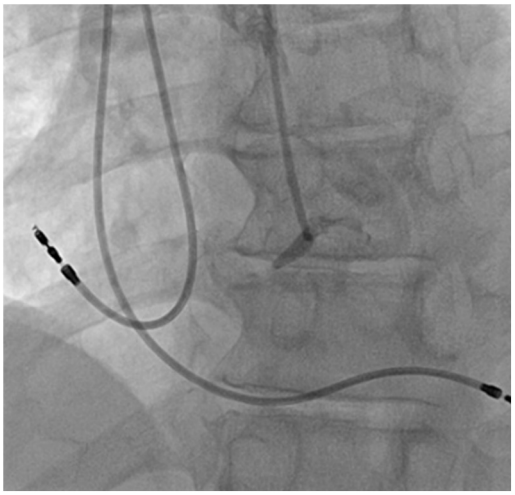
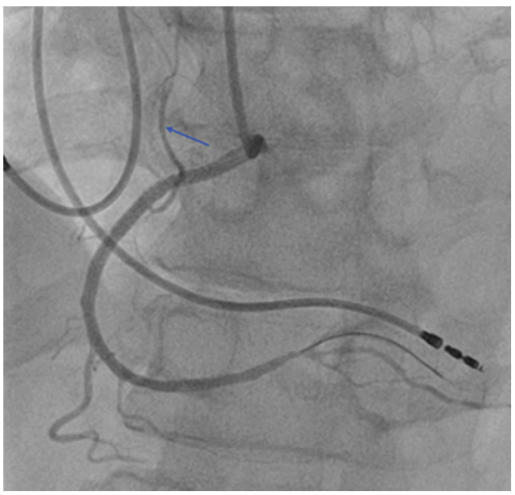
FIGURE 2 Initial Coronary Angiography



Angiography view showing complete occlusion of the proximal right coronary artery.

bradycardia, and therefore system revision was pursued on day 3 after the percutaneous coronary intervention. A new active-fixation bipolar pacing lead was advanced into the RA, and multiple RA sites were evaluated for sensing and capture.

FIGURE 3 Post-PCI Coronary Angiography



Angiography view after deployment of drug-eluting stent and restoration of flow. A large atrial branch **(blue arrow)** is seen arising from the proximal right coronary artery, distal to the location of the previous obstruction. PCI = percutaneous coronary intervention.

Throughout the lateral RA and RA appendage, no intrinsic P-wave was sensed and no capture with high output pacing. On the anterolateral RA near the annulus, there was a low-voltage atrial signal and a capture threshold of 4.5 V @ 2 ms. On the superior limbus of the RA septum, intrinsic P waves were present with capture passively. The lead was fixed in place here (**Figure 4**), with sense P-wave amplitude of 0.3 to 0.6 mV, and pacing threshold 0.6 V @ 0.4 ms. The old RA lead was capped and abandoned.

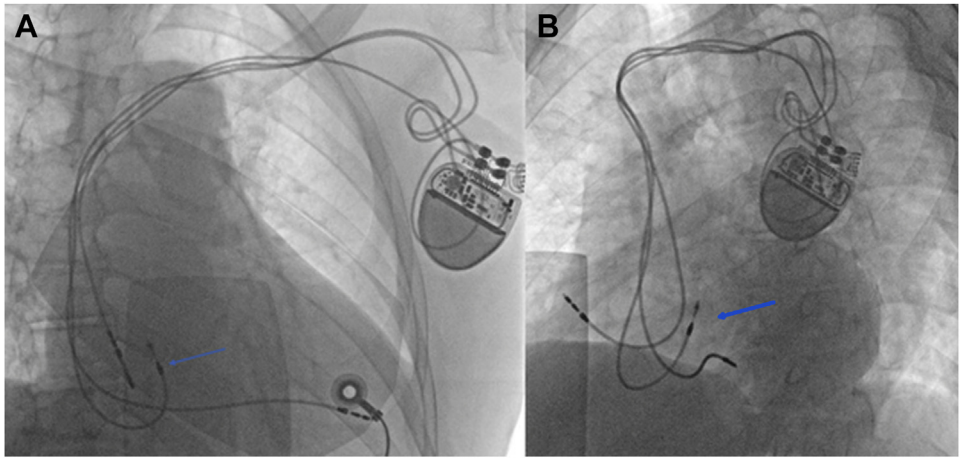
ECG performed after the new RA lead insertion and reprogramming of the device to restore atrioventricular synchrony showed atrial pacing and ventricular sensing with inferior Q waves consistent with previous myocardial infarction in the RCA distribution (**Figure 5**). There was significant improvement in the patient's clinical symptoms, especially dyspnea, and he was discharged on hospital day 7 after an otherwise unremarkable postoperative course.

DISCUSSION

Although uncommon, atrial infarction may lead to acute failure of a long-term right atrial pacing lead.[5,6] In cases previously reported, the patients had recovery of RA lead function during long-term follow-up, although when recovery occurred is unknown. Given our patient's worsening symptoms with RV pacing, we elected to revise his system rather than monitor for recovery of atrial capture. Our patient might have been less likely to recover RA lead function, given the delay in coronary revascularization after the onset of symptoms. In this case, the new onset of ventricular pacing obscured evaluation of the ST-segment changes that might have made a diagnosis of an inferior ST-segment elevation MI apparent. The inferior Q waves seen on ECG once intrinsic QRS complexes were available with effective atrial pacing suggests that irreversible ventricular myocardial damage had occurred. Inasmuch as the old lead was capped and abandoned, we may be able to evaluate for recovery of atrial electrical activity at a future generator change.

The coronary blood supply to the right and left atria typically arises from the right coronary artery and left circumflex artery, respectively, although overlap and redundancy may exist.[2,3] In our patient, proximal RCA occlusion led to myocardial injury along the lateral aspect of the RA, given that no viable tissue was identified in the area at the time of lead implantation. This finding is congruent

FIGURE 4 **Final Position of New Right Atrial Lead**

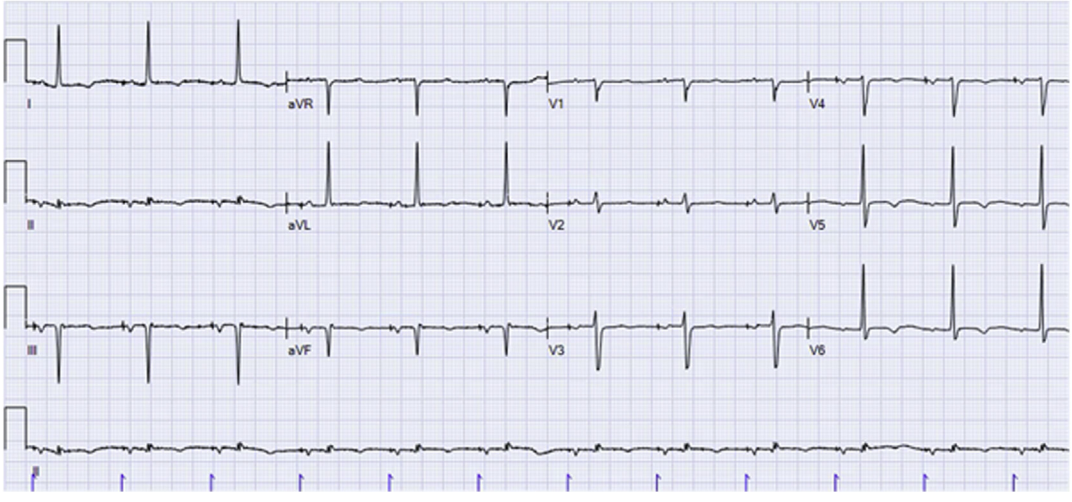


(A) Right anterior oblique and **(B)** left anterior oblique projections showing final position of the new right atrial lead **(blue arrow).**

with that observed during an autopsy study where the right atrial appendage was the area most frequently affected by infarction.[4] We suspect that the RA septum must be receiving its blood supply from a vessel other than that seen at the time of coronary angiography. Perhaps in patients with a history or suspicion of right coronary artery infarction without acceptable lead position parameters along the lateral RA, a septal position may be considered.

This case also highlights the importance of recognizing that rhythm changes and new ventricular

FIGURE 5 **Electrocardiogram After Right Atrial Lead Revision**



Electrocardiogram showing inferior Q waves previously obscured on electrocardiogram at presentation because of ventricular pacing.

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pacing can obscure the signs of acute myocardial injury. In this case, the elevated troponin and echocardiographic findings prompted urgent coronary angiography, where the diagnosis of acute MI was confirmed and treated.

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KEY WORDS cardiac pacemaker, electrocardiogram, myocardial infarction