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CASE REPORT

BEGINNER

CLINICAL CASE

Dual Chamber Pacemaker Implantation Complicated by Left Anterior Descending Coronary Artery Injury

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ABSTRACT

A 53-year-old female underwent dual-chamber pacemaker implantation for tachy-brady syndrome, which was complicated by anterior ST-segment elevation myocardial infarction and ventricular fibrillation due to right ventricular lead impingement on the left anterior descending coronary artery. Coronary artery injury is a rare complication of cardiac device implantation which requires a multidisciplinary team for management. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2019;1:633–7) © 2019 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/).

CASE PRESENTATION

A 53-year-old female patient was referred for dual-chamber pacemaker implantation due to paroxysmal atrial fibrillation with symptomatic tachy-brady syndrome. She was taken to the operating room for implantation of a magnetic resonance imaging device (Assurity, St. Jude Medical/Abbott, Lake Forest, Illinois) with a Tendril STS (St. Jude Medical/Abbott) 46-cm right atrial (RA) lead and a Tendril STS 52-cm right ventricular (RV) lead under fluoroscopic guidance with orthogonal views for confirmation of lead placement. Intraoperative atrial lead capture threshold was 0.75 V at 0.4 ms, sensing at 4.3 mV, and impedance of 550 Ω. Ventricular lead capture threshold was 1.0 V at 0.6 ms, somewhat reduced sensing at 3.2 mV but with good injury current, and impedance of 790 Ω. Upon closing the surgical wound, the patient developed ventricular fibrillation (VF) requiring electrical cardioversion at 200 J. A 12-lead electrocardiographic tracing was obtained which demonstrated ST-segment elevations in leads V_2 to V_6 as well as the inferior leads (**Figure 1**).

LEARNING OBJECTIVES

- To understand the relationship between coronary anatomy and cardiac implantable electronic device leads.
- To be able to recognize and manage coronary artery injury as a rare complication of lead implantation.

MEDICAL HISTORY. The patient previously had atrial flutter and had undergone a successful cavotricuspid isthmus ablation 4 months prior to the index event. However, she had since developed paroxysmal atrial fibrillation with tachy-brady syndrome. Monitoring (Zio Patch, iRhythm Technologies, San Francisco, California) demonstrated heart rates ranging from 40 to 170 beats/min with post-conversion pauses up to 4.7 s, which correlated with symptoms of presyncope. She otherwise had no significant medical history and was taking no medications. She was not taking anticoagulation as she had no CHA_2DS_2-VASc (i.e.,

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Informed consent was obtained for this case.

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ABBREVIATIONS AND ACRONYMS

CIED = cardiac implantable electronic device

LAD = left anterior descending

LAO = left anterior oblique

RA = right atrial

RAO = right anterior oblique

RV = right ventricular

STEMI = ST-segment elevation myocardial infarction

VF = ventricular fibrillation

Congestive heart failure, Hypertension, Age ≥75 years, Diabetes mellitus, Prior stroke, transient ischemic attack [TIA], or thromboembolism, Vascular disease, Age 65-74 years, Sex category [female]) risk factors independent of sex.

DIFFERENTIAL DIAGNOSIS. Lead dislodgement resulting in mechanical stimulation of the ventricular endocardium causing ventricular ectopy and arrhythmias was the initial primary concern. This condition may occur when the atrial lead prolapses into the RV and stimulates the outflow tract; however, this would not explain the ST-segment elevations seen on 12-lead electrocardiography. Lead perforation was another concern, with a pericardial effusion potentially explaining ST-segment changes, but VF would be an unusual sequela. Takotsubo or stress-induced cardiomyopathy might have explained these findings, although that would be a diagnosis of exclusion. Spontaneous development of plaque rupture leading to anterior ST-segment elevation myocardial infarction could also explain those findings but, given the lack of coronary disease risk factors in this patient, that also was believed to be unlikely. Finally, other causes of myocardial infarction with nonobstructive coronary artery disease, such as vasospasm or spontaneous coronary artery dissection, were also among the differential diagnoses for this patient's condition.

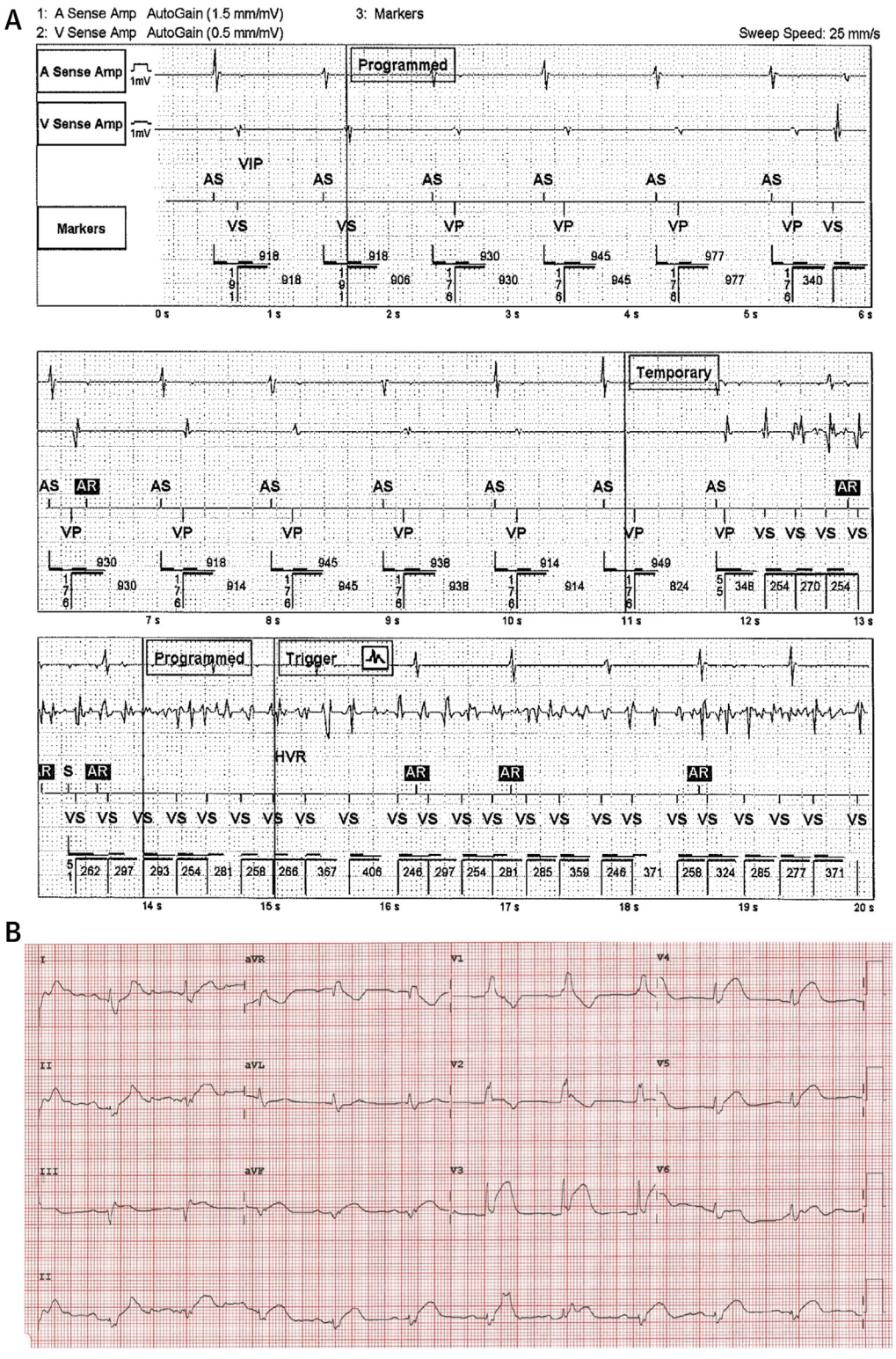
INVESTIGATION. The device was monitored by wireless telemetry throughout the case, and at the time the patient went into VF, the lead parameters demonstrated stable impedance, sensing, and capture values, making lead dislodgement unlikely. Bedside ultrasonography was performed, which showed no pericardial effusion, making lead perforation less likely. The patient was taken urgently to the cardiac catheterization laboratory, and coronary angiography was performed (**Figure 2A**). Results demonstrated significant vasospasm of the mid-to-distal left anterior descending (LAD) coronary artery, which improved with administration of intracoronary nitroglycerin, although the mid-distal LAD continued to demonstrate Thrombolysis In Myocardial Infarction (TIMI) flow scores of 1 to 2 (**Figure 2B**). ST-segment elevations persisted; thus, the decision was made to intervene with an attempt to improve coronary flow and consideration of an intravascular ultrasonography investigation. Heparin was administered to achieve an activated clotting time of >250 s, and a coronary wire (Choice Floppy, Boston Scientific, Boston, Massachusetts) was then passed to the distal LAD, which immediately restored a TIMI flow grade of 3 to the apical, wraparound segment. When the wire was pulled back, flow was again lost to the mid-distal LAD, suggesting the wire was allowing the vessel to remain patent. The coronary wire was readvanced to the distal LAD, and multiple angiographic views then clearly demonstrated the RV lead to be impinging on (and possibly through) the mid-distal LAD (**Figure 2C**, Video 1).

MANAGEMENT. The decision was made to reposition the RV lead in the catheter laboratory while maintaining the intracoronary wire as this was preserving flow to the apical LAD and also could provide a means of quickly managing coronary perforation if the patient began to decompensate. Although pocket-site bleeding was a concern, the decision was made to continue heparin for this purpose. The wound was reopened, and the RV lead was disconnected from the generator. The active fixation helix was retracted, and the lead was retracted. Immediately upon repositioning the lead, coronary angiography demonstrated a small blush of contrast at the prior location of the RV lead tip, suggesting a microperforation (**Figure 2D**, Video 2). Repeated angiographic images taken within 2 min showed complete resolution of the contrast extravasation (**Figure 2E**, Video 3). The lead was repositioned to a more inferior septal position, retested, and sutured in place (**Figure 2F**). Floseal (Baxter, Deerfield, Illinois) and Surgicel (Johnson & Johnson, New Brunswick, New Jersey) were applied to the pocket, and the wound was closed. Next day, echocardiography showed a small inferoapical wall motion abnormality but no pericardial effusion and otherwise preserved systolic function. Troponin I value peaked at 11.9 ng/ml. Follow-up echocardiography 3 months later showed resolution of wall motion abnormality. She has done well clinically on follow-up, with no cardiac complaints or episodes of arrhythmia on device interrogation.

DISCUSSION

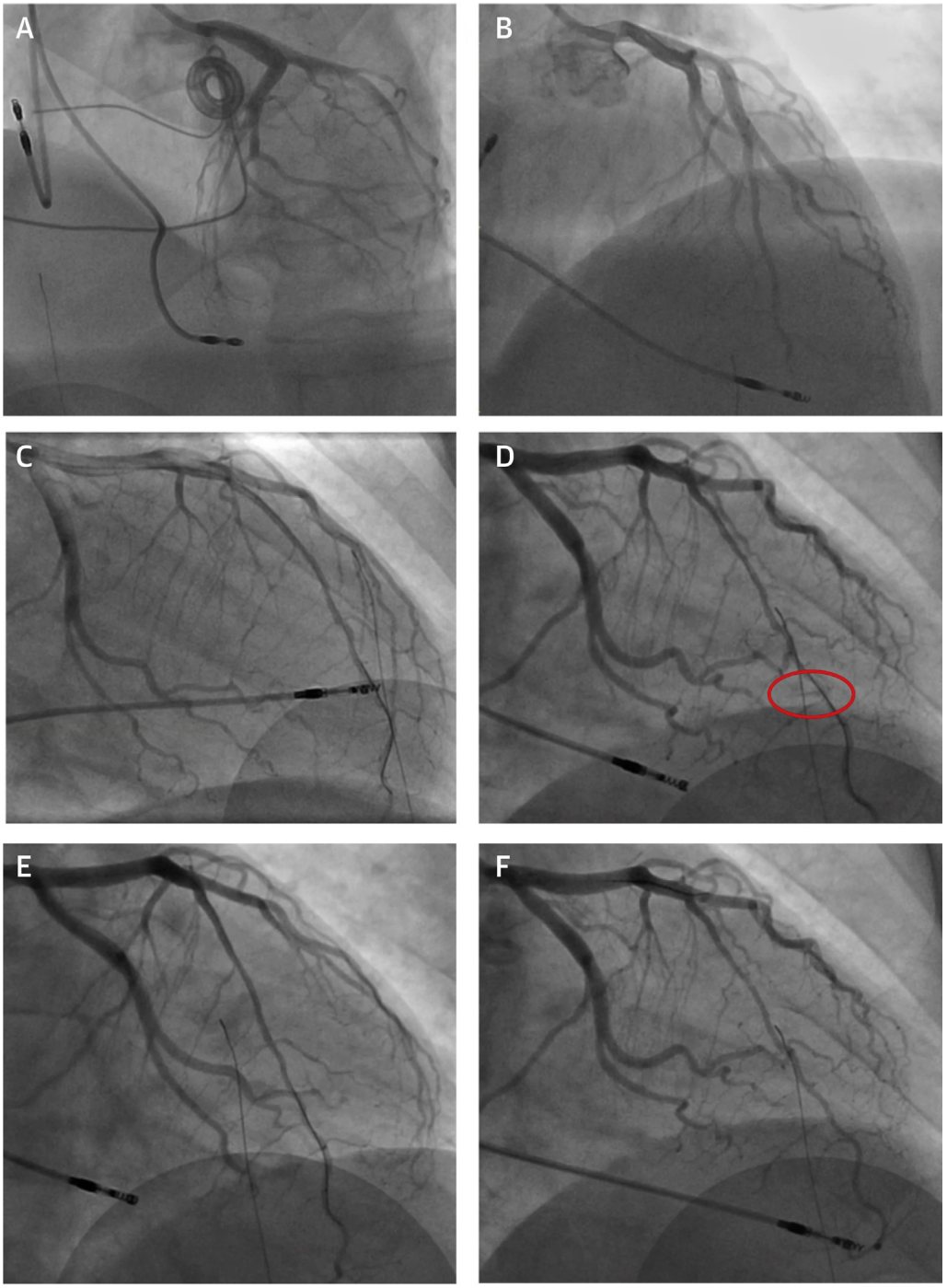
Coronary artery injury is a rare complication of cardiac implantable electronic devices (CIED) (1). There have been several reported cases of LAD injury due to right ventricular pacemaker or defibrillator lead implantation (2–4). This occurs with an anteroseptal placement of an active fixation RV lead, which can cause mechanical injury to the LAD. Computed tomography imaging data observed by Pang et al. (5) demonstrated that RV leads in an anteroseptal position were a median 4.7 mm from the LAD. To the present authors' knowledge, this represents the fourth reported case of a LAD injury due to RV lead

FIGURE 1 **Device Interrogation**



(A) Device interrogation demonstrating A-sensed, V-paced rhythm with initiation of spontaneous ventricular fibrillation, which required external 200-J defibrillation. **(B)** Twelve-lead ECG demonstrates sinus rhythm with a right bundle branch block and ST-segment elevations in leads V_2 to V_6, II, III, and aVF. ECG = electrocardiography.

FIGURE 2 Cranial Angiographic Images



(A) LAO cranial angiographic image demonstrates severe vasospasm of the mid-to-distal LAD, with the RV lead clearly seen to the right of the interventricular septum but with insertion near the distal LAD. **(B)** RAO cranial angiographic image after intracoronary nitroglycerin administration demonstrates improvement in LAD vasospasm but obstruction of flow at the RV lead insertion site. **(C)** RAO view with Choice Floppy intracoronary wire, with restoration of flow to the distal LAD. The RV lead can be seen with insertion obstructing the LAD. **(D)** Angiographic image obtained immediately after pull-back of the RV lead, with a small area of contrast extravasation at the original RV lead insertion site **(red circle)**. **(E)** Repeated angiographic image <2 min after RV lead pull-back demonstrates resolution of contrast extravasation from the LAD. **(F)** Final RV lead position with no evidence of contrast extravasation. See Videos 1, 2, and 3. LAD = left anterior descending; LAO = left anterior oblique; RAO = right anterior oblique; RV = right ventricular.

placement. The present case is unique in that coronary perforation was clearly shown; however, no covered stent or other intervention was required due to self-resolution of the contrast extravasation. The authors hypothesized that, because the perforation was likely small and located intramyocardially, this allowed the coronary injury to self-resolve quickly as the very next angiographic image showed no further evidence of perforation. Of note, neither the previously reported cases nor the present case developed pericardial effusion.

Other coronary complications also can occur with CIED implantation. Coronary injury to the right coronary artery has been reported as a result of the right atrial lead (6,7). Those cases required surgical intervention to correct. This complication is most likely to occur with RA lead placement in close proximity to the tricuspid annulus, as this can injure the artery coursing in the AV groove.(5) Finally, obstruction of the left internal mammary artery graft has also been reported as implanted leads pass through the subclavian vein and cause external compression of the left internal mammary artery (8).

FOLLOW-UP. The patient was seen at 2 weeks after implantation and was doing well, with no episodes of arrhythmia on device interrogation.

CONCLUSIONS

Coronary artery injury is a rare complication of CIED implantation. A multidisciplinary team approach is critical in managing these complex sequelae.

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KEY WORDS cardiac pacemaker, coronary angiography, electrophysiology, myocardial infarction, ventricular fibrillation

APPENDIX For supplemental videos, please see the online version of this paper.