A CASE FOR EDUCATION

Loss of right ventricular capture: When it is not the lead's fault

Albert Y. Liu, MD, MS,[1] Gregory M. Marcus, MD, MAS, FHRS,[1] Byron K. Lee, MD,[2] Joshua D. Moss, MD, FHRS,[1] Cara N. Pellegrini, MD, FHRS[1,3]

From the [1]Division of Cardiology, University of California, San Francisco, San Francisco, California, [2]Division of Cardiology, Palo Alto Veterans Affairs Medical Center, Palo Alto, California, and [3]Division of Cardiology, San Francisco Veterans Affairs Medical Center, San Francisco, California.

Introduction

Cardiac implantable electronic devices are crucial in treating various cardiac arrhythmias and conduction disorders. Contemporary pacemakers have grown increasingly sophisticated and complex, and device-related complications can be difficult to recognize. Successful device troubleshooting requires a systematic approach. The differential for pacemaker lead failure of capture is broad, but includes lead dislodgement or perforation; loss of lead integrity, such as fracture or insulation defect; and issues at the device electrode–myocardium interface. This case report explores a unique instance of isolated right ventricular (RV) lead loss of capture due to a pericardial mass.

Case report

An 85-year-old man presented to the emergency department with several weeks of fatigue and shortness of breath. He had a history of paroxysmal atrial fibrillation and complete heart block requiring a dual-chamber pacemaker, which was later extracted due to a device infection and was reimplanted on the right side. He developed pacing-induced cardiomyopathy and had his pacemaker upgraded to a Percepta cardiac resynchronization therapy pacemaker (Medtronic, Minneapolis, MN) 4 years prior to presentation. One year later, he underwent surgical aortic valve replacement for severe aortic regurgitation. Coronary angiography performed at this time showed nonobstructive coronary disease.

Presently, the patient reported a few weeks of reduced energy and shortness of breath when climbing stairs, but denied chest pain, syncope, new palpitations, or lightheadedness. His wife described unusual night-time episodes of guttural breathing, reminiscent of episodes around the time of his diagnosis of complete heart block, and she raised concerns about device malfunction. Vitals signs were normal and the patient appeared comfortable, without signs of venous congestion, lung crackles, or lower extremity edema. Laboratory evaluation was unremarkable, including an undetected troponin I level, normal thyroid levels, and normal electrolyte levels. His 12-lead electrocardiogram (ECG) showed an atrioventricular paced rhythm with premature atrial and ventricular complexes. The QRS morphology had changed significantly compared with a prior ECG, suggesting left ventricular (LV)-only pacing (Figure 1). A chest x-ray (CXR) showed a right-sided cardiac resynchronization therapy pacemaker with the coronary sinus lead in a basal anterolateral position, with no obvious lead fracture, lead dislodgement, or poor connection between the lead pin and the generator set screw (Figure 2).

Bedside device interrogation revealed a subacute but rapid rise in device-measured RV pacing threshold, with stable lead impedance and no lead noise (Figure 3). Both unipolar and bipolar capture thresholds on the RV lead were confirmed to be elevated at 4.5 V at 1.5 milliseconds and 5 V at 1.5 milliseconds, respectively. LV lead diagnostics were stable, with a capture threshold of 1.25 V at 0.4 milliseconds with bipolar pacing from LV3 to LV4. AdaptivCRT (Medtronic) was on with LV to RV and auto V-V Pace Delay. Ventricular capture management was disabled to prevent algorithm-related loss of LV capture and RV lead output was increased to 8 V at 1.2 milliseconds to restore biventricular pacing, with immediate improvement in the patient's symptoms. A computed tomography (CT) chest scan demonstrated a necrotic pericardial mass at the RV apex in close approximation to the lead tip (Figure 4). This mass was positron emission tomography (PET) avid on a subsequent PET CT scan, with additional concern for a potential tract of avidity between the lead and the mass. The patient underwent excisional biopsy of the mass, with a histopathologic

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Address reprint requests and correspondence: Dr Cara N. Pellegrini, Dr Cara N. Pellegrini, San Francisco Veterans Affairs Medical Center, 4150 Clement Stree, 111C, San Francisco, CA 94121. E-mail address: Cara.Pellegrini@ucsf.edu.

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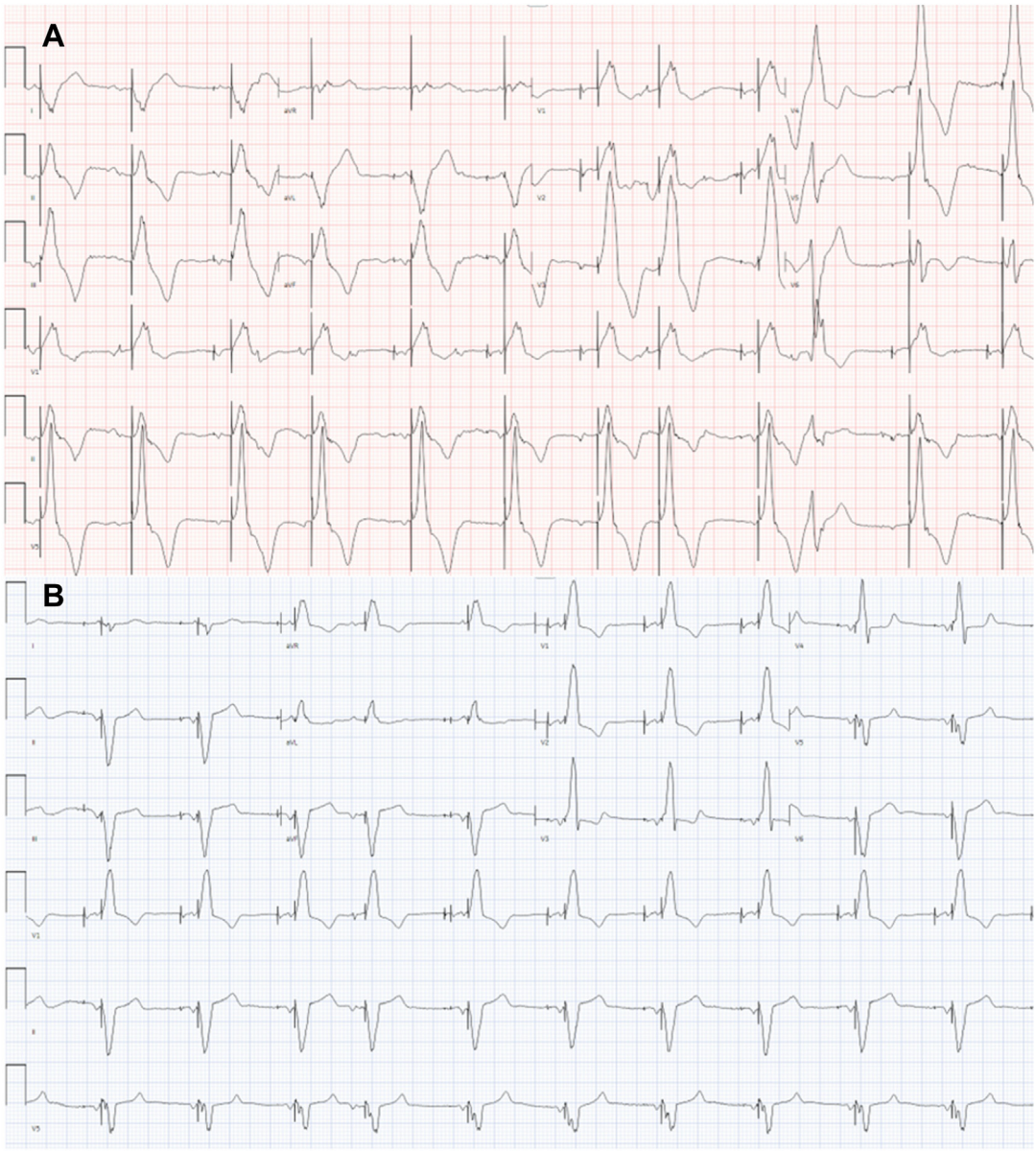

Figure 1 **A:** Electrocardiogram (ECG) at presentation: an atrioventricular (AV) paced rhythm with premature atrial and ventricular complexes. QRS morphology is consistent with left ventricular (LV)-only pacing from the basal anterolateral LV. This is markedly different from (**B**), an ECG obtained after cardiac resynchronization therapy pacemaker implantation, which shows an AV paced rhythm with biventricular pacing.

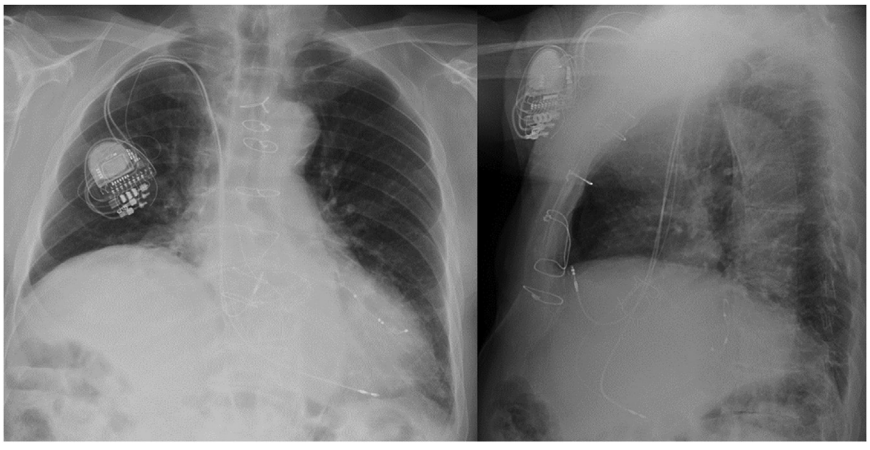

Figure 2 Posteroanterior and lateral chest x-ray showing right-sided cardiac resynchronization therapy pacemaker with leads terminating in the right atrial appendage, right ventricular apex, and anterolateral branch of the coronary sinus.

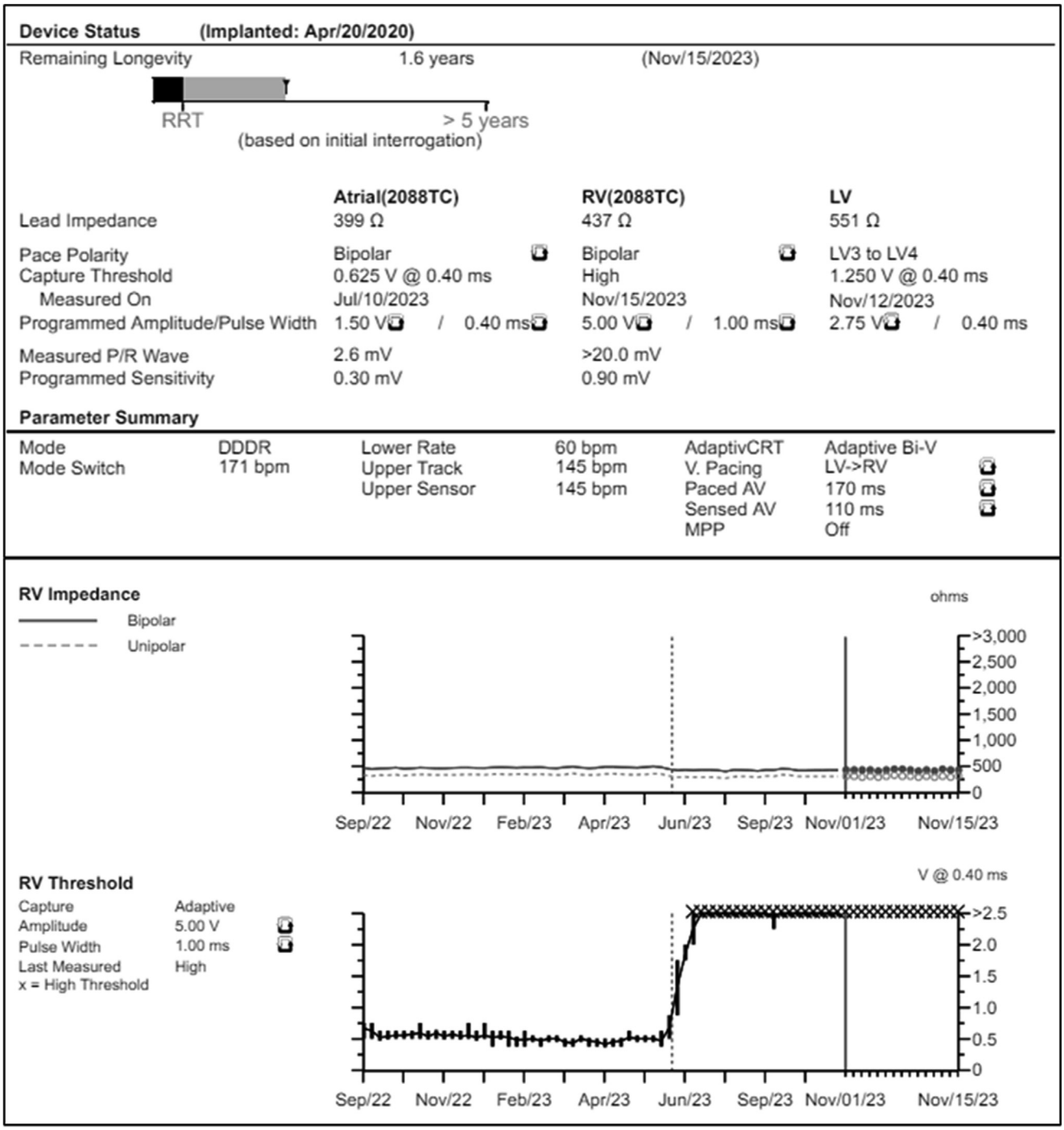

Figure 3 Initial device interrogation showing rapid rise in the right ventricular (RV) lead threshold with stable lead impedances and sensing. Right atrial and left ventricular lead parameters remained stable from prior.

diagnosis of fibrosis with a central region of inflammation and granulation tissue, consistent with an organizing abscess. Intraoperative cultures grew *Cutibacterium acnes* and *Staphylococcus epidermidis*. Device extraction was discussed with the patient due to concerns for lead-related infection, inadequate pacing safety margin, and substantial battery drain from high pacing outputs, but was deferred due to potential morbidity and patient preference. He received a course of antibiotics, followed by ongoing suppressive antibiotics, but continued to have persistently elevated RV thresholds. A repeat PET CT scan 2 months after excisional biopsy demonstrated a residual fluid collection with enhancement at the operative site, thought to be most consistent with a postoperative seroma. A few months later, he was found to have complete dislodgment of the RV lead and underwent successful extraction and reimplantation of the RV lead.

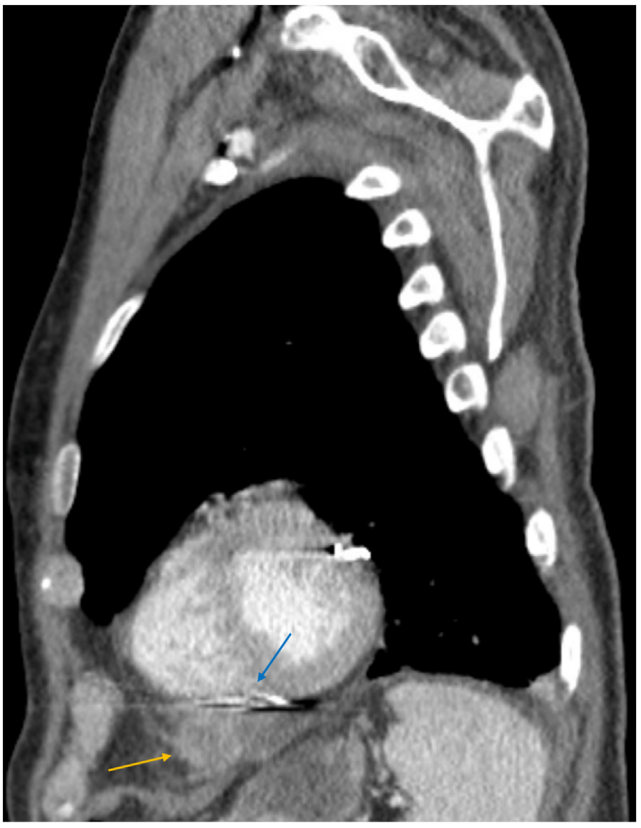

Figure 4 Computed tomography chest scan shows a necrotic 4-cm pericardial mass (*orange arrow*) adjacent to the right ventricular lead tip (*blue arrow*).

Discussion

Failure to capture is a common problem encountered in pacemaker troubleshooting. Determination of the etiology requires a thorough evaluation, including a focused history and physical, routine blood work evaluating for metabolic and electrolyte abnormalities, ECG, CXR, and device interrogation. Symptoms of device malfunction can be subtle and nonspecific, such as fatigue, weakness, syncope, lightheadedness, or palpitations.[1] The ECG is often a critical first step in identifying failure to capture.

The differential diagnosis should be guided by the timing of dysfunction.[1,2] Lead dislodgement, problems with the lead pin–header connection, and lead perforation usually occur early, although late perforation has been described.[3] Lead fracture, insulation failure, battery failure, and issues at the electrode–myocardial interface usually occur later. Metabolic, electrolyte, or drug effects could occur at any time. Many severe metabolic disturbances can affect pacing thresholds, with the most common being hyperkalemia.[4–8] Class 1C antiarrhythmics are the most common culprits of drug-induced failure to capture.[9–12]

Issues at the pin–header connection, including loose set screws, misalignment, or air in the header, are often associated with high impedances. Lead fracture is typically a late complication and often accompanied by high impedance, failure to pace, and sensing problems. These electrical abnormalities can be intermittent and provocative maneuvers may be necessary to reveal the fracture. If the fracture is at the outer coil, unipolar programming may restore the ability to capture. Similarly, an insulation break is often a late complication and accompanied by signature abnormalities, including low impedance, failure to output, and sensing problems. If unipolar programming restores normal pacing, the defect is likely at the outer insulation.

Issues at the electrode–myocardial interface can be related to either the electrode or myocardium. Myocardial infarction or infiltrative cardiomyopathies can render the myocardium at the site of the electrode electrically inert, increasing capture thresholds. There can also be changes in capture threshold after cardioversion, due to local myocardial burns from transmission of electrical current at the lead tip.[13] Transient rise in capture threshold has also been reported with pulsed field ablation.[14] Excessive fibrosis over time can lead to exit block at the lead tip.[15,16] It can be difficult to distinguish exit block from microdislodgement or microperforation, which typically improve with repositioning of the leads.

An algorithm for identifying the cause of failure to capture is detailed in Figure 5. First, assess the lead pacing impedance. Contemporary lead impedances usually range from 200 to 2000 Ω.[1,17] A low pacing impedance suggests insulation breach. Programming the lead to unipolar pacing and sensing will allow for differentiation between inner and outer insulation break, as unipolar pacing and sensing will be preserved with an outer insulation break. If the lead pacing impedance is high, then the concern is either for conductor coil fracture or poor connection at the device header. If unipolar pacing and sensing are intact, the problem is likely with the outer conductor coil. With a high pacing impedance, it is crucial to pay close attention to the CXR to evaluate for fracture in the lead and assess the connection at the connector block. Lead pin–header misalignment can be seen on CXR, but loose set screws may be difficult to detect. For leads placed via subclavian puncture, fractures are often found at the intersection between the first rib and the clavicle.[18] If the pacing impedance is normal, then look for dislodgement on the CXR. If there is no dislodgement, then the cause can likely be narrowed down to an issue with the electrode–myocardial interface, microdislodgement not identifiable on CXR, new drugs, or an electrolyte or metabolic abnormality.

In our case, the patient's presenting symptoms were increased fatigue and shortness of breath. These symptoms were thought to be due to worsening heart failure from loss of biventricular pacing. The patient's wife's description of nocturnal guttural breathing was further concerning for episodes of asystole during Medtronic's Ventricular Capture Management algorithm. His ECG showed a wider QRS morphology with a change in axis suggestive of loss of biventricular pacing and LV-only pacing. This was confirmed by device interrogation, which showed a rise in the pacing threshold of the RV lead, accompanied by failure to capture. The LV pacing threshold remained stable, was programmed bipolar LV3 to LV4, and showed no anodal

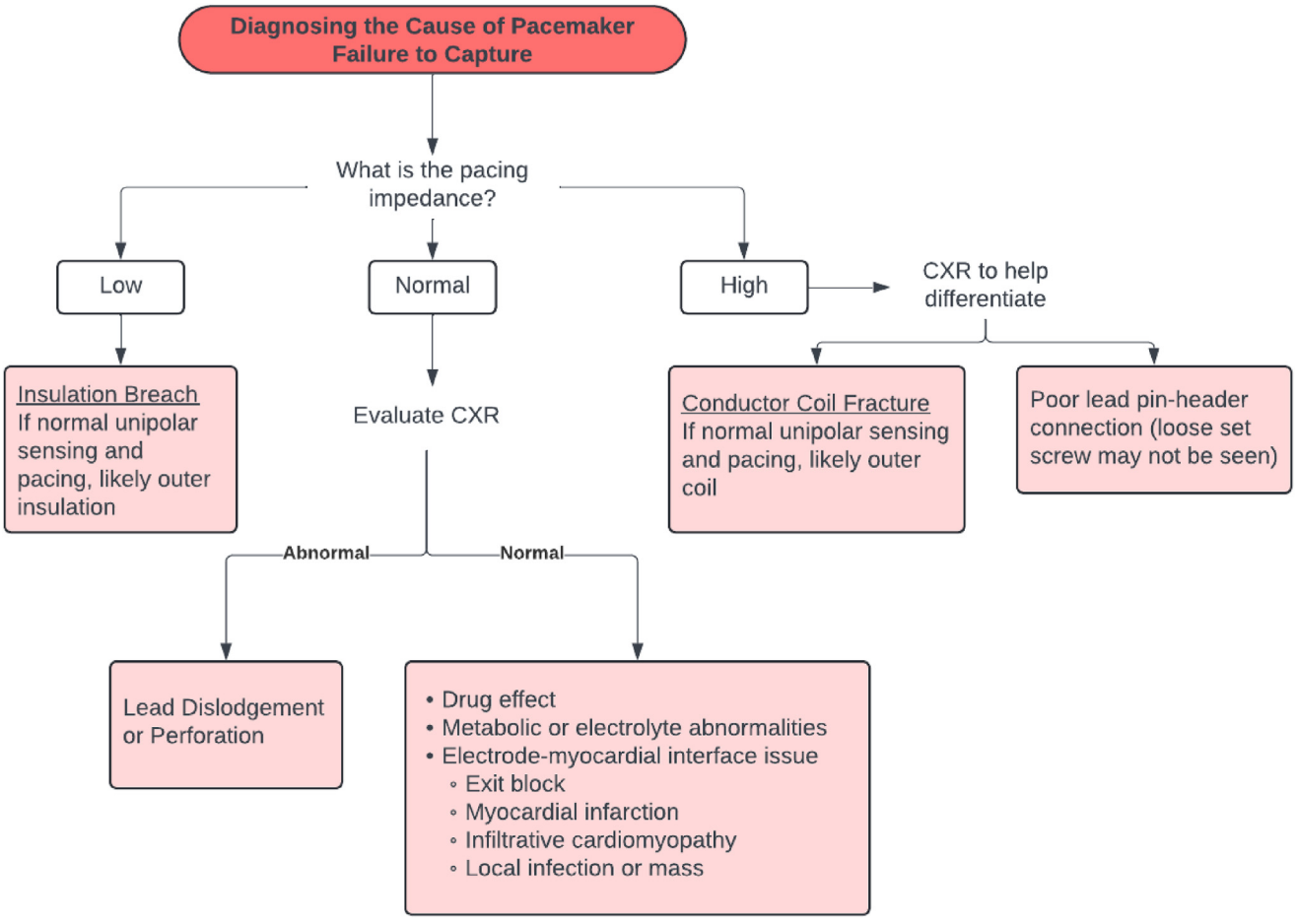

Figure 5 Algorithm for identifying cause of pacemaker failure to capture. CXR = chest x-ray.

reliance on the RV lead. Thus, there was no concern for loss of LV capture with RV lead disruption. The RV lead pacing impedance was stable, making lead fracture, insulation break, and loose set screw less likely. This was supported by the CXR showing stable lead positioning with no evidence of fracture or air in the connector block. No macrodislodgement was identified. The patient's laboratory evaluation was normal with no evidence of an acute metabolic or electrolyte abnormality, and there were no new medications associated with pacing threshold changes. This left an issue with the electrode–myocardial interface, microdislodgement or microperforation as the primary explanation. Ultimately, this led to a CT scan of his chest and a diagnosis of a pericardial abscess. The PET CT scan showed a potential tract of avidity between the lead tip and the mass, suggesting microperforation and subsequent abscess formation over time as the mechanism. The subsequent excisional biopsy then contributed to macrodislodgement of the lead a few months later. To our knowledge, this is the first described report of an isolated rise in pacing threshold with failure to capture related to an adjacent pericardial mass. In this case, prompt identification led to an expedited diagnosis of a pericardial abscess and treatment with excisional biopsy and antibiotics.

Conclusion

The assessment of lead malfunction requires a systematic approach and a thorough understanding of the etiologies for types of failure. This case underscores the importance of considering electrode–myocardial interface issues in patients with unexplained changes in pacemaker lead thresholds. We provide a discussion of the causes and a clinical algorithm for working through patients who present with failure to capture.

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