A rare case of delayed right atrial lead perforation in an adolescent patient with Brugada syndrome

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Introduction

Myocardial lead perforation following cardiac implantable electronic device (CIED) implantation is rare. Lead perforations are classified as acute, subacute, or chronic based on the timing of presentation after the device implantation. The majority occur within the first 24 hours after device implantation. We report a case of a pediatric patient with implantable cardioverter-defibrillator (ICD) lead perforation leading to cardiac tamponade who subsequently underwent surgical repair of the perforation without lead extraction.

Case report

A 15-year-old female patient with genotype-positive Brugada syndrome, sinus node dysfunction, and atrial flutter status post catheter ablation underwent dual-chamber ICD placement. Her father also had a history of atrial tachycardia and sinus node dysfunction. He died suddenly 1 week after pacemaker implantation. Her ICD consisted of a right atrial lead (Medtronic #4076 45 cm) and right ventricular lead (Medtronic Sprint Quattro #6935 55cm), in conjunction with a Medtronic Evera MRI XT DR Surescan ICD generator, Model #DDMB1D4. Her lower rate limit was programmed to 70 pace per minute (PPM) and upper limit rate set to 150 PPM. The patient had no complications after the initial placement of the device. She was followed up in the office and with remote transmissions every 3 months and the device was functioning appropriately. The patient was an accomplished ballet dancer.

Six months after ICD placement, she presented to the emergency department with a 1-day history of worsening substernal chest pain, which improved with leaning forward. On presentation, her vital signs were normal. Laboratory evaluation revealed a normal comprehensive metabolic panel, complete blood count, troponin level, NT-pro-brain natriuretic peptide, and C-reactive protein levels. Chest radiograph showed no evidence of cardiomegaly (Figure 1A and 1B) or detectable changes in lead position compared with previous images. Her electrocardiogram demonstrated atrial-paced rhythm with prolonged atrioventricular conduction and no ST elevation (Figure 2). ICD device interrogation revealed stable lead parameters with no change in pacing threshold and lead impedance with bipolar pacing. Four hours after her initial evaluation, she developed nausea and emesis associated with pallor, lightheadedness, and diaphoresis. She was hypotensive with a narrow pulse pressure (blood pressure of 68/53 mm Hg), heart rate of 71 beats per minute, and oxygen saturation of 100% on room air. Physical examination revealed distant heart sounds, weak peripheral pulses with delayed capillary refill, and cold and clammy skin. A transthoracic echocardiogram revealed a moderate-sized circumferential pericardial effusion and preserved systolic function (Figure 3).

The patient was resuscitated with boluses of isotonic crystalloid and the lower limit rate on the ICD was increased to 100 PPM to assist with increasing cardiac output in the setting of sinus node dysfunction. A diagnostic and therapeutic pericardiocentesis performed at bedside revealed about 50 mL of frank blood, concerning for a hemopericardium. She underwent chest computed tomography (CT) to evaluate for lead perforation, which was inconclusive owing to an

KEY TEACHING POINTS

- Myocardial lead perforation following cardiac implantable electronic device (CIED) implantation is rare, but it can be a fatal complication if not diagnosed promptly.
- High index of suspicion for lead perforation is required in patients with CIEDs when they present with chest pain.
- Lead perforations sometimes may not present with changes in lead impedance and sensing threshold.

KEYWORDS Cardiac implantable electronic device; Right atrial lead perforation; Hemopericardium; Brugada syndrome; Cardiac tamponade
(Heart Rhythm Case Reports 2024;10:582–585)

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https://doi.org/10.1016/j.hrcr.2024.05.018

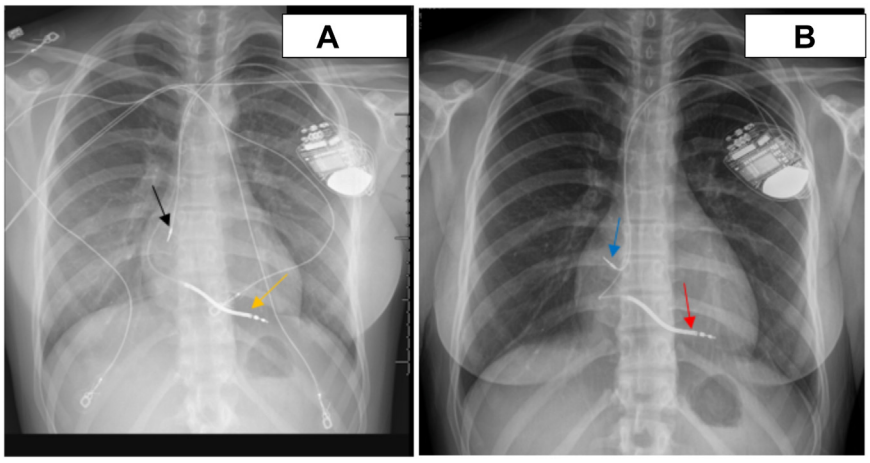

Figure 1 **A:** Initial chest radiograph performed after implantable cardioverter-defibrillator implantation showing defibrillator lead in right ventricle (*yellow arrow*) and pacing lead in right atrium (*black arrow*). **B:** Chest radiograph performed upon presentation to the emergency department showing defibrillator lead in right ventricle (*red arrow*) and pacing lead in right atrium (*blue arrow*).

artifact. Because of concern for hemodynamic instability, the patient underwent surgical exploration and repair. She was found to have 250 mL of blood in her pericardium. A perforation was noted in the lateral aspect of the base of the right atrial appendage by the fixation screw of the atrial pacing lead with the terminal part of the screw helix perforating the heart. There were also stigmata of injury by the screw with granulation tissue formation on the right pericardial surface. The atrial lead was interrogated and was adequately functioning, with lead impedance 456 ohms, capture threshold 1.75 V at 0.4 ms, and sensing 1.3 mV. The decision was made to preserve the lead, and the site of perforation was imbricated onto the exit site of the lead with a purse-string suture, which was reinforced with Teflon pledgets. There was no evidence of right ventricular lead perforation. Her lower limit rate on ICD was reprogrammed to 70 PPM and she was transfused with 495 mL of packed red blood cells. A follow-up echocardiogram obtained 2 days later showed no signs of pericardial effusion. The patient was discharged on postoperative day 3. At her most recent follow-up 3 months later, her device interrogation revealed appropriate atrial lead impedance, capture threshold, and sensing. Her echocardiogram revealed normal biventricular systolic function and no evidence of pericardial effusion.

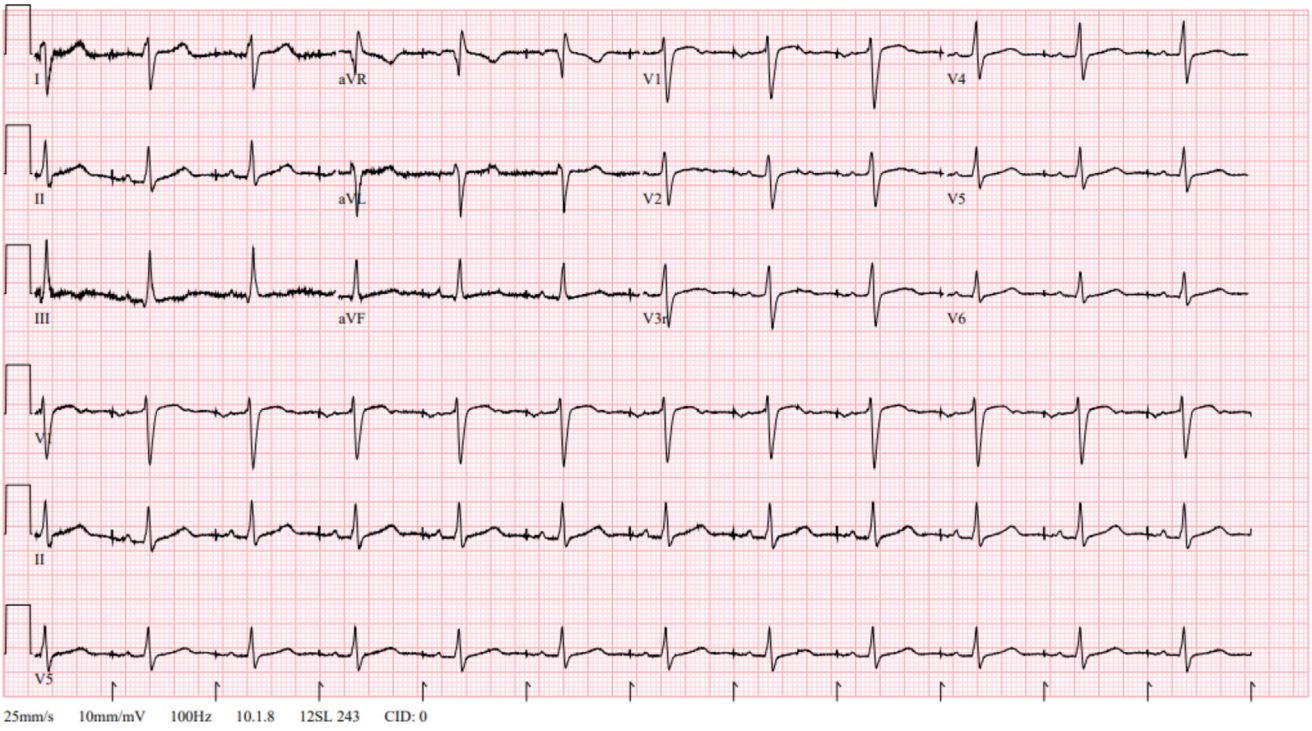

Figure 2 Electrocardiogram on initial presentation showing atrial pacing and stimulation to activation delay with normal PR interval.

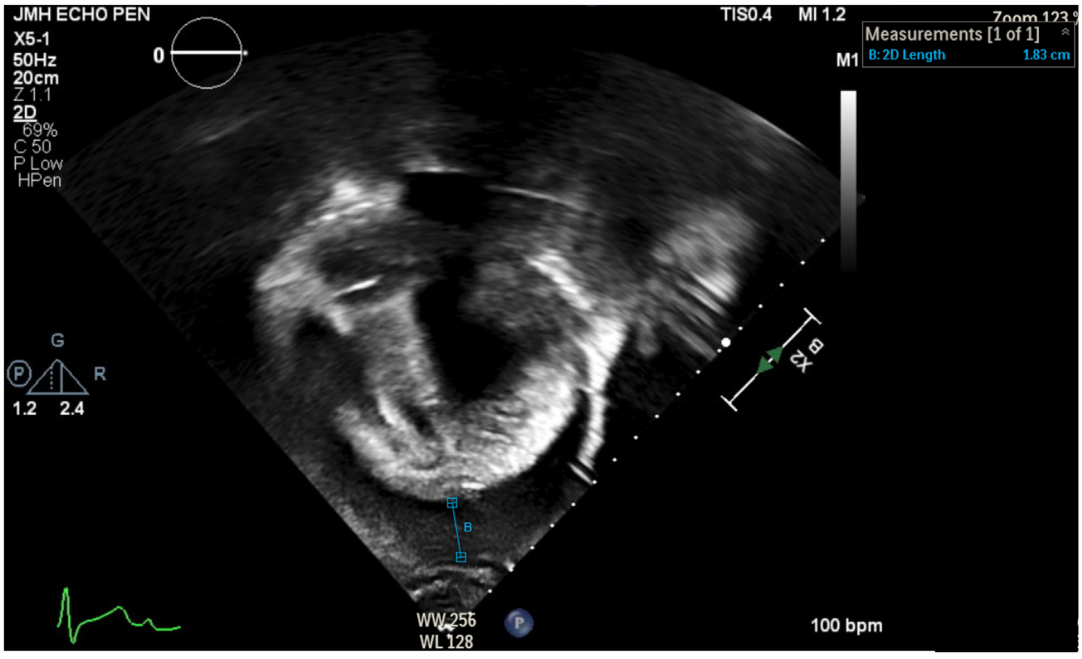

Figure 3 Apical 4-chamber view showing moderate-size circumferential pericardial effusion.

Discussion

Lead perforation of myocardium following CIED implantation is rare. The reported incidence of lead perforation involving the adult population is 0.1%–0.8% after pacemaker implantation and 0.6%–5.2% after ICD implantation.[1,2] Pediatric cardiac lead perforations have not been well documented in the literature.

Lead perforations are classified as acute, subacute, or chronic if they occur within 7 days, between days 7 and 30, or greater than 30 days postimplantation, respectively.[3] Acute lead perforations are significantly more common.[4] Acute lead perforation may present with a pericarditis type of chest pain to pericardial effusion and cardiac tamponade, whereas delayed perforation (ie, lead perforation after 1 month postimplantation) rarely presents with cardiac tamponade, probably owing to self-sealing properties of myocardial wall by contraction, hemostasis, and subsequent fibrosis.[5] The incidence of right atrial lead perforation is greater than right ventricular lead perforation.[6]

The risk factors associated with myocardial perforation reported in the adult population include recent steroid use, anticoagulant use, implant techniques, design characteristics of the lead, increasing age, female sex, and low body mass index.[7–9] Right ventricular septal lead placement is associated with lower risk of perforation owing to thick ventricular septal tissue.[10] A strong suspicion for lead perforation despite a normal device interrogation is important when patients present with chest pain. A chest radiograph may demonstrate lead perforation, but subtle perforations can often be missed owing to the inability of plain radiography to differentiate between the vascular cavity, myocardium, and pericardium.[5,11] Transthoracic echocardiogram is a good initial imaging modality, especially when there is a suspicion for pericardial effusion and to visualize lead position.[12] Three-dimensional echocardiography has been proposed to be superior in lead visualization, as it provides a more comprehensive view of the intracardiac structure.[13] Chest CT is a sensitive imaging modality for diagnosis of lead perforation, but beam hardening, bloom, and motion artifacts of the high-density metallic pacing leads remain issues inherent in a CT study.[14,15] Management of lead perforation varies from lead extraction to repositioning to thoracotomy with lead removal. When a patient is asymptomatic, there is no pericardial effusion, and device interrogation identifies normal electrical parameters, the lead may be left in situ. The modality of intervention should be based on the timing of perforation, hemodynamic stability, comorbidities, and the presence of complicating factors such as pneumothorax and pericardial tamponade.[16] Surgery is the treatment of choice for patients with unstable hemodynamic conditions. For stable patients, traction of the leads, with or without a percutaneous lead extraction sheath, is feasible and safe.[17]

The mechanism of this rare, late atrial lead perforation in our patient is unclear. While the lead was placed in the right atrial appendage, it may have been located just between the pectinate muscles at a thinner portion of the atrial wall. Chronic erosion may have been exacerbated by repetitive exertion during dance training. An alternative theory, though quite speculative, may be that there may be atrial wall vulnerability associated with Brugada syndrome.[18] The presence of atrial flutter and sinus node dysfunction, a feature previously described in patients with Brugada syndrome, may indicate atrial tissue abnormalities.[18]

Conclusion

In conclusion, our case report highlights that lead perforations may not present with changes in lead impedance and sensing threshold. A prompt suspicion and diagnosis is important to prevent a fatal complication such as cardiac tamponade in patients with CIEDs with lead perforation.

Funding Sources: None.

Disclosures: All the authors declare that they have no conflicts of interest to disclose.

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