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Bow-and-arrow sign on point-of-care ultrasound for diagnosis of pacemaker lead-induced heart perforation: A case report and literature review

POCUS and lead perforation

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Abstract

BACKGROUND

Pacemaker lead-induced heart perforation is a rare but life-threatening complication of pacemaker implantation, and timely diagnosis remains a challenge for clinicians. Here, we report a case of cardiac perforation by lead rapidly diagnosed by a “bow-and-arrow” sign on point-of-care ultrasound (POCUS).

CASE SUMMARY

A 74-year-old Chinese female who had undergone permanent pacemaker implantation 26 d before suddenly presented with severe dyspnea, chest pain, and hypotension. The patient had received emergency laparotomy for an incarcerated groin hernia and was transferred to the intensive care unit (ICU) 6 d before. Computed tomography (CT) was not available due to unstable hemodynamic status, so POCUS was performed at the bedside and revealed severe pericardial effusion and cardiac tamponade. Subsequent pericardiocentesis yielded a large volume of bloody pericardial fluid. Further POCUS by an ultrasonographer revealed a unique “bow-and-arrow” sign indicating right ventricular (RV) apex perforation by the pacemaker lead, which facilitated the rapid diagnosis of lead perforation. Given the persistent drainage of pericardial bleeding, urgent off-pump open chest surgery was performed to repair the perforation. However, the patient died of shock and multiple organ dysfunction syndrome (MODS) within 24 h post-surgery. In addition, we also reviewed the literature on the sonographic features of RV apex perforation by lead.

CONCLUSION

POCUS enables the early diagnosis of pacemaker lead perforation at the bedside. A step-wise ultrasonographic approach and the “bow-and-arrow” sign on POCUS are helpful for rapid diagnosis of lead perforation.

Key Words: Point-of-care ultrasound; Heart perforation; Pacemaker lead; Cardiac pacemaker; Review; Case report

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Core Tip: Pacemaker lead-induced heart perforation is a rare but life-threatening complication of pacemaker implantation. Further, timely diagnosis remains a challenge for clinicians. Here, we report a novel case of right ventricular apex perforation by pacemaker lead associated with pericardial tamponade rapidly diagnosed by a unique “bow-and-arrow” sign on point-of-care ultrasound (POCUS). We also propose a step-wise diagnostic approach using POCUS to enhance diagnostic speed and accuracy for lead-induced heart perforation. This step-wise ultrasonographic approach and the “bow-and-arrow” sign on POCUS are helpful for rapid diagnosis of lead perforation.

INTRODUCTION

Cardiac pacing has revolutionized the care of severe bradycardia^[1]. However, patients receiving pacemaker therapy are at high risk of complications such as infection, bleeding, pneumothorax, deep venous thrombosis, lead dislodgement, and heart perforation^[2]. While heart perforation by lead occurs in only about 1% of implantation cases, it is one of the most life-threatening complications^[3]. The clinical manifestations of lead perforation vary markedly, ranging from chest pain, dyspnea, and syncope to lethal cardiac tamponade and arrest^[4]. Presently, there is no consensus on the optimal diagnostic procedure for lead-related heart perforation, and timely diagnosis remains a great concern for cardiologists and emergency and critical care professionals^[5].

Point-of-care ultrasound (POCUS) has become a valuable tool for diagnosis in the acute care setting over the past two decades, especially for heart-related conditions; indeed, POCUS has been referred to as “the new stethoscope”^[6]. POCUS using small

and portable ultrasound machines provides real-time, low-cost, non-invasive, and non-radiative diagnostic information at the bedside, and has proven especially beneficial for the early diagnosis of critically ill patients unable to receive other imaging examinations, such as computed tomography (CT) scan^[7]. Here, we report a rare case of pacemaker lead-induced right ventricular (RV) apex perforation associated with acute cardiac tamponade diagnosed based on a distinctive “bow-and-arrow” sign on POCUS. We also describe a step-wise POCUS-based approach for diagnosis of RV apex perforation, and present a literature review on the sonographic features of lead-induced RV apex perforation.

CASE PRESENTATION

Chief complaints

A 74-year-old Chinese female (body mass index [BMI] of 20 kg/m²) suddenly presented with chest pain, dyspnea, and cyanosis 26 d after permanent pacemaker implantation.

History of present illness

The patient had received permanent pacemaker implantation with an active fixation mechanism (a helical screw lead) and a dual chamber pacing mechanism (DDD) for progressive worsening of bradycardia 26 d before the index presentation. The patient had also undergone emergency laparotomy and enterectomy for closed-loop intestinal obstruction caused by left groin hernia 6 d previously, and was then transferred to the intensive care unit (ICU) to receive critical care until the onset of chest pain and dyspnea. On ICU admission, a chest X-ray revealed a mildly enlarged heart but proper positioning of both pacemaker leads (**Figure 1**). The electrocardiogram (ECG) revealed atrial fibrillation but satisfactory sensing and pacing of the pacemaker (pacing ventricular rate of 60 bpm).

History of past illness

She had a 10-year history of poorly controlled hypertension and a 1-year history of bradycardia and atrial fibrillation untreated with oral anticoagulants.

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Personal and family history

The patient was retired without a history of smoking, drinking, and drug abuse.

Physical examination

On physical examination, the patient was pale, restless, and in acute respiratory distress. Examination of vital signs revealed hypotension (88/60 mmHg) with infusion of noradrenaline ($1.4 \mu\text{g/kg} \times \text{min}$) as measured by an arterial catheter, elevated respiratory rate (38 breaths/min), and severe dyspnea with decreased oxygen saturation as measured by pulse finger oximetry (SpO_2 85% on room air), but a normal pacing heart rate of 60 bpm. Given the patient's recent abdominal surgery, acute abdomen, such as gut perforation, gut necrosis, and septic shock were initially suspected. However, there was no distension and tenderness with rebound on abdominal examination. Cardiovascular examination revealed remarkable jugular venous distention and distant heart sounds without murmurs. These findings indicated acute hypoxic respiratory failure and circulatory shock. In addition, cardiac tamponade was suspected given the jugular venous distention and hypotension.

Laboratory examinations

Laboratory studies prior to and after the onset of chest pain and dyspnea were obtained and are summarized in Table 1. After symptom onset, blood analysis revealed leukocytosis (white cell count $25.85 \times 10^9/\text{L}$), decreased hemoglobin (103 g/L), normal platelet count ($144 \times 10^9/\text{L}$), significantly elevated alanine aminotransferase (2840 IU/L) and aspartate aminotransferase (9650 IU/L), and slightly elevated serum creatinine (1.14 mg/dL), indicating acute kidney injury. Blood plasma coagulation tests revealed prolonged activated partial thromboplastin time (36.9 s) and prothrombin time (27.2 s). In addition, cardiac injury markers were elevated, including hypersensitive troponin T

(46.65 ng/L) and myohemoglobin (201 ng/mL). Arterial blood gas measurements while receiving an inspiratory oxygen concentrations (FiO_2) of 30% prior to symptom onset and while receiving 50% FiO_2 after onset by high-flow nasal cannula, respectively, were as follows: pH, 7.29 and 7.12; PaCO_2 , 61 and 38.8 mmHg; PaO_2 , 47.6 and 36.3 mmHg; lactate, 1.1 and 17 mmol/L; HCO_3^- , 25 and 11.5 mmol/L. In general, these findings suggested acute hypoxic respiratory failure, severe circulatory shock, and metabolic acidosis.

Imaging examinations

CT scan was not obtained due to the critical status of the patient. Urgent POCUS using a portable machine (Mindray M9, Shenzhen, China) to assess hemodynamic status at the bedside revealed a large volume of pericardial effusion associated with diastolic collapse of the RV free wall, supporting a diagnosis of cardiac tamponade. The patient then received emergency percutaneous pericardiocentesis with POCUS guidance. Briefly, a pig-tail catheter was successfully implanted into the pericardial sac at the cardiac apex and drainage yielded a large amount (600 mL) of bloody pericardial effusion within half an hour. Drainage markedly improved the dyspnea and increased blood pressure (140/71 mmHg) with decreased infusion of noradrenaline ($1.1 \mu\text{g}/\text{kg} \times \text{min}$). However, there was still ongoing pericardial bleeding with an accumulative total of 970 mL within 7 h after the initial drainage, strongly suggesting life-threatening active bleeding in the pericardial cavity.

An urgent ultrasonography consultation was obtained and cardiac POCUS was conducted by an experienced ultrasonographer at the bedside. This examination revealed elevated left ventricular ejection fraction (62%) and roughly normal-sized heart chambers. Given the recent history of cardiac pacemaker implantation, lead-related complications were suspected and we focused subsequent imaging investigations on the pacemaker lead in the RV. The parasternal four-chamber view showed mild pericardial effusion and the outline of a pacemaker lead projecting across the RV toward the cardiac apex (Figure 2A). A nonstandard RV view precisely at the cardiac

apex showed pericardial effusion and the lead tip penetrating the RV wall at the apex (Figure 2B), an imaging manifestation we termed the “bow-and-arrow” sign (Figure 3) as the RV wall formed the outline of a bow, the lead in the ventricular chamber appeared as an arrow shaft, and the lead tip associated with a hematoma at the perforation resembled an arrow head. Other localized and zoomed views demonstrated that the lead tip penetrated into the free pericardial space, confirming the diagnosis of lead perforation (Figure 2C and D).

FINAL DIAGNOSIS

Lead-induced RV apex perforation associated with active pericardial bleeding and pericardial tamponade was diagnosed based on the findings of POCUS.

TREATMENT

The time-line in Figure 4 illustrates the clinical course of the patient. Urgent off-pump cardiac surgery was conducted, and revealed a large hematoma on the RV surface at the cardiac apex near the interventricular septum. After removal of the hematoma, the tip of the cardiac pacemaker lead was observed on the surface of the RV associated with ongoing bleeding. Thus, lead-induced heart perforation was definitively diagnosed. The perforation was repaired by the mattress suture method, and the chest was closed after confirming the absence of residual bleeding. During the operation, blood pressure fluctuated between 128/85 and 108/65 mmHg with infusion of norepinephrine ($1.3\text{--}2.0\text{ }\mu\text{g/kg} \times \text{min}$). After the operation, the patient presented with refractory circulatory shock as evidenced by decreased blood pressure (75/41 mmHg) during infusion of high-dose norepinephrine ($2.2\text{ }\mu\text{g/kg} \times \text{min}$), as well as hypothermia ($35.5\text{ }^{\circ}\text{C}$), coma, and anuria. The patient received blood transfusion, fluid resuscitation, continuous renal replacement therapy (CRRT), and advanced life support treatment including invasive mechanical ventilation and cardiac inotropic support with venous pumped milrinone ($0.5\text{ }\mu\text{g/kg} \times \text{min}$).

OUTCOME AND FOLLOW-UP

On the second postoperative day, the patient developed recurrent ventricular tachycardia, fibrillation, and cardiac arrest. Cardiac pulmonary resuscitation (CPR) was attempted and both intravenous epinephrine and an antiarrhythmic drug (lidocaine) were administered; however, the patient died from shock and multiple organ failure syndrome (MODS).

DISCUSSION

More than one million permanent pacemaker implantation procedures are performed annually across the globe, of which approximately 1% result in lead-induced heart perforation^[8]. Lead perforation may involve numerous structures, including right atrium, interventricular septum, and tricuspid valve, but RV apex perforation is the most common site^[9], as exemplified by the current case. The onset of cardiac perforation may be acute (within one day of implantation), subacute (within 30 d), or chronic (more than 30 d after implantation)^[5]. Predicators of lead-induced cardiac perforation include steroid use for 7 d prior to implant, older age, female sex, BMI < 20, longer fluoroscopy time, ventricular lead in an apical position, temporary pacemaker placement, and use of helical screw ventricular leads^[10]. In this patient, female sex, older age, a helical screw lead tip, and an apical lead position may have increased vulnerability to lead perforation.

Most acute cardiac perforations by pacemaker lead occur soon after implantation and are relatively easy to detect by routine postoperative chest radiography and fluoroscopy. However, the diagnoses of subacute and chronic perforations remain a challenge due to an insidious or a sudden onset and a spectrum of heterogenous clinical presentations^[11-13]. As demonstrated by the current case, the classical manifestations of lead-induced heart perforation include chest pain, dyspnea, and hypotension caused by pericardial effusion and cardiac tamponade. Chest pain is the most frequent symptom of lead perforation, but numerous other presentations are reported, such as hiccups, dizziness, syncope, pneumopericardium, hemothorax, and pneumothorax ^[4,11,13,14]. This

heterogeneity of symptom presentation reflects not only variation in intra-cardiac perforation site, but also possible lung, liver, or chest wall perforation. Moreover, these symptoms alone do not distinguish heart perforation by lead from acute pulmonary embolism, diaphragmatic stimulation, and myocardial infarction^[12,15]. In addition, many asymptomatic cardiac perforation cases with or without malfunction of pacing have been reported^[16]. Given this heterogeneity of presentations, lead perforation should be included in the differential diagnosis when patients with a history of pacemaker therapy present with any of the aforementioned symptoms regardless of time since implantation.

To date, there is no consensus on the optimal diagnostic approach for lead perforation^[5]. For suspected cases, chest CT scan, chest radiography, and transthoracic echocardiography (TTE) are usually recommended. Although chest radiography is conducted routinely to assess lead perforation, accuracy is low according to a recent report^[5]. Alternatively, TTE can detect pericardial effusion and visualize the pacemaker lead within cardiac chambers on several imaging planes with documented accuracy of 82.7%^[17]. However, TTE is limited in some cases by a poor acoustic window and artifacts produced by lead reverberations^[9]. Also, transesophageal echocardiography (TEE) can provide better visualization of the entire lead path in the cardiac chambers^[18]. Recent studies has reported that chest CT scan, especially ECG-gated contrast-enhanced cardiac CT, has greater diagnostic accuracy (> 90%) than either TTE or chest radiography^[5,17]. Additionally, three-dimensional (3-D) reconstruction by cardiac CT has been reported helpful for diagnosis of lead perforation^[19]. In our case, however, CT scan was not obtained due to unstable hemodynamic status.

POCUS is among the most important recent developments for the management of critically ill patients. A recommended approach to goal-directed POCUS for rapidly identifying the causes of respiratory failure and circulatory shock is to assess cardiac function first^[20]. Therefore, POCUS was performed at the bedside using a portable device, and revealed cardiac tamponade and a pacemaker lead penetrating the RV apex. We summarize the main steps in the diagnosis of lead-induced RV apex perforation by

POCUS in **Figure 5**. Most RV apex perforation cases result in pericardial effusion, which is detected using our diagnostic protocol in the first step. However, the operator should recognize that pericardial effusion may be absent in some cases with RV apex perforation by lead, and that lead perforation involving other intra-cardiac structures rather than the free wall of the heart chambers usually does not lead to pericardial effusion^[21]. The position and orientation of the lead within the cardiac chambers are then visualized in the parasternal or apical four-chamber view. Subsequently, the operator precisely adjusts the transducer at the cardiac apex to capture the lead penetrating the ventricular wall. The final diagnosis is reached based in part on the appearance of a “bow-and-arrow” sign (**Figure 3**) consisting of the curved outline of the ventricular wall (the bow), the lead shaft (arrow shaft), and the lead tip (arrow head). Finally, local and zoomed imaging confirms lead perforation by identifying the lead tip penetrating through the ventricular wall and into the free pericardial space.

A systematic English language literature search of PubMed and China National Knowledge Infrastructure (CNKI) was conducted for the period 2002–2022 using key words “heart perforation”, “cardiac perforation”, “pacemaker lead”, and “ultrasound” with reasonable Boolean connectors to identify previous cases of RV apex perforation by lead diagnosed using ultrasound. Eventually a total of 22 cases ^[18,22-42] diagnosed by ultrasound with definite imaging information were identified. These cases are summarized in **Table 2**. Most cases with RV apex perforation included three ultrasound manifestations, pericardial effusion, passage of the lead through the RV wall, and the tip reaching the free pericardial space. The right ventricular apex is the free wall of the right ventricle, so pericardial effusion occurred in most of these cases. However, absence of pericardial effusion was also reported in 4 cases of RV apex perforation^[18,26,27,30]. In addition, these cases reported distinct features such as lead mimicking a “spear”^[22], the lead entering and retracting from the RV with each cardiac movement^[29], and discontinuation of the RV wall^[35]. 3-D ultrasound^[23,24,36] and TEE^[18] may facilitate the diagnosis of lead perforation by improving the visualization of the lead and intra-cardiac structures. Overall, these reports indicate that RV apex

perforation by lead presents a very wide range of imaging variations depending on the sonographic features of the perforated myocardium, the route and orientation of the lead, the ultrasound system used, and operator skill. Knowledge of these representative imaging features as well as the “bow-and-arrow” sign described in this report can help clinicians rapidly reach a diagnosis of lead perforation.

The optimal management of lead perforation is also a matter of ongoing debate. Asymptomatic cases without pacing malfunction may not require special management^[16]. However, an expert consensus statement recommends that lead extraction should be performed in cases with significant manifestations such as pericardial bleeding, chest pain, and device malfunction^[43]. Surgical repair and percutaneous transvenous lead extraction (TLE) are the two major approaches to remove the culprit lead. A number of recent reports have suggested that surgical repair is useful and reliable, especially for lead perforation beyond the pericardial sac, delayed perforation with severe bleeding, chronically implanted devices, and when there is suspicion of adjacent organ injuries^[39,44]. There is also mounting evidence that TLE is an efficacious, safe, and convenient tool to manage most patients who are hemodynamically stable without visceral organ injury^[5]. In specific cases, TLE may be superior to surgery for reducing length of hospital stay, medical costs, morbidity, and mortality, but requires trained operators to achieve satisfactory outcome^[45,46]. Nevertheless, the case patient required thoracotomy to repair the heart perforation given the severe pericardial bleeding.

The strengths of this study are as follows: (1) we present the complete clinical course of subacute lead perforation rapidly diagnosed by POCUS; (2) we propose a step-wise diagnostic approach using POCUS; (3) we identify a “bow-and-arrow” sign on POCUS that facilitates the rapid diagnosis of RV apex perforation by lead. This study also has several limitations. First, the diagnostic efficacy of POCUS compared to CT examination and other imaging modalities must be supported by a retrospective observational trial or more ideally by a randomized clinical trial. Second, the step-wise approach and the “bow-and-arrow” sign on POCUS have not been validated in other cases.

CONCLUSION

Delayed heart perforation by pacemaker lead is difficult to diagnose and manage due to sudden onset, complex clinical presentation, and the potential for life-threatening cardiac tamponade and arrest. POCUS is a useful tool for the real-time evaluation of suspected lead perforation, especially for critically ill patients with severe respiratory failure and circulatory shock. A “bow-and-arrow” sign on POCUS can be easily recognized that facilitates the rapid diagnosis of lead perforation. A step-wise POCUS training program to improve detection of this “bow-and-arrow” sign and other ultrasound manifestations may enhance diagnostic speed and accuracy for improved clinical outcomes of lead perforation.

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