

Clinical Image

A case of delayed right ventricle perforation following permanent pacemaker insertion complicated with sepsis

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Case

A 47-year-old Bangladeshi gentleman, who was a known case of DM, HTN, IHD, was admitted on 21st December, 2023 through Emergency under the department of Critical Care Medicine with the complaints of fever for 4 days which was high grade intermittent with highest recorded temperature 103^o F. He also complained of shortness of breath for 2 days which became severe on the day of admission. On query he gave history of NSTEMI with high nodal AV block with recurrent syncopal attack two months back for which he underwent dual chamber PPM insertion 25 days prior to ICU admission.

On examination patient was conscious and oriented with no focal neurological deficit, severely tachypnoeic with respiratory rate 35/min, SpO₂ 80% with 15 lit/min O₂ inhalation via non rebreathing mask, Non recordable BP with tachycardia (120-130/min, feeble, regular), temperature 102^o F, on auscultation there was bilateral crepitation up to second intercostal space of both sides of chest. As part of immediate resuscitation he was intubated and put on MV with AC/VC mode with high FiO₂ and PEEP. Immediately high dose of vasopressor were started after establishing CV line. Immediate bed side CXR revealed suspected one of the leads of PPM displaced piercing the wall of right ventricle reaching upto the left diaphragm (figure 1) with cardiomegaly and widening of mediastinum.

In view of that, immediate bed side echocardiography was done which revealed pericardial effusion measuring about 7mm anteriorly, 10 mm inferiorly and 12 mm postero-laterally with no features of tamponade. RV lead of the PPM

was seen perforating the wall of right ventricle and tip of the pacemaker lead was in the pericardial space. EF was 27%.

Subsequent CT chest also revealed RV lead perforating the wall of right ventricle near the ventricular apex and tip of the lead in the pericardium creating “star artifact” (figure 3)

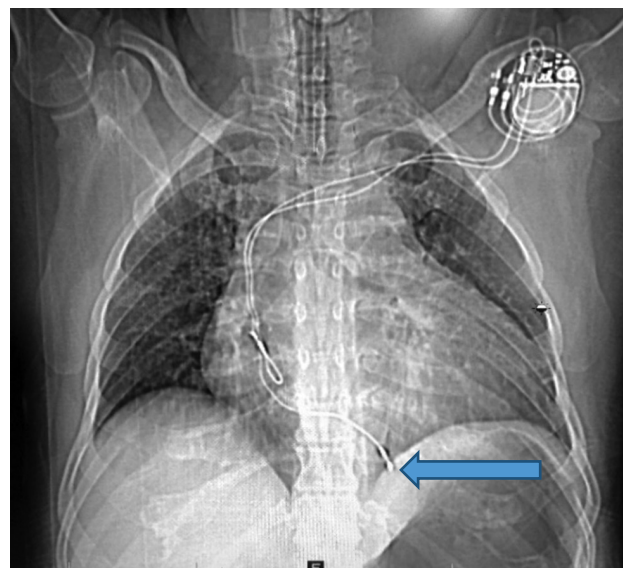


Figure 1: CXR of patient showing one of PPM lead piercing wall of right ventricle (blue arrow marking) with cardiomegaly and wide mediastinum.

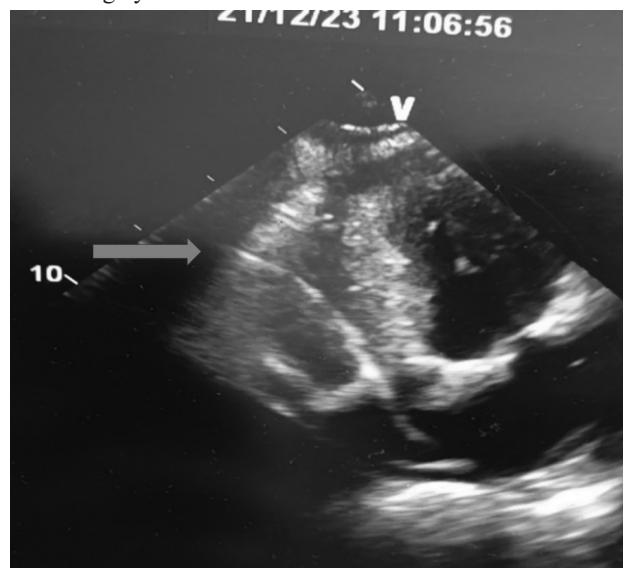


Figure 2: Echocardiography of patient showing PPM lead piercing wall of right ventricle (arrow marking)

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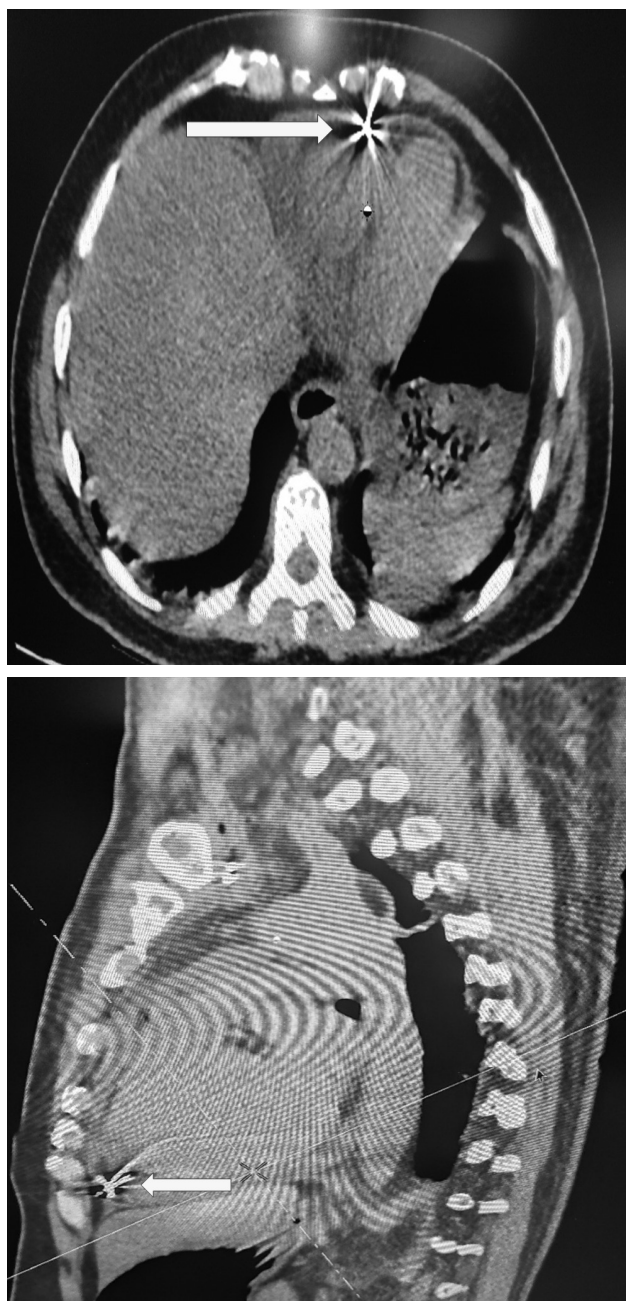


Figure 3: CT chest showing RV lead perforating the wall of right ventricle near the ventricular apex and tip of the lead in the pericardium creating “star artifact” (arrow)

His blood investigations revealed severe sepsis (procalcitonin 75, CRP 470) with DIC (platelet count 32000/cumm, D-Dimer 48500, FDP: high), AKI (s. creatinine 3.61, B. urea 124), and NSTEMI with ALVF (hsTropI 6232, NT-proBNP: 20195). Patient was treated accordingly with broad spectrum antibiotics covering gram positive (MRSA) organism, fluid therapy, antipyretic, vasopressor and mechanical ventilation with close monitoring of vitals, intake/output and frequent bedside echocardiography monitoring for deterioration of pericardial effusion and development of any feature of tamponade. Consultation from cardiology and cardiac surgery was also taken and followed accordingly. Unfortunately few

hours later of ICU admission patient developed cardiac arrest which didn't revert even after resuscitation for 30 minutes according to ACLS protocol. His final blood culture report revealed growth of staphylococcus aureus which fall under MRSA group.

Discussion:

Pacemakers are electronic devices that stimulate the heart with electrical impulses to maintain or restore a functional heartbeat¹. These devices are used for temporary as well as permanent management options for various cardiac conduction abnormalities and arrhythmias. Complication rates of implanting such devices vary between 3% and 7.5%²⁻⁴. The most frequently occurring and potentially significant acute complications include pneumothorax (1.9%–3.7%), lead displacement (0.5%–4.8%), myocardial perforation (0.37%–1%) and infectious complications related to pacemaker pocket infection or endocarditis²⁻⁵. Right ventricular perforation is a rare but serious complication of permanent pacemaker with a reported prevalence rate of 0.1–6%⁶. It can affect most parts of the heart that come in contact with a lead but the majority of perforations occur through the right ventricular apex, primarily because this remains a common site of deployment of the ventricular lead, and the myocardial wall is thinner here than at other common pacing sites such as the septum and right ventricular outflow tract⁷. It is diagnosed when at least the tip of a passive fixation lead or the screw of an active fixation lead passes through the myocardium and extends into the pericardial cavity⁸. Its prevalence might be underestimated because the majority of patients with sub-acute or delayed cardiac perforation remain asymptomatic⁹. Risk factors predisposing to lead perforation include lead characteristics (stiffness, tip diameter, and active-fixation mechanism), endocardial location (right ventricular apex), operator experience, and patient characteristics (older age, female, low body weight, and concurrent use of systemic steroids)¹⁰.

Perforation can occur acutely (within 24 h after implantation), sub-acutely (between 24 h and one month after implantation) and chronically (occurring more than one month after implantation)¹¹. Acute perforations often present with chest pain, dyspnea and signs of pericardial effusion or tamponade. The most common presentation of a delayed perforation is hemopericardium (with or without cardiac tamponade), pericarditis, diaphragm or chest wall muscle stimulation, loss of capture and pneumothorax¹². The presentation of patients with cardiac perforations can be variable, therefore a high index of suspicion needs to be maintained for a rapid diagnosis. Patients may present with chest pain, shortness of breath, presyncope or syncope¹³. However our case presented with features of hemopericardium without tamponade but complicated with sepsis and multiorgan failure.

Different modalities of imaging are used to diagnose device related complications. A diagnosis of ventricular lead perforation may be confirmed with chest radiography, fluoroscopy, echocardiography, or chest CT scan. When the lead migrates too far from the heart, chest radiography and fluoroscopy can easily detect the problem. However, in cases

with minimal perforation of the heart, these tests are often non-diagnostic¹⁴. A simple chest X-ray (preferably postero-anterior and lateral view) should be the starting point in the diagnosis of cardiac perforation. An enlarged cardiac silhouette, pneumothorax, pleural effusion can also be supportive in making the diagnosis. Usually the lead tip should be within 3 mm of cardiac border; if the tip extends outside the cardiac boarder a perforation should be suspected¹³, which is in our case. However we could not perform the lateral or PA view chest X ray as our patient was intubated just after admission, hence we performed the AP view. Two and three dimensional echocardiography can also be helpful for the detection of pacing wire perforation when the path of the wire is visualized in the spatial orientation of the echocardiography beam¹⁵. Echocardiography can detect pericardial fluid, cardiac tamponade, malposition as well as extension through the myocardium. On occasions perforations may not cause any detectable pericardial effusion¹³. Non-contrast cardiac CT provides visualization of the heart and can be the most important diagnostic method to confirm myocardial perforation not detected by other modalities. It can evaluate the intracardiac lead position and even small amounts of pericardial effusion. However, the metal edges cause streaks surrounding the electrode tip, known as the “star artifact”, which affect the quality of the image and make it difficult to detect the lead tip¹⁴. However regarding MRI, with new generations of MRI-conditional devices, this imaging modality, with fewer lead artifacts compared with CT, may become the gold standard for the detection of lead perforation in the future¹⁶.

The optimal management of lead perforation is still unclear¹⁴. The management depends upon the clinical presentation, pericardial effusion, and hemodynamic status¹⁷. Acutely the pacing lead can be repositioned and the patient is monitored with serial echocardiography¹³. Subacute or delayed perforations are dealt with case by case basis. If the perforation is asymptomatic or not associated with pacing/sensing malfunction or mediastinal bleeding or if the risk of lead removal outweighs the non-removal, the lead can be left in place¹³. In the case of symptomatic heart perforation or lead malfunction without significant pericardial effusion, the lead may be removed manually by direct traction with the aid of a regular stylet, after retraction of the active fixation screw. This procedure should be performed in a hybrid operating room under trans-esophageal echocardiography (TEE) observation with a cardiac surgical team on standby. The leads may be repositioned to a new site¹⁸. Cardiac tamponade can be successfully managed with closed pericardiocentesis and a pericardial drain in place. Surgical intervention seems to be the treatment of choice in the case of other visceral injuries, hemodynamic instability, rapid progression of pericardial effusion, or if closed pericardiocentesis fails⁷. However treatment for other symptomatic complaints should run side by side. In conclusion, although uncommon, cardiac perforation is a serious complication following pacemaker implantation and our case demonstrates an extreme presentation of delayed perforation, serving as a reminder to always consider delayed

lead perforation as a differential for pericardial effusion in a post PPM insertion patient.

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