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DEVICE ROUNDS

Hip to pieces, torpid heart?

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1 | CASE PRESENTATION

A 87-year old patient received a dual chamber pacemaker because of third degree atrioventricular block after surgical aortic valve replacement. Passive fixation pacemaker leads were used. The postoperative chest X ray is shown in Figure 1A. Shortly after the implantation, the patient complained of chest pain. A transthoracic echocardiography showed a large pericardial effusion for which pericardiocentesis was performed with aspiration of a serosanguinolent fluid. His condition remained stable and he was discharged a few days later. Yearly pacemaker follow-up showed normal device function. Three years after pacemaker implantation, he was admitted to the hospital after an accidental fall with hip fracture for which hip surgery was performed. In the postoperative period, the patient started complaining of dizziness and chest pain. He was hypotensive and had a regular heartbeat of 35 bpm. The 12-lead ECG is shown in Figure 2. His blood chemistry showed a normal kidney function, normal electrolytes and normal troponin levels. A device analysis was performed. His pacemaker was programmed in the DDD-mode with a lower rate of 60 bpm. Atrial and right ventricular (RV) sensing and impedance values were stable and within normal range. The atrial threshold was normal, the RV threshold however had risen to 5 V at a pulse duration of 1 ms in both the uni- and bipolar pacing configuration. Battery status was normal, with a remaining battery life of approximately 4 years. There were no ventricular high rate episodes. A chest X ray is shown in Figure 1B.

Questions:

- What is your electrocardiographic diagnosis?
- What is the most likely cause of this abnormality?
- How should this problem be solved?

TABLE 1 Causes of late loss of capture

- Lead failure (e.g., conductor fracture or insulation breach)
- Pacemaker battery end of life
- Medication induced alterations of capture threshold (e.g., flecainide)
- Electrolyte abnormalities (e.g., hyperkalemia, acidosis)
- Lead tip fibrosis
- Cardiomyopathy (e.g., myocardial infarction)
- Lead displacement +/- cardiac perforation
- Programming error

2 | DISCUSSION

The 12-lead ECG shows correct atrial sensing and pacing. However, there is consistent loss of RV capture with absence of ventricular depolarization following the RV pacing spike. There is an intrinsic ventricular escape rhythm at 35 bpm.

Pacing capture is confirmed by documenting a stable relationship between the pacing stimulus and depolarization of the cardiac chamber. In our patient, as there is no such relationship between RV pacing stimulus and cardiac depolarization, there is thus loss of capture. This is a potentially life-threatening situation in a pacemaker-dependent patient.

There are many causes, both cardiac and noncardiac, for loss of capture. It can occur early (within the first 6 weeks after device implantation) or late (after this period of time).[1–3]

Table 1 shows an overview of the most common causes of threshold rises occurring late (e.g., >6 weeks) after implantation.

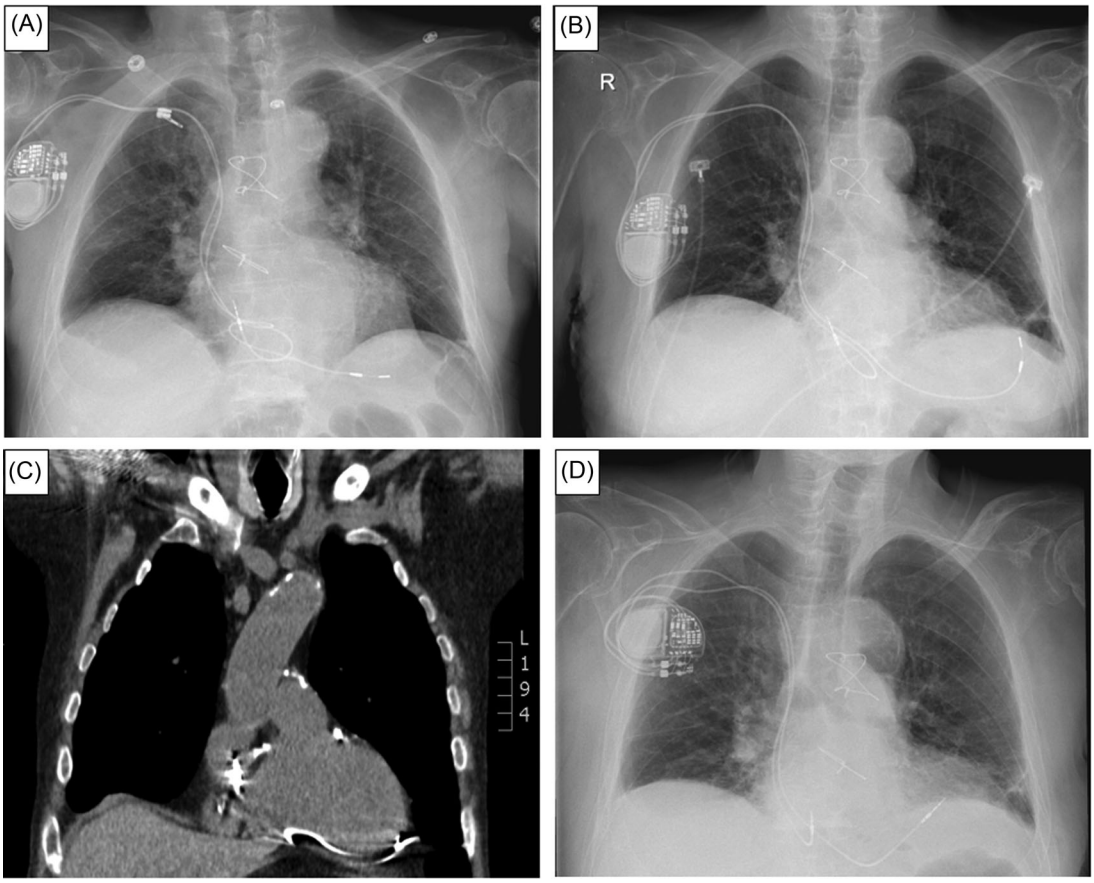

FIGURE 1 (A) Chest X ray after implantation of a dual chamber pacemaker. (B) Chest X ray 3 years after pacemaker implantation. (C) CT-scan 3 years after pacemaker implantation. (D) Chest X ray after extraction of the passive fixation RV lead and reimplantation of an active fixation RV lead in a septal position

In our patient, RV sensing and lead impedance were stable and within normal range and there were no ventricular high rate episodes, rendering the possibility of RV lead failure less likely.

Battery life was within normal range, excluding battery depletion as a reason for noncapture (failure to generate sufficient energy).

Clinical history may reveal initiation of new medication. Notably, Vaughan–Williams class I antiarrhythmic agents such as flecainide have been shown to increase pacing thresholds.[4]

The presence of renal failure and abnormalities in serum electrolytes such as acidosis or hyperkalemia should always be ruled out. Especially, when the potassium level reaches 7 mmol/L or higher, the pacing threshold has been shown to increase significantly.[5]

Fibrosis at the site of the lead tip fixation can cause a substantial rise in threshold. Nowadays, steroid-eluting lead tips have reduced the occurrence of fibrosis. A history of recent procedures such as radiofrequency ablation, electrical cardioversion, MRI scan or radiation therapy may point toward secondary lead or device abnormality.

Also, myocardial infarction or cardiomyopathy can result in a late rise in pacing threshold. A chest X ray is mandatory to diagnose lead dislodgement and may identify lead fracture. The chest X ray of our patient showed a clear displacement of the RV pacing lead compared to its position immediately after pacemaker implantation 3 years earlier. An additional computed tomography (CT)-scan confirmed the lead displacement, and also showed perforation of the lead tip into the thoracic cavity (Figure 1C). Therefore, a diagnosis of loss of capture due to late lead displacement and cardiac perforation was withheld.

Late lead displacement is rare (0.1%—0.8% of pacemaker implantations).[2,6] The clinical presentation is diverse: patients can remain asymptomatic or present with chest pain, dyspnea, syncope, abdominal pain, hiccups, cardiogenic shock due to haemopericardium, pleural effusion, and device pacing and sensing abnormalities.[1–3,6] Risk factors associated with late cardiac perforation include concomitant temporary transvenous pacing at the time of implantation, steroid use, older age (>80 years), active fixation leads, BMI < 20 kg/m^2, anticoagulation therapy and female gender.[1–3,6] Diagnosis of lead migration and perforation can usually be made on a chest X-ray or, when in doubt, on a CT-scan of the heart or thorax. Echocardiography is useful to quantify a pericardial effusion and exclude tamponade.

The optimal management of delayed lead perforation is still unclear. Treatment depends on perforation type and timing, lead fixation type, and lead tip position. For an asymptomatic, incidentally detected, chronically perforated lead that is otherwise functioning well, it is suggested that extraction is not necessary.[2,6] When lead extraction is performed, the lead can be removed using dedicated extraction tools such

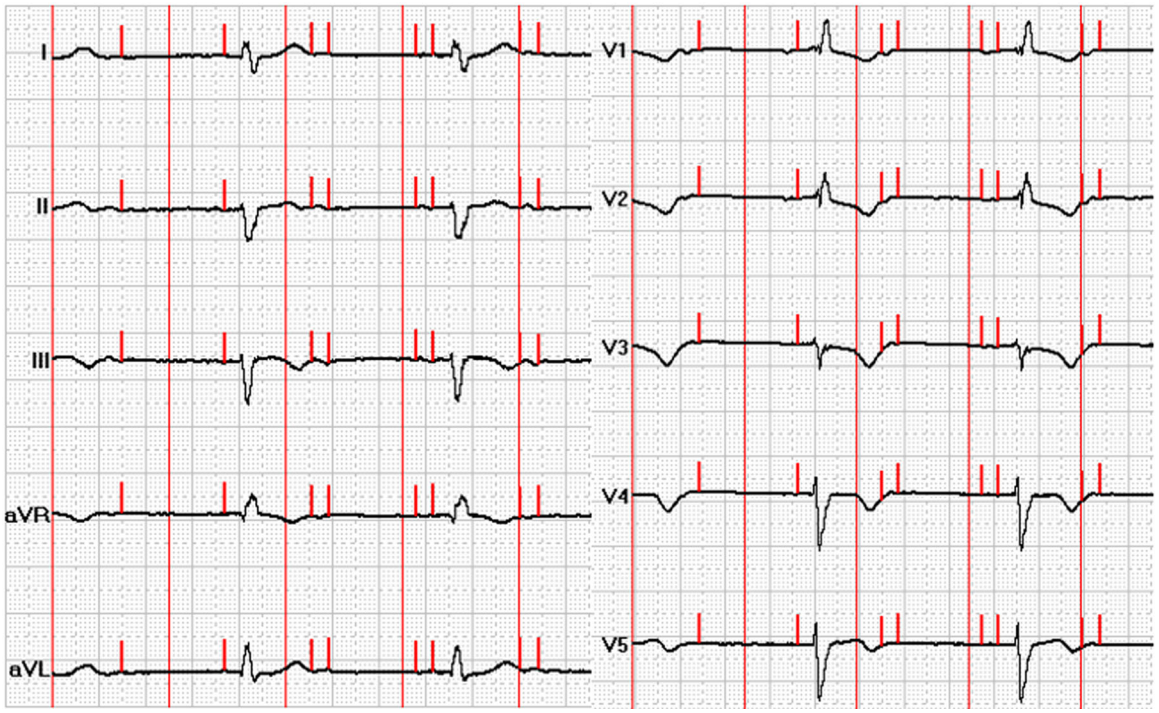

FIGURE 2 12 lead ECG (lead aVF and V6 not shown) after hip surgery. (Paper speed 25 mm/s) [Color figure can be viewed at wileyonlinelibrary.com]

as locking stylets, telescoping sheaths, femoral snares, mechanical cutters, and laser sheaths. Depending on the estimated procedural risk this procedure can be performed in the electrophysiology (EP)-lab or in a hybrid operating room under TEE observation and with a cardiac surgeon team on standby.[2,6]

In this case, we hypothesize that there must have occurred a (micro)-perforation during initial pacemaker implantation, given the occurrence of a pericardial effusion postoperatively. We assume that 3 years later due to fall, the RV lead displaced and perforated this weak point in the RV. The pacemaker was temporarily programmed at a maximum RV output (8 V@1 ms) and the patient was scheduled for transvenous RV lead extraction in the EP-lab. We performed a successful transvenous extraction of the RV lead without complications and implanted a new, active fixation RV lead in a septal position (Figure 1D).

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CONFLICT OF INTEREST

Both authors have no conflicts of interest to declare

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