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Early displacement of an atrial lead: A fatal presentation of a relatively frequent complication

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ABSTRACT

An 81-year-old woman was referred to Parc Taulí Hospital Universitari with complete atrioventricular block and a dual-chamber pacemaker was implanted. She subsequently presented an episode of ventricular tachycardia that degenerated into ventricular fibrillation requiring cardiopulmonary resuscitation. After review of the device, as well as the electrocardiogram and chest X-ray, displacement of the atrial lead into the right ventricle was confirmed. Contrary to expectations, the ventricular event was not secondary to endocardial irritation by the displaced lead, so the hypothesis of a possible depolarization-repolarization heterogeneity as a cause of the increased arrhythmogenicity was raised.

KEYWORDS: Dual chamber pacemaker; Atrial lead displacement; Ventricular arrythmia; Sudden cardiac death.

INTRODUCTION

Pacemaker lead displacement can be defined as a change in the lead position of the lead relative to its final implant position, with the potential to result in malfunction of the pacing system and serious adverse events[1].

In this context, atrial lead displacement is often considered a minor and relatively common complication. Although, although it does not usually represent as high a morbidity as ventricular lead displacement, it does represent a significant increase in the need for reintervention and its associated complications and can even occasionally fall into fatal outcomes.

CASE REPORT

An 81-year-old female patient with no significant medical history was admitted to hospital with a diagnosis of complete atrioventricular block at a rate of 40 bpm, resulting in symptoms of shortness of breath and dizziness. A dual-chamber pacemaker (Biotronik Amvia Edge DR-T) was successfully implanted without any periprocedural complications.

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However, during the initial 24-hour period following the procedure, the patient exhibited frequent ventricular extrasystoles (VEs), sometimes triggering episodes of non-sustained ventricular tachycardia (NSVT). Since she was clinically asymptomatic, the on-call cardiologist was not notified. However, early in the morning, the patient experienced an episode of sustained ventricular arrythmia (VA), which required cardiopulmonary resuscitation, subsequent intubation, and transfer to the intensive care unit.

Review of the 12-lead electrocardiogram (ECG) was conducted, which revealed atrioventricular dissociation with paced ventricular rhythm alternating with coupled VEs. Further interrogation of the device revealed various episodes of NSVT in the preceding hours, as well as the episode of sustained VT that degenerated into ventricular fibrillation causing cardiac arrest. All episodes were triggered by VE that showed greater precocity in the ventricular electrogram (EGM) channel compared to the atrial EGM channel. Intracardiac recordings demonstrated atrial pacing with probable ventricular capture, showing simultaneous depolarization detected on the ventricular EGM channel during the safety window. Conversely, ventricular pacing at the end of the safety window showed no capture due to physiological refractoriness (Fig. 1).

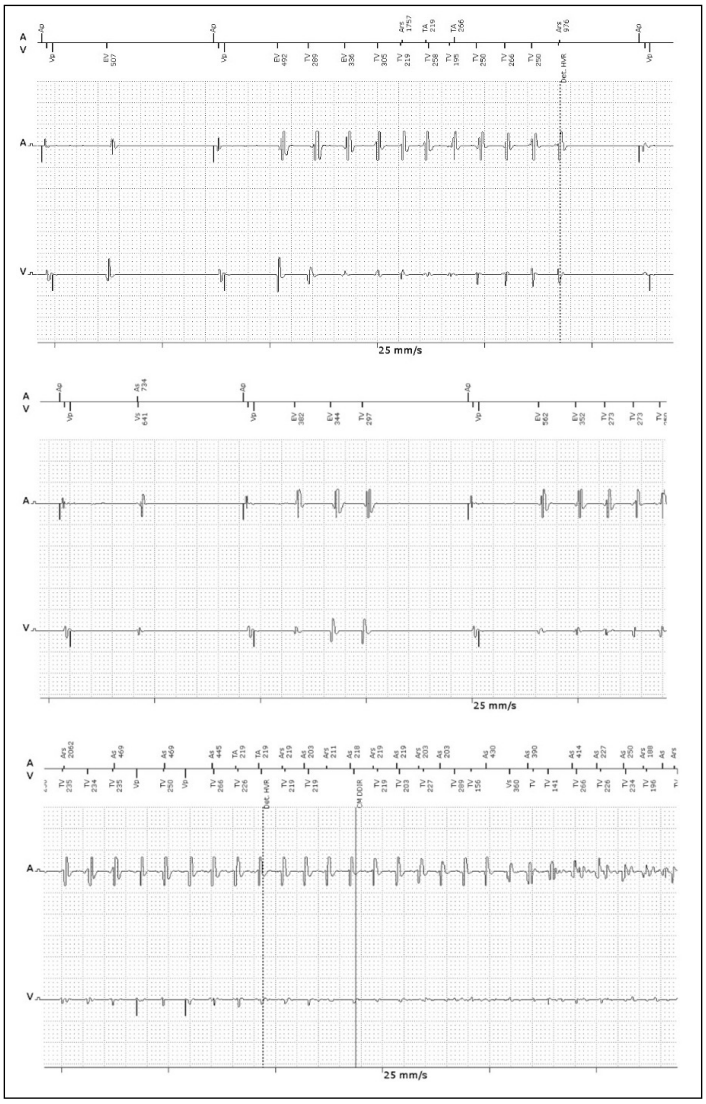

Figure 1. Different intracardiac electrogram recordings suggesting atrial lead displacement. (a) Atrial pacing produces depolarization on the ventricular electrogram channel. Ventricular extrasystole (VE) is frequently observed, occasionally triggering non-sustained ventricular tachycardia (VT); (b) Onset of a sustained VT episode triggered by VE; (c) Previous VT degenerates into ventricular fibrillation.
Fonte: Elaborated by the authors.

These tracings raised suspicion of atrial lead displacement, with the presence of both leads in the ventricle. Consistent with this diagnosis, pacing on both AAI and VVI showed ventricular capture on the ECG (Fig. 2).

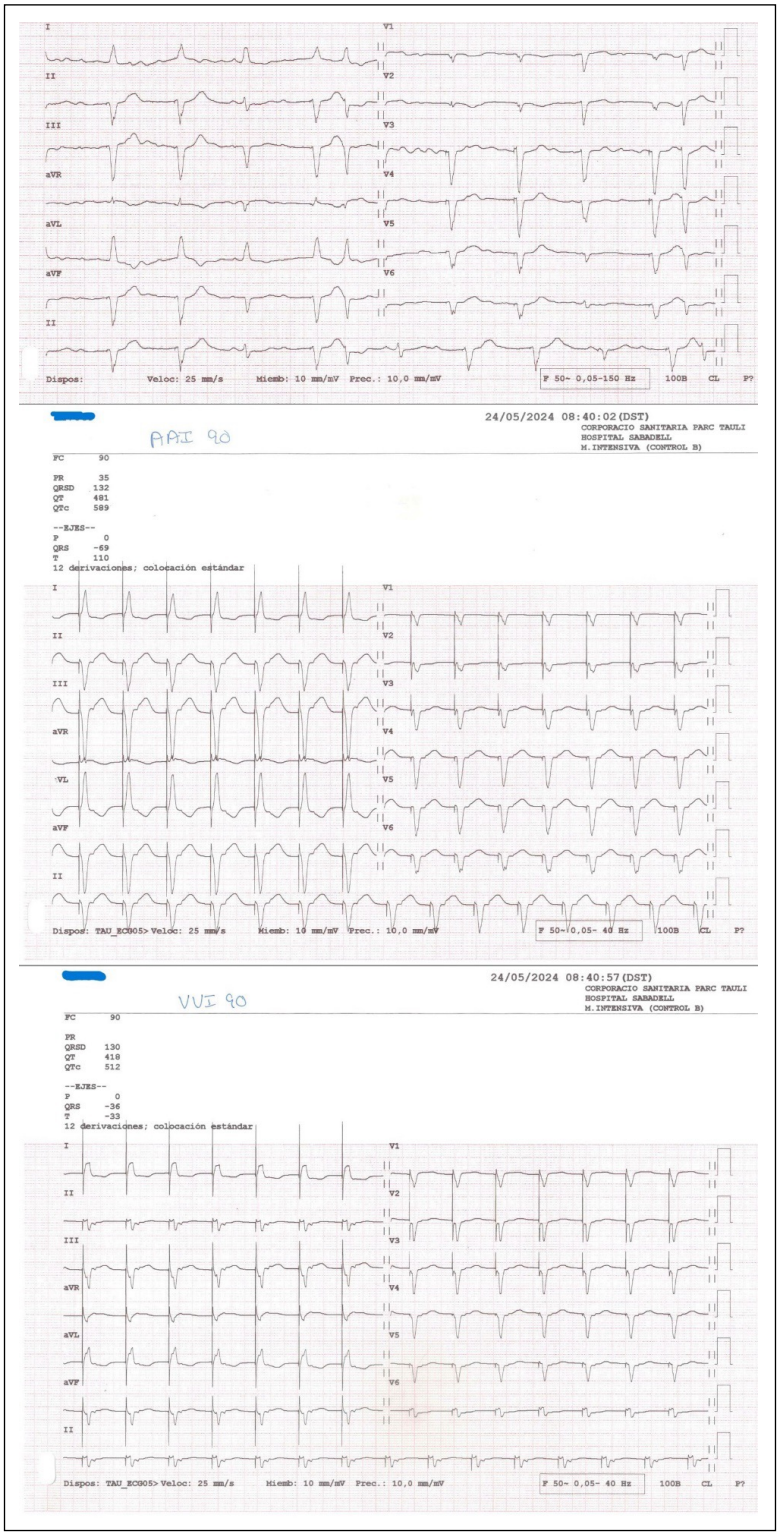

Figure 2. Electrocardiogram (ECG) after atrial lead displacement. (a) Baseline ECG showing AV dissociation; (b) AAI mode pacing shows ventricular capture on ECG; (c) VVI mode pacing shows ventricular capture on ECG with narrower QRS.
Fonte: Elaborated by the authors.

Chest X-ray confirmed lead displacement from the right atrium into the right ventricle, showing the ventricular lead normally positioned at the level of the left bundle brunch (Fig. 3). The patient was promptly transferred to the operating room, where the lead was repositioned on the lateral atrial wall. This procedure ensured that device could resume its normal function. No further VEs or VT were observed during subsequent follow-up.

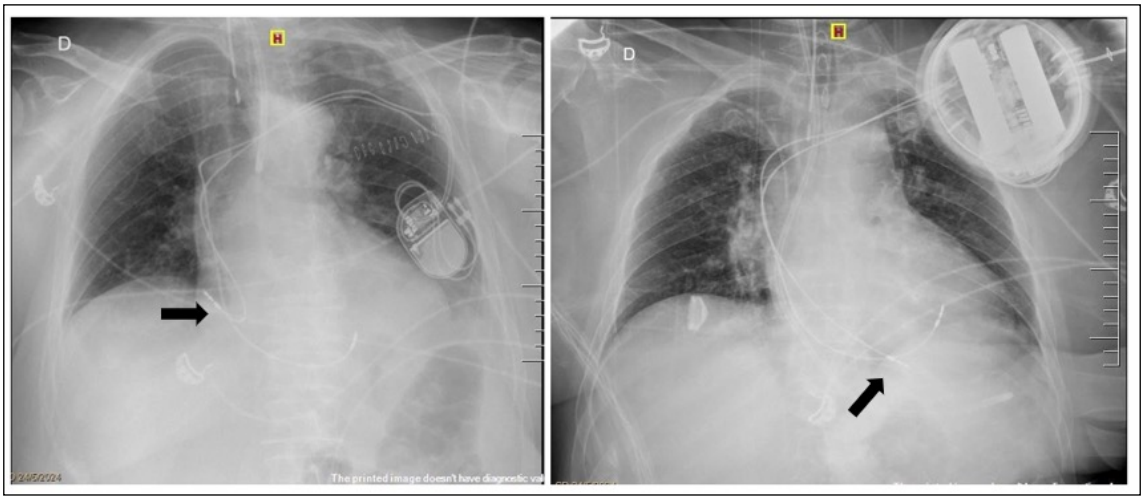

Figure 3. Anteroposterior chest X-Ray. Left: image obtained just after pacemaker implantation. The arrowhead shows the tip of the atrial lead within the right atrial appendage. Right: image obtained 24 hours later, showing the displacement of the atrial lead within the right ventricle.
Fonte: Elaborated by the authors.

DISCUSSION

Early atrial lead displacement occurs within the first six weeks post-implantation and is more prevalent than late displacement, happening in 3.8% of patients with dual-chambre devices. Moreover, it constitutes one of the most common reasons for re-intervention following pacemaker implantation[2]. The actual basis for this prevalence remains unclear; however, the current literature identifies Twiddler and Reel syndromes as the most commonly identified causes[1,3].

The ECG is usually the primary indicator of lead displacement following device implantation. On the other hand, interrogation of the device often reveals abnormal findings, sometimes with loss of capture, inadequate sensing, or impedance abnormality, or may be completely normal. However, a definitive diagnosis of lead displacement can only be established by radiography.

Atrial lead displacement subsequent to pacemaker implantation is usually clinically subtle at first, although it is capable of inducing adverse effects over time. In exceptional circumstances, it can appear early on with serious consequences for the patient. The severity of this complication is contingent on the position of the new lead and the extent to which the patient relies on the pacemaker. The most common symptom is usually dizziness, malaise or palpitations due to stimulation of the right phrenic nerve, increased atrial pacing, or even the presence of tachyarrhythmias such as irritation-induced atrial fibrillation[4]. Furthermore, in patients with sinus dysfunction but preserved atrioventricular conduction, atrial lead displacement may cause excessive unnecessary ventricular pacing, resulting in non-physiological pacing leading to heart failure[3]. Induction of VAs by displacement of the atrial lead is less common, but occasionally, if the lead falls into the ventricle, it may cause irritation and activate arrhythmogenesis or present as a paced ventricular tachycardia with the appearance of electrical alternans[5].

The present case demonstrates the induction of VEs and complex VAs in the context of atrial lead displacement. Contrary to expectations, it is not the fallen lead that produces irritation and ectopic activity, as demonstrated by the earlier activation in the ventricular EGM channel. If the displaced lead itself were to produce mechanical irritation, activation would be focal from this point, showing a sequence of activation from the atrial to the ventricular channel, as observed after atrial pacing. Furthermore, focal activation from the lead when there is mechanical irritation has been confirmed by electroanatomical mapping analysis in previously published cases[6]. In this case, it is possibly an underlying electrical condition due to continuous overexcitation by double ventricular pacing, even if the second spike is not captured. In a context of possible heterogeneous depolarization-repolarization, frequent ectopy would occur with the potential to produce R-on-T phenomenon when falling into the vulnerable period of repolarization, which in turn could lead to fatal VAs.

Although this hypothesis cannot be confirmed with the available information, it is supported by the disappearance of VE and secondary arrythmias after repositioning of the displaced lead. Conducting an invasive electrophysiological study prior to lead repositioning would have facilitated a more comprehensive understanding of the arrhythmia mechanism; however, the severity of the clinical episode precluded such an approach.

Atrial lead displacement is usually trivial, although we have seen that it can sometimes be fatal, especially when the lead dislodges into the ventricle. Therefore, it is important to detect any change in lead position before discharge by periodic interrogation of the device and performing a follow-up X-ray with posteroanterior and lateral views.

Treatment for lead displacement depends on the time elapsed since pacemaker implantation. However, other factors such as the patient's clinical condition, the type of fixation of the displaced lead, and the operational status of the generator and other implanted leads must also be considered. Surgical relocation of the displaced lead is the most effective treatment, especially at an early stage when it is still possible to reopen the pocket and manipulate the lead, as its tip has not been caught and fixed by the endocardial fibrous reaction. On the other hand, percutaneous transcatheter relocation of displaced pacemaker leads have been advocated before considering standard surgical relocation. This procedure appears easy and safe, reduces the morbidity and cost associated with surgical lead revision, and has shown good results in some specific cases[7]. In the case of late displacement, implantation of a new lead may be more appropriate in terms of risk and benefit[1].

Preventing electrode displacement is even more important than its treatment. For this, surgical implantation must be technically adequate. It is important to create a small pocket to prevent displacement of the generator and the consequent pressure on the electrode, considering subpectoral implantation in obese patients to avoid progressive displacement of the generator due to sagging of the subcutaneous tissue. On the other hand, it is important to use active fixation electrodes, especially in patients without trabeculations, and to fix their proximal end well to the muscular plane. There are also intraprocedural maneuvers such as visualizing all the electrodes during deep inspiration to assess their redundancy or evaluating the loss of capture during deep breathing or coughing that seem to reduce the risk of subsequent displacement[8].

To our knowledge, this is the only case reported in which VAs occur secondary to atrial lead displacement. Furthermore, unlike some previous publications describing mechanical arrythmias caused by continuous tissue irritation secondary to lead instability, our case does not seem to be related to this mechanism.

CONFLICT OF INTEREST

Nothing to declare.

AUTHOR CONTRIBUTIONS

Conceptualization: Caixal G; Methodology: Caixal G, Ascoeta-Ortiz MS, García-Hernando V; **Original Draft:** Caixal G; **Writing – Review and Editing:** Caixal G, Puntí J, Gusi G; **Supervision:** Martinez-Rubio A; **Final aproval:** Caixal G.

DATA AVAILABILITY STATEMENT

The data will be available upon request.

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