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ECHO and magnetic resonance imaging in a patient with high bleeding risk and ventricular perforation: a case report and literature review

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Abstract. – Myocardial perforation is a complication following pacemaker implantation that may cause cardiac tamponade.

We present an original case of myocardial lead perforation not complicated by acute cardiac tamponade.

The patient with an acute myocardial infarct had a high bleeding risk both in the acute phase of lead insertion (anticoagulant and triple platelet anti-aggregation therapy) and after few days, the percutaneous extraction lead for the double platelet antiaggregant therapy.

Torrent-Guasp's theory is considered for explaining the clinical course of patient.

Echocardiography and magnetic resonance imaging (MRI) evaluation showed a diffuse pericardial non-hemorrhagic fibrinous effusion and guide the clinical management.

Key Words:

Echocardiography, Pacing, Radiology, High bleeding risk patient, Myocardial lead perforation, Anticoagulant therapy, Antiaggregant therapy.

Case Report

A 68 year-old man was admitted to our Department for an episode of syncope associated to neurovegetative symptomatology. Patient's rate was 90 bpm in sinus with episodes of AIVR (Accelerated Idioventricular Rhythm) and Mobitz type-II 2nd-degree AV block. The blood pressure was 140/80 mmHg. ECG documented ST- segment elevation in the inferior walls with increase of myocardial necrosis markers.

Se had had an anterior myocardial infarct in the past; risk factors and familiarity for cardiovascular diseases were found in his history. His daily therapy was: aspirin, nitrate and statin.

An immediate coronary angiography showed severe coronary artery disease and the patient was submitted to primary Percutaneous Transluminal Coronary Angioplasty (PTCA) and two non drug eluting stents were implanted to the right coronary artery. Another critical lesion of the left anterior descending artery was treated with three non drug eluting stents. The onset of a complete AV block with pauses longer than three seconds required, during the procedure, a temporary pacemaker implant with passive leads fixation (tip lead Ø 2 mm). Fluoroscopy guided its insertion in the cardiac catheterization laboratory using a femoral approach.

Glycoprotein IIb-IIIa receptor inhibitor, clopidogrel, ASA and low-molecular-weight heparin (LWWH) were administered.

Echocardiography, performed the following day, revealed a low ejection fraction (EF: 35%), akinesia of the anterior wall, an aneurism tic tip and a marked hyperkinesias of the inferior one. In the right ventricle the temporary pacemaker lead was caught in the free wall, engaging in the pericardial space and protruding for beyond one cm. Minimal pericardial effusion was observed (Figure 1).

The patient didn't complain about symptoms and the electrocardiogram was normal.

Two clinical and therapeutic approaches were hypothesized:

- an urgent surgery after withdrawal of the double platelet antiaggregant therapy (clopidogrel and ASA) associated with high risk of acute and early stent thrombosis;

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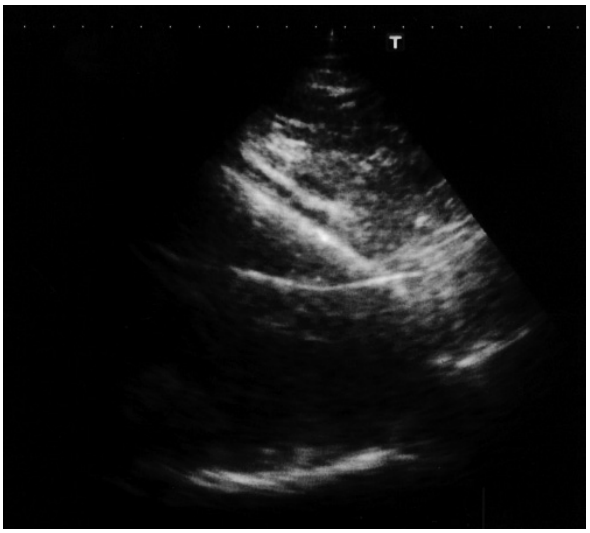

Figure 1. Transthoracic echocardiogram shows lead protrusion through right ventricular myocardium.

- a transvenous extraction that was related to an elevated risk of haemorrhage and acute heart tamponade.

We decided for a non surgical management considering the high risk of early stent thrombosis.

Electrocardiogram and Transesophageal Echocardiography (TEE)-guided temporary lead extraction in the operating theatre. The procedure was completed without complications.

Four days later, a cardiac Magnetic Resonance (CMR) exam was performed to study the right ventricular wall damage before the implantation of a permanent dual-chamber pacemaker with passive fixation leads. CMR was more sensitive to evaluate tissue characterization of myocardium and produced a better definition in comparison with ultrasound images. This exam revealed a delayed contrast-enhanced inversion recovery sequence on short axis view and an hyper-enhanced areas at anterior and inferior walls as results of previous ischemic and necrotic events. Not enhancement of pericardial layers or right ventricular myocardium was observed and a moderate non corpuscular pericardial effusion, with a heterogeneous content, was due to the presence of fibrin in the fluid (Figure 2). No pericardial tamponade, pericardial inflammation or right ventricular (RV) delayed-enhancement areas were visualized (Figure 3). At the perforation site the pericardium appeared thinner with a heterogeneous signal intensity, cine sequences demonstrated adhesions between right ventricular myocardium and pericardial layers.

Discussion

Lead perforation is a complication of pacemaker or cardioverter-defribrillator implantations[1].

The characteristics of the leads have markedly improved: less stiff, more flexible and thinner leads are now available. It is supposed that current thinner leads carry an increased perforation risk, because they would present a higher force per unit area at the tip compared with prior larger-calibre ones. As Singhals et al[2] reported, in fact, the lead tip is subjected to complex forces that change during the cardiac cycle and over time, in relation to the interplay with resistance

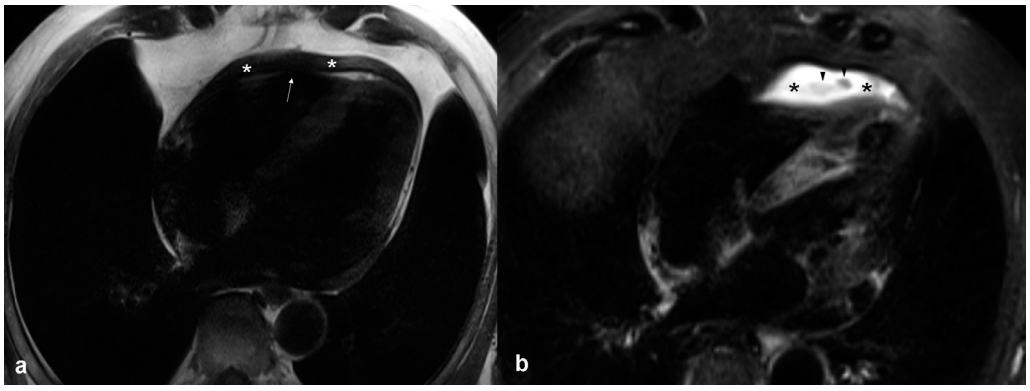

Figure 2. Four-chamber view SE T1w sequence ***(A)*** and STIR T2w sequence ***(B)*** show a diffuse pericardial effusion *(asterisks)* with increased-signal-intensity fibrin us exudates within the pericardial space (*arrowheads*) and a disomogeneous signal intensity of the pericardial visceral layer on the right ventricle free wall near the apex (*arrow*).

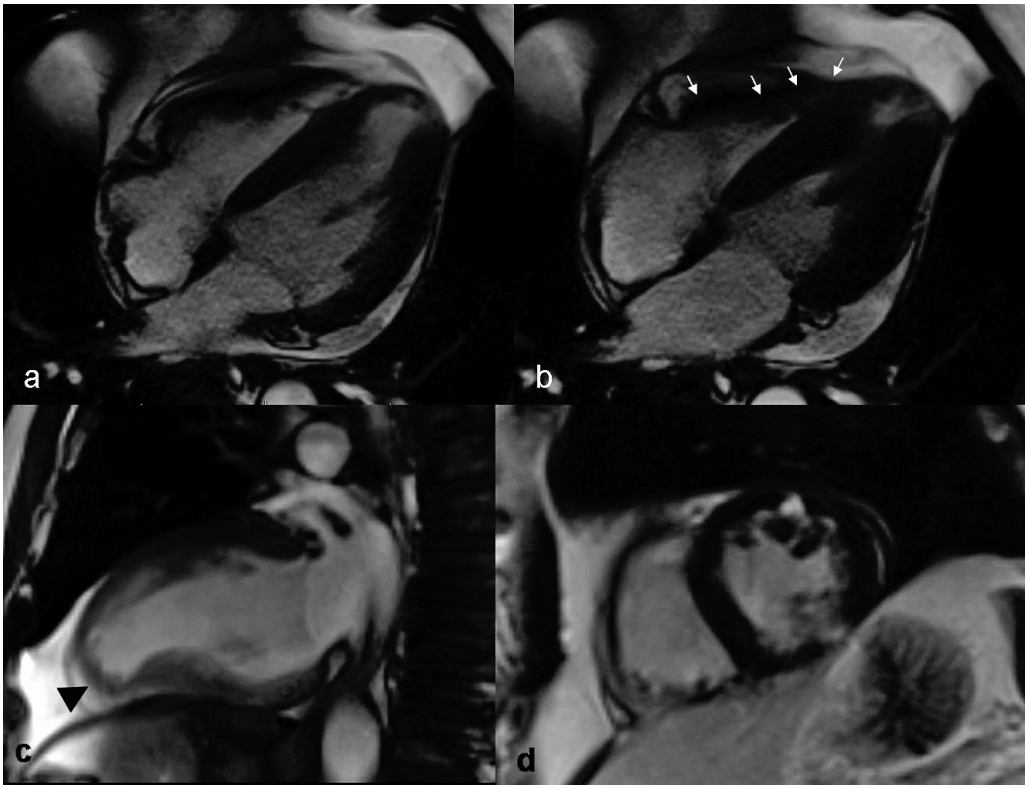

Figure 3. Balanced steady-state free precession cine sequence on four-chamber view ***(A-B)*** shows thinning of the right ventricular free wall with a corresponding discynetic systolic movement (*arrows*). A vertical long axis view ***(C)*** confirms the presence of an apical left ventricular aneurysm (*arrowhead*). Delayed contrast-enhanced inversion recovery sequence on short axis view ***(D)*** shows hyper enhanced areas at anterior and inferior walls as results of previous ischemic/necrotic events. Not enhancement of pericardial layers or right ventricular myocardium was observed.

of the local myocardium. The lead positioning plays a role, with placement on the RV apex or free wall presenting a higher perforation risk than in the septum. The effects can result in lead stability or migration. In this case, for more, the inferior wall infarct was a further risk factor.

We emphasize that the lead ventricular perforation occurred without tamponade and relevant symptoms, despite the high bleeding risk both in the acute phase (anticoagulant and triple platelet antiaggregant therapy) and after the percoutaneous extraction lead without suspending double platelet antiaggregant therapy.

We underline the safety of conservative management used in this case report as Lopes et al.[3] and Wilkoff et al[4] have referred. Lead extraction guided by transesophageal echocardiography (TEE) is safety and advisable. According to us, for more, double platelet antiaggregant therapy is not a contraindication to lead extraction.

The absence of complications could be connected with the spatial organization of the myocardial fibres during heart contraction, which has long been considered a kind of ”Gordian knot” of anatomy.

Torrent-Guasp et al.[5], recently, showed that the ventricular myocardium consists of a muscular band that extends from the pulmonary artery to the aorta, curling in a helical manner. The architectural organization consists in two spirals: a basal and an apical loop, in which fibres cross each other at 90° angle around the ventricular apex (“vortex cordis”). We believe that the overlapping of muscular layers with different orientation could provide a contractile mechanism explaining the orifice’s closure.

Conclusion

The echocardiography evaluation, at the beginning, and the CMR, in the following days, have showed the myocardial damage and allow us to choose the best treatment.

In particularly, the pericardial effusion at the echocardiography suggested and drove us the trans-femoral venous extraction. The CMR completed the tissue evaluation before installing a definitive dual-chamber pacemaker with passive fixation leads, in an extremely narrow times.

So, the integration of these diagnostic techniques will implement, in the future, the ability of clinical management of all patients hospitalized in a Coronary Care Unit.

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