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CASE REPORT

Post-cardiac injury syndrome following transvenous pacing: Case report

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KEYWORDS
Post-cardiac injury syndrome;
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Abstract Post-cardiac injury syndrome (PCIS) is an inflammatory process involving the pericardium secondary to cardiac injury. It can develop after cardiac trauma, cardiac surgery, myocardial infarction, and, rarely, after certain intravascular procedures. We report a rare case of an iatrogenic cardiac rupture followed by PCIS with delayed inflammatory pericardial effusion after pacemaker implantation. A comprehensive literature review on this topic is provided.

PALAVRAS-CHAVE
Síndrome pós-lesão cardíaca;
Rotura cardíaca;
Implantação de *pacemaker*

Síndrome pós lesão cardíaca após *pacing* transvenoso – a propósito de um caso clínico

Resumo A síndrome pós-lesão cardíaca (SPLC) corresponde a um processo inflamatório envolvendo o pericárdio, secundário à lesão cardíaca. Pode desenvolver-se após traumatismo cardíaco, cirurgia cardíaca, enfarte agudo do miocárdio, e, raramente, após alguns procedimentos intravasculares. Os autores apresentam o caso invulgar de uma rotura cardíaca iatrogénica após implantação de um *pacemaker*, seguida pelo desenvolvimento de um derrame pericárdico retardado, inflamatório, correspondendo a SPLC. A propósito do referido caso clínico, é efetuada uma revisão compreensiva da literatura acerca desta entidade.

Introduction

Pacemaker implantation is a classic technique in cardiology. Materials have changed considerably in recent years, making the procedure safer and the indications broader. However,

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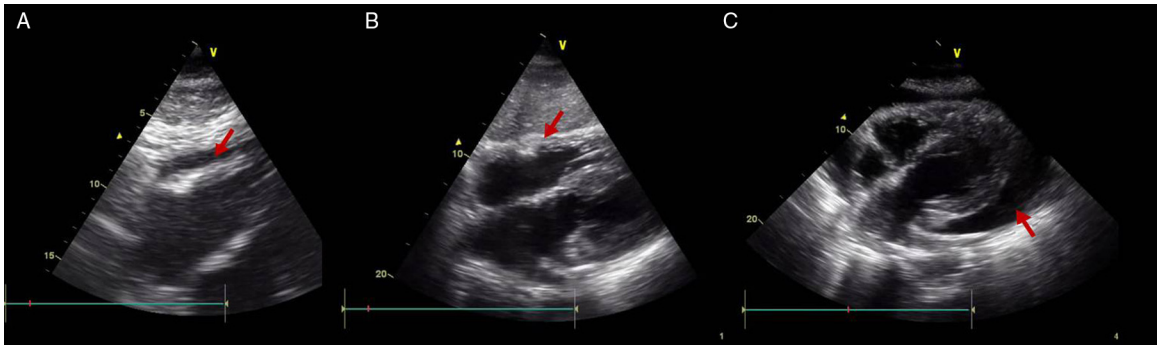

Figure 1 Echocardiographic subcostal views: (A) after permanent pacemaker implantation revealing a new moderate pericardial effusion (arrow); (B) at hospital discharge, after a few days of clinical surveillance with no evidence of pericardial effusion (arrow); and (C) at readmission, with a large pericardial effusion and 'swinging heart' (arrow).

as in all invasive procedures, there is the risk of immediate and delayed intra and postoperative complications, such as system infection, lead displacement and post-cardiac injury syndrome (PCIS).

We report a rare case of an iatrogenic cardiac rupture followed by PCIS with delayed pericardial effusion after a pacemaker implantation. A comprehensive literature review on this topic is provided.

Case report

An 89-year-old woman was admitted for syncope. She had a history of hypertension and dyslipidemia.

At admission, she reported a brief episode of loss of consciousness, without prodromes or head trauma and with spontaneous recovery. Her physical examination revealed bradycardia but no other relevant findings. The electrocardiogram showed advanced heart block with mean heart rate of 30 bpm. She was not under any negative chronotropic medication and laboratory tests showed no relevant electrolyte disturbances. Summary echocardiography revealed mild systolic dysfunction without pericardial effusion.

A temporary pacemaker was inserted via the right femoral vein, without immediate complications, followed by implantation of a dual chamber permanent pacemaker the next day. During this procedure, the patient presented a brief period of hypotension and pleuritic chest pain. The echocardiogram showed moderate systolic dysfunction and new moderate pericardial effusion (16 mm), with no signs of hemodynamic compromise (Figure 1). A diagnosis of iatrogenic right ventricle rupture was made and the patient was kept under clinical, electrical and echocardiographic monitoring. She presented progressive reduction of the pericardial effusion and was discharged by the 5th day, asymptomatic and without pericardial effusion (Figure 1).

Four weeks later, she was readmitted for pleuritic chest pain and asthenia. Physical examination revealed reduced heart sounds with no other significant alterations, including in the pacemaker scar, which presented no inflammatory signs. The electrocardiogram showed sinus rhythm with P-wave synchronous ventricular pacing. Blood tests showed leukocytosis (17×10^9/l) and elevated C-reactive protein (CRP) (110 mg/dl), with no evidence of systemic infection or fever. Blood cultures were negative and the chest X-ray showed cardiomegaly without pleural effusion. The echocardiogram revealed a large pericardial effusion (25 mm), with 'swinging heart' and signs of hemodynamic compromise (inferior vena cava dilatation, mitral and tricuspid flow variation >50%, abnormal septal motion, mild diastolic compression of right heart chambers), suggestive of incipient tamponade physiology (Figure 1).

Pericardiocentesis was performed, with drainage of 350 ml of light yellow fluid, which was found to be a sterile exudate (Table 1). Post-procedural echocardiography still showed moderate pericardial effusion (18 mm), but with no signs of hemodynamic compromise. Autoimmunity study was negative. There was no sign of pacemaker dysfunction, sensing and pacing thresholds being optimal.

The patient was diagnosed with PCIS and medicated with aspirin (500 mg four times a day) and colchicine (1 mg twice a day). As there was no favorable clinical evolution or remission of the pericardial effusion, prednisolone was added on the seventh day (1 mg/kg/day), with eventual resolution of symptoms, laboratory parameters and pericardial effusion. She was discharged on the 15th day and remained asymptomatic at follow-up.

Discussion

PCIS is an inflammatory process involving the pleura (pleural effusion) and/or pericardium (pericarditis, pericardial effusion) secondary to cardiac injury.[1] It can develop after cardiac trauma, cardiac surgery, myocardial infarction and certain intravascular procedures, including transvenous

Table 1 Analysis of pericardial fluid.

Appearance	Light yellow
Nucleated cell count, 10^9/l	0.3
Neutrophils, %	66
Lymphocytes, %	26
Total protein, g/dl	49.20
LDH, U/l	781
ADA, U/l	28.5
pH	8
Microbiology	Sterile

ADA: adenosine deaminase; LDH: lactate dehydrogenase.