Post-Cardiac Injury Syndrome after Permanent Pacemaker Implantation

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The mechanism of post-cardiac injury syndrome (PCIS) was thought to be an autoimmune-mediated process and can involve the pleura and pericardium. PCIS may result in pleural effusion, pericardial effusion or even acute pericarditis leading to cardiac tamponade following chest trauma, or other invasive procedures such as coronary angioplasty or transvenous pacemaker implantation, especially in dual chamber mode with active atrial fixation leads. Typically, the outcomes of PCIS were good, and most cases were treated conservatively without surgical intervention, with no mortality case reported. Here, we report on a patient with PCIS after permanent pacemaker implantation, who was successfully treated with non-steroid anti-inflammatory drugs (NSAIDs) and colchicine.

Key Words: Pacemaker • Pericardial effusion • Pericarditis • Pleural effusion

CASE REPORT

A 78-year-old female had suffered from frequent dizziness and transient loss of consciousness for three weeks before admittance. Sick sinus syndrome was confirmed via 24 hours of Holter EKG monitoring, and the patient was treated with permanent pacemaker implantation (Medtronic Adapta, DDDR mode, active fixation leads, SN PJN2068017, Minneapolis, MN, USA) with the right ventricular lead screwed towards the septum region, and the right atrial lead screwed over the anterior wall of the right atrium. No chest pain was noted after the procedure, however paroxysmal atrial fibrillation (PAF) developed on the following day and was terminated after intravenous amiodarone infusion. Afterward, PAF recurred again on the 2nd, 3rd and 4th day.

Unfortunately, the patient developed shortness of breath with a low grade fever (37.5 °C) on the 5th day, but her physical examination revealed no pericardial friction rub; a follow-up chest x-ray showed increased cardiac silhouette without pacemaker leads protruding into the pericardium. Leukocytosis with neutrophil predominance and an elevated level of C-reactive protein (CRP = 7.07 mg/dL) were also noted. The subsequent computed tomography (CT) scan of chest (Figure 1) and echocardiography disclosed a moderate amount of pericardial effusion without diastolic collapse, and bilateral pleural effusion with mild partial passive atelectasis in both lungs. Without invasive therapeutic pericardiocentesis, her symptoms improved after conservative treatment with non-steroid anti-inflammatory drugs (NSAIDs), naproxen 750 mg once a day, and colchicine 0.5 mg twice a day. The subsequent chest x-ray and echocardiography showed a reduced heart size (Figure 2, left part) and a decreased amount of pericardial fluid. The patient was discharged after her symptoms were relieved, and there was no clinical or echocardiographic sign of recurrent pericardial effusion after a follow-up one month later (Figure 2, right part).
Post cardiac injury syndrome (PCIS) refers to several terms, including post myocardial infarction syndrome (PMIS), post comissurotomy syndrome (PCS) and post pericardiotomy syndrome (PPS). It is a common complication after cardiac surgery and the reported incidence varies from 10% to 50%.\(^1\) In 1959, William Dressler\(^2\) first described the pericarditis-like presentation which developed days to weeks after myocardial infarction, and the autoantigens-related hypersensitivity reaction was considered to be the mechanism. Meanwhile, Janton et al.\(^3\) observed PCS in patients who underwent surgical intervention for rheumatic mitral stenosis. Again, Ito et al.\(^4\) mentioned PPS occurred in patients who had received surgical correction of congenital heart disease. In the years that have followed, several authors reported that PCIS could be observed after percutaneous coronary angioplasty,\(^5\) radiofrequency catheter ablation\(^6\) and pacemaker implantation.\(^7\)

The clinical manifestations of PCIS are nonspecific, with the most common symptoms being pleuritic chest pain (91%) and dyspnea (57%). Additional signs of PCIS are fever (66%), and pericardial friction rub (63%), with the most common laboratory findings being an elevated erythrocyte sedimentation rate (96%) and leukocytosis (49%).\(^8,9\)

Greene et al. reported 123 consecutive patients who received dual-chamber pacemaker insertion with active atrial fixation biopolar leads, and six patients (4.9%) developed acute symptomatic pericarditis.\(^10\) In these six patients, the atrial leads were screwed into the lateral or anterior lateral wall of the right atrium, a location which is similar to that used in our case. Fortunately, all patients were successfully treated with NSAIDs and had resolution of symptoms within two weeks. In 2004, Zeltser et al. conducted a retrospective study in children and young adults regarding PCIS after permanent pacemaker implantation. In that study, 443 pacemakers (237 epicardial and 206 transvenous) were implanted in 370 patients, and eight (2%) episodes of PCIS (6 epicardial and 2 transvenous) occurred in seven patients.\(^11\) They were managed successfully with medical therapy including a NSAID and corticosteroids. The patient’s refrac-
tory to medical therapy were successfully treated with pericardiocentesis or surgical creation of a pericardial window.

Spindler et al.\textsuperscript{12} reported 10 similar cases in the literature and subsequently added this case into analysis (n = 11, 8 women and 3 men). Most of his subjects (n = 9, 81.8\%) received dual-chamber pacemaker implantation, and active atrial fixation leads were inserted into five patients (45.4\%). PCIS developed between five and 56 days after pacemaker implantation (mean 21.5 days), and all patients had pericardial effusion identified via echocardiography and CT scan of the chest. The outcomes of these cases were good and most of them (n = 7, 63.6\%) could be treated conservatively without surgical intervention. Recently, Hsu et al. reported Takotsubo cardiomyopathy developed after permanent pacemaker implantation, and could be a potential complication after pacemaker implantation.\textsuperscript{13}

In conclusion, although the pacemaker implantation procedure is safe and feasible, some clinical presentations should remind us to be suspicious of PCIS and its symptoms, especially during dual-chamber pacemaker insertion with active atrial fixation leads, or when a history of temporary transvenous pacemaker use before the permanent pacemaker implantation is involved. These symptoms included shortness of breath, fever, elevated sedimentation rate, and even an unexplained drop in hemoglobin in the absence of an obvious inflammatory process. If identified early, most patients can be successfully treated with NSAIDs, colchicine and/or prednisone, and further surgical intervention may be avoided.

REFERENCES