Pacemaker-Associated Post-cardiac Injury Syndrome Presenting with Tamponade and Recurrent Pleural Effusion

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Young Ju Lee[1], Mahmood Mubasher[1], Abir Zainal[1], Tausif Syed[1], Mouhand F.H. Mohamed[2], Matthew Ferrantino[1] and Ryan Hoefen[1]

[1]Unity Hospital, Rochester Regional Health, Rochester, New York, USA. [2]Internal Medicine Department, Hamad Medical Corporation, Doha, Qatar.

ABSTRACT: Post-cardiac injury syndrome (PCIS) is presumed to be an immune-mediated process. It affects the pericardium and, to a lesser extent, the epicardium, myocardium, and pleura. It has been rarely reported following pacemaker insertion with an estimated incidence of 1% to 2%. We present the case of a 62-year-old female who developed PCIS 8 weeks following pacemaker insertion. She presented with impending cardiac tamponade requiring pericardiocentesis; recurrent pleural effusions subsequently complicated her condition. The pleural effusion recurred despite trials of steroids, eventually requiring talc pleurodesis. This case highlights the need to consider PCIS as a possible etiology of recurrent pleural effusion following pacemaker insertion.

KEYWORDS: Pleurodesis, postpericardiotomy syndrome, PPS, post-cardiac injury syndromes, PCIS, pericardial effusion, pleural effusion

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CORRESPONDING AUTHOR: Mouhand FH Mohamed, Internal Medicine Department, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar. Email: dr.m.oraiby@hotmail.com

Background

Post-cardiac injury syndrome (PCIS) is presumed to be an immune-mediated process with unclear etiology that may be driven by cardiac tissue injury. It includes three significant entities: (1) post-myocardial infarction syndrome, (2) postpericardiotomy syndrome (PPS), and (3) post-traumatic pericarditis. PCIS is estimated to complicate 15% to 30% of cardiac surgeries and 1% to 2% of cardiac pacemaker insertions.[1,2] The clinical diagnosis requires a history of cardiac injury, additionally, the presence of at least two of the following: fever without alternative cause, pleuritic or pericardial chest pain, pericardial or pleural rub, pericardial effusion, and/or pleural effusion with elevated C-reactive protein (CRP).[3] Transthoracic echocardiography (TTE) and chest radiography (CXR) can support the diagnosis. Delay in treatment resulting from a failure to identify and diagnose this condition promptly will increase its morbidity. Thus, we here present an unusual case of PCIS complicated by cardiac tamponade and recurrent pleural effusions. Additionally, we highlight the management of this condition through a brief literature review.

Case Presentation

We present the case of a 62-year-old woman known to have hypothyroidism and osteopenia. She developed dyspnea and fatigue during a cruise in Central America. She was seen at the ship's infirmary and found to have a complete heart block. Upon disembarking, she was taken to a nearby hospital, where she underwent permanent pacemaker (PPM) placement. The evaluation of her conduction system disease's underlying causes included basic laboratories and a Lyme titer, which were normal.

Upon returning home, she initially felt better. However, she saw her primary care physician several days later with dyspnea symptoms on exertion and intermittent palpitations. Initial blood work revealed no normal complete blood count and basic metabolic panel. There was a mild rise in the inflammatory markers (erythrocyte sedimentation rate [ESR] and C-reactive protein [CRP]). She was referred to a cardiologist for further management.

Her pacemaker was interrogated and found to be functioning normally with no evidence of arrhythmias. A transthoracic echocardiogram (TTE) showed normal left ventricular function (ejection fraction 55%), normal valves, and a moderate circumferential pericardial effusion without evidence of tamponade physiology with the pacemaker leads well placed at the anterolateral wall of the right ventricle. Five days later, she presented to the emergency department (ED) with complaints of low-grade fevers, fatigue, dry cough, and orthopnea and increasing dyspnea. She denied paroxysmal nocturnal dyspnea. On physical exam, she was afebrile, with a heart rate of 120 breaths per minute, blood pressure 145/112 mmHg, respiratory rate 20 breaths per minute, and hypoxemic with an O_2 saturation of 89% on room air. Pertinent examination findings revealed jugular vein distention, fine bibasilar crackles, distant heart sounds, and absence of lower extremity edema. CXR revealed a small left pleural effusion with left lower lobe consolidation. TTE, this time, revealed a moderate to large pericardial effusion with impending tamponade physiology (Figure 1). Antibiotics were started for possible pneumonia. An ECG showed an atrial-sensed, ventricular-paced rhythm with a rate in the 110 to 120 seconds (Figure 2). Pericardiocentesis was performed with immediate removal of 500 ml of bloody fluid and an additional

210 ml of drainage over the next 24 hours. The fluid analysis revealed a sterile, exudative fluid. Cytology was negative for malignancy. She was discharged on colchicine.

She returned to the ED a week later with progressive dyspnea on minimal exertion. Computed tomography (CT) angiography was performed to rule out a pulmonary embolism, which showed a small to moderate pericardial effusion and moderate left pleural effusion. Thoracentesis removed 11 and again revealed sterile, exudative fluid (Figure 3). She was discharged with a close follow-up with cardiology and advised to continue colchicine.

Four days later, the patient again returned to the ED with dyspnea. CXR demonstrated the re-accumulation of the left pleural effusion. Repeat thoracentesis was attempted demonstrating sterile, exudative fluid. This time, TTE ruled out the presence of pericardial effusion. Device interrogation showed normal pacemaker function, and imaging confirmed normal lead placement. Due to her travel history, she was evaluated for infectious causes, including a Lyme titer, *Trypanosoma* sp., *Babesia* sp., *Leishmania* sp., and *Plasmodium* sp. Anti-Ro/SSA and La/SSB antibodies, antinuclear antibody, rheumatoid factor, and acid-fast stains were also negative. Her symptoms improved with steroids. PCIS was deemed the likely diagnosis after excluding common causes of her combined pericardial and pleural effusion. Colchicine and steroids were continued in an attempt to mitigate the inflammatory response.

An outpatient CXR 6 weeks later revealed almost complete resolution of the left-sided pleural effusion. Thus, steroids were tapered over the course of several weeks, and she remained on colchicine. Following the steroid taper, she developed a recurrence of pleural effusion. She was eventually referred to thoracic surgery and underwent video-assisted thoracic surgery (VATS) with talc pleurodesis. The patient remained without evidence of recurrent pleural effusion on surveillance imaging.

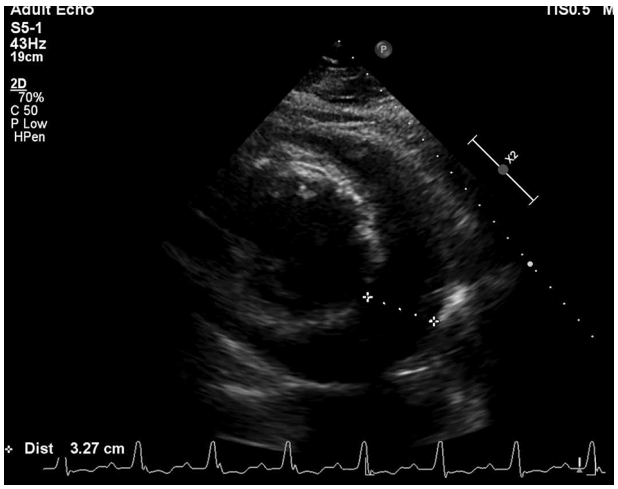

Figure 1. TTE showing a large pericardial effusion.

Discussion

Pericarditis is the most common disease of the pericardium.[3] The most common causes include viruses such as enteroviruses and adenoviruses and tuberculosis in endemic countries. Noninfectious causes include autoimmune, neoplastic, and rarely traumatic/iatrogenic causes. PCIS can be triggered by any insult to the pericardial or pleural lining. A small prospective study in children demonstrated an association between antimyosin antibodies and PCIS; titer levels correlated with the disease severity.[4] The latency period between mesothelial injury and onset of PCIS, as well as the positive effect of immune-modulating agents, support the hypothesis of autoimmune-mediated pathogenesis.[5-7] PCIS includes (1) post-myocardial infarction (MI) pericarditis, which includes early post-MI pericarditis occurring in the first 5 days, and late post-MI pericarditis that occurs 2 to 8 weeks post-MI, also known as

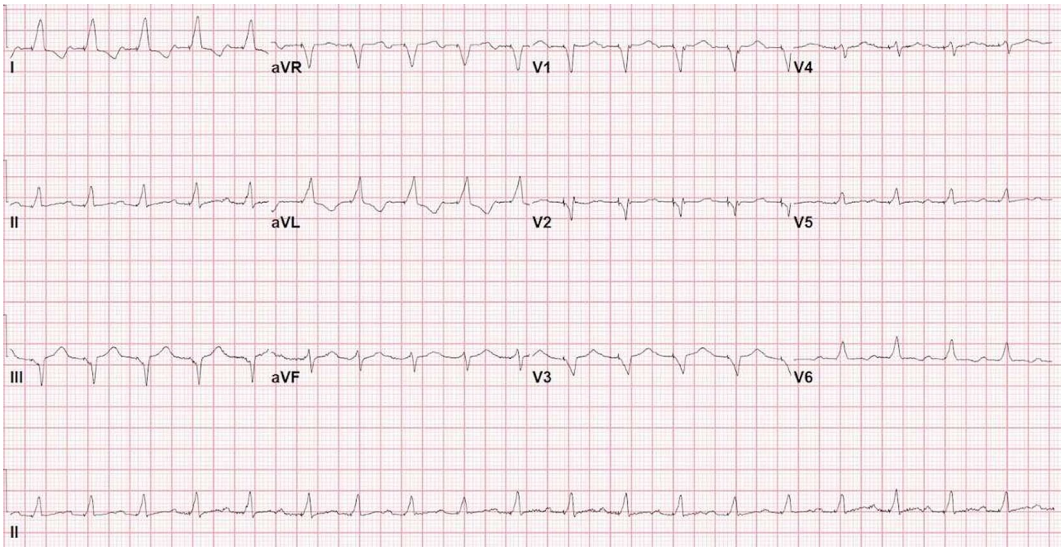

Figure 2. ECG taken on admission showing a ventricular paced rhythm.

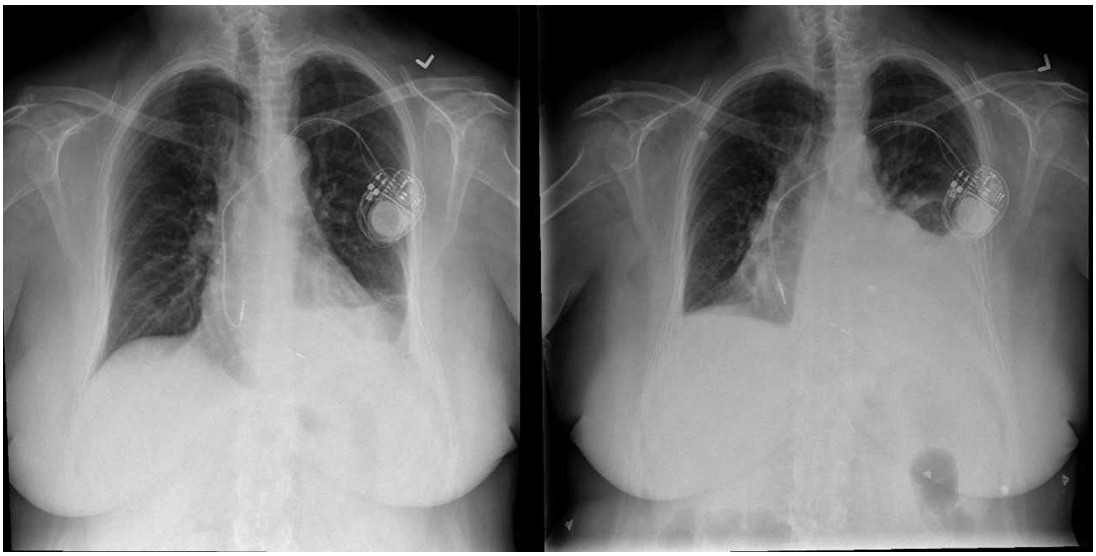

Figure 3. CXR showing a large left pleural effusion, before (left) and after (right) thoracentesis confirmed sterile exudative fluid.

Dressler's syndrome; (2) postpericardiotomy syndrome (PPS) after any open-heart surgery, such as coronary artery bypass surgery or valve surgery; and (3) post-traumatic pericarditis due to accidental or iatrogenic injury such as pacemaker insertion.[8]

While PCIS is a relatively common complication of cardiac surgery that classically occurs days to several weeks after cardiac surgery with an incidence of 10% to 40%,[9-11] its incidence following MI is declining due to early reperfusion strategies and the immunomodulatory effects of standard medications, including ACE inhibitors, some beta-blockers, statins, and aspirin. Cardiac tamponade and constrictive pericarditis were rarely reported in post-MI PCIS.[12-16] PCIS triggered by percutaneous coronary interventions, pacemaker lead insertion, radiofrequency ablation, and even post Swan-Ganz catheterization has a low incidence of 0.5% to 5%.[6,7,17] The estimated risk is 1% to 2% post pacemaker implantation.[18,19] Iatrogenic trauma with or without bleeding due to wall perforation has been proposed as a possible mechanism of PCIS after pacemaker implantation.[8] According to Ohlow et al,[18] female sex, advanced age, and use of active fixation leads are independent risk factors for the development of post pacemaker PCIS. Our patient was a 62-year-old female, which may have put her at relatively high risk for post pacemaker insertion PCIS.

The proposed diagnostic criteria from the 2015 ESCG guidelines require a history of cardiac injury in addition to two of the following: "(1) fever without alternative causes, (2) pericarditic or pleuritic chest pain, (3) pericardial or pleural rubs, (4) evidence of pericardial effusion and/or (5) pleural effusion with elevated CRP."[3] The guidance stresses that the demonstration of an inflammatory response or activity is necessary to rule out mechanical causes and establish the diagnosis. Supplementary findings, according to the underlying disease process, may be present, such as fever and non-specific evidence of inflammation (ie, CRP, ESR, leukocytosis). It is essential to rule out overt perforation and micro-perforation before diagnosing post-pacemaker PCIS.[18,19]

TTE and CXR are essential in evaluating the lead position. Our patient's interrogation revealed normal capture threshold and impedance, making perforation less likely. However, we acknowledge that a normal pacemaker interrogation does not rule out lead perforation.

Given the low incidence of PCIS, there is no current indication for prophylaxis. However, the postpericardiotomy syndrome may contribute to morbidity after cardiac surgery. Meta-analysis reveals that colchicine's use reduced the rate of PCIS in that cohort of patients.[6] Simultaneously, non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids were not beneficial for that purpose.[7]

Treatment of post pacemaker PCIS is not different from other forms of PCIS. NSAIDs and colchicine are the mainstays of therapy with steroids reserved for refractory cases or when contraindications to therapy exist (colchicine dose is 0.5 mg if the patient's weight >70 kg and once daily if <70 kg).[2,3] The choice of treatment depends on potential side effects and the severity of symptoms.[2] Treatment with NSAIDs should be continued until symptoms abate, and CRP normalizes, at which point the dose can be tapered. Aspirin can be used instead in patients with coronary artery disease who are already on aspirin, as NSAIDs interfere with aspirin's antiplatelet effect, which can increase the risk of myocardial rupture post-myocardial infarction. If glucocorticoids are initiated, doses should be tapered slowly to prevent recurrence and before colchicine is discontinued.

In rare instances, the patient may become steroid-dependent or resistant, further intervention such as pleurodesis may prove efficacious in such instances as highlighted by our case. The recurrence rate of this entity is less than that of idiopathic

pericarditis; however, it is associated with a higher risk of constrictive pericarditis.[20]

Conclusion

PCIS can rarely complicate cardiac pacemaker insertion. Our case highlights that it may lead to recurrent pleural effusion resistant to anti-inflammatory agents, necessitating surgical interventions.

Authors Note

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Author Contributions

YJL wrote the initial draft of this paper. This was critically reviewed and edited by all authors. All the authors approved the final version for publication considerations.

Patient Consent

The patient has consented the publication of this case.

ORCID iDs

Mahmood Mubasher https://orcid.org/0000-0002-1146-2732
Tausif Syed https://orcid.org/0000-0002-5872-1711
Mouhand F.H. Mohamed https://orcid.org/0000-0002-4761-8014

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