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Case Report
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Recurrent Pericardial Effusions Following a Pacemaker Implant: A Case Report

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Abstract

Background: Post-cardiac injury syndrome (PCIS) is described as the onset of pericarditis with or without pericardial effusion after recent cardiac injury. Pacemaker placements can cause PCIS, but its incidence is often overlooked. This case report describes a patient who experienced recurrent pericardial effusion after pacemaker implant due to PCIS. While cases of PCIS after pacemaker implantation have been reported, this case is unique in its kind due to the presentation of recurrent pericardial effusions.

Case Description: A 91-year-old male, with a history of bradycardia that was recently treated with a pacemaker, presented to the emergency department in September 2023 with a chief complaint of dyspnea. His medical history was significant for type 2 diabetes mellitus, chronic obstructive pulmonary disease, and chronic kidney disease. Two months prior, the patient received a dual-chambered Biotronik pectoral pacemaker. One month prior, the patient suffered a pericardial effusion with pericarditis and pericardial tamponade. Vitals demonstrated tachypnea and hypertension, while CBC and CMP reveal leukocytosis and elevated creatinine and D-dimer levels. Chest CT results indicated a pericardial effusion along the apex of the heart. After admission, the patient's pacemaker leads were repositioned in the catheter lab, and he was prescribed colchicine prior to discharge. Based on the patient's clinical presentation and medical history, the patient's primary differential diagnosis is pericardial effusion secondary to PCIS.

Discussion: This case shows that PCIS can present as recurrent pericardial effusions after a history of pacemaker implantation, a cardiac insult. Comorbidities including chronic kidney disease, diabetes mellitus, and anticoagulation therapy may increase the risk of cardiac injury.

Keywords: post cardiac injury syndrome, PCIS, pacemaker, pericardial effusion

Introduction

Pericardial effusion is the accumulation of fluid in the sac surrounding the heart. The pericardial sac is bound by the visceral pericardium attached to the heart and the parietal pericardium attached to the thicker fibrous pericardium. Normally, there is between 15 and 50 milliliters (mL) of serous fluid in between the serous pericardium to lubricate the heart during contraction and relaxation (Willner et al., 2017). Symptoms of pericardial effusion include shortness of breath and chest pain. Cardiac tamponade can occur when the pericardial sac accumulates excessive fluid that leads to increased pressure on the heart, impairing ventricular filling and resulting in decreased cardiac output. This case describes a post-cardiac injury syndrome of recurrent pericardial effusion resulting from implantation of a pacemaker.

Pericardial effusion may accompany pericarditis, a condition defined as the inflammation of the pericardial sac. Pericarditis can be further divided into four diagnoses based on timing of symptoms: acute pericarditis lasts approximately one month, recurrent incessant pericarditis lasts between 4-6 weeks and 3 months, chronic pericarditis lasts longer than 3 months, and recurrent pericarditis contains a 4-6 week symptom-free interval between episodes (Dababneh & Siddique, 2023). Diagnosis of pericarditis requires two of any of the following signs or symptoms: chest pain, pericardial rub, saddle-shaped ST-elevation and/or PR-depression, and new or worsening pericardial effusion (Ismail, 2020). Since the visceral pericardium is innervated by afferent sympathetic pain fibers in a cardiac distribution, chest pain is the most common complaint among pericarditis patients. Chest pain is often worsened on inspiration and improved by leaning forward. Because the parietal and fibrous pericardium are innervated by the phrenic nerve, it may also result in referred shoulder pain (Ismail, 2020).

Permanent pacemakers consist of a battery placed within the upper chest, which is then attached to a lead within the myocardial tissue. Impulses from the battery are conducted to the lead in order to induce depolarization within the myocardium and facilitate coordinated heart contraction (Mulpuru et al., 2017). Therefore, any dysfunction of the sinus node, where the propagation of depolarized impulses for heart contraction is located, is an indication for permanent pacemaker placement. Currently, atrioventricular block and bradycardia secondary to sinus node dysfunction are the leading indications for permanent pacemaker implantation worldwide (Markos et al., 2024). Other indications also include congestive heart failure in patients with a lowered ventricular ejection fraction (Madhavan et al., 2017).

Pacemakers are indicated for individuals experiencing extreme ranges of bradycardia or tachycardia experiencing symptoms. Kosztin et al. (2018) find that cardiac resynchronization therapy (CRT), a type of pacemaker therapy, is strongly indicated in heart failure patients with left ventricular ejection fractions (LVEF) less than 35%. Patients with left bundle branch block (LBBB) and a QRS width greater than 130 ms can also benefit from (CRT) (Kosztin et al., 2018). Additionally, pacemakers are indicated for sinus node dysfunction, atrioventricular block, neurocardiogenic syndromes, and uncontrollable cases of atrial fibrillation with rapid ventricular response (Samii, 2015).

A portion of patients will experience side effects due to permanent pacemaker placement including a pneumothorax, left–sided or right–sided lead complication, or pocket hematomas (van Rees et al., 2011). Data from Mulpuru et al. (2017) support these findings, indicating 5.7% of patients experience left ventricular lead complications, 3.5% experience pocket hematomas, and 0.9–1.2% experience pneumothorax.

The goal of this paper is to understand the patient's clinical presentation of dyspnea and relate the findings to his pacemaker implantation. First, the patient's signs, symptoms, and pertinent history will be described and analyzed. Then, a primary differential diagnosis will be established, along with pertinent positives and negatives supporting the diagnosis. Finally, the possible risk factors leading to the patient's diagnosis will be investigated.

Case Report

September 5th, 2023 - Emergency Department

On September 5th, a 91-year-old male presented with dyspnea to the ED. Patient reported that the shortness of breath worsened upon exertion, and he also experienced chest pain. He has a significant medical history of bradycardia, GERD, pulmonary embolism (PE) with deep vein thrombosis (DVT), COPD, erectile dysfunction, Type 2 diabetes mellitus (T2DM), chronic kidney disease, obstructive sleep apnea, and benign prostatic hyperplasia. Treatment for his deep vein thrombosis initially included apixaban, after which he temporarily switched to warfarin and then continued with apixaban. In July 2023, the patient had a pacemaker placement, specifically a dual-chambered Biotronik pectoral pacemaker. In August 2023, the patient had difficulty breathing with underlying pericarditis and pericardial tamponade, for which he received two pericardiocentesis procedures. Upon questioning, the patient indicates that he has had no experience with nausea, vomiting, headache, dizziness, abdomen pain, numbness or tingling, or loss of bowel and bladder function. Patient was admitted on September 6th, 2023.

During his visit to the ED, the patient had a temperature of 98.3°F, a heart rate of 73 beats per minute, a respiration of 22 respirations per minute, and oxygen saturation of 96%. His systolic blood pressure was elevated, as his blood pressure was 131/66 mmHg, for which he was requested to follow-up in the clinic a few days later. Due to the patient's extensive medical history and episode of pericarditis with pericardial effusion in August of 2023, an ECG and chest x-ray were ordered. A complete blood count (CBC) with differential was ordered. The patient's lipase, D-dimer, brain natriuretic peptide (BNP), and complete metabolic panel (CMP) values were also assessed.

Table 1: CBC with Differential Patient Results.

CBC Test	Patient's Values	Reference Values
RBC	4.90 x10^6/uL	4.3-5.9 x10^6/uL
Hemoglobin	14.4 g/dL	13.5-17.5 g/dL
Platelet	174 x10^3/uL	150-400 x 10^3/uL
WBC	14.14 x10^3/uL	4.5-11.0 x10^3/uL
Neutrophils	82.3%	54-62%
Lymphocytes	11.2%	25-33%
Monocytes	5.1%	3-7%
Eosinophils	0.3%	1-3%
Basophils	0.2%	0-0.75%

Table 1. Test results for the complete blood count (CBC) with red blood cell (RBC), hemoglobin, platelet, and white blood cell (WBC) differential values, along with the normal ranges.

Table 2. CMP Patient Results.

CMP Test	Patient's Values	Reference Values
Glucose	245 mg/dL	65-99 mg/dL
Urea Nitrogen	48 mg/dL	7-25 mg/dL
Creatinine	1.9 mg/dL	0.7-1.3 mg/dL
Calcium	8.5 mg/dL	8.6-10.3 mg/dL
Lipase	39 U/L	11-82 U/L
Estimated Glomerular Filtration Rate (eGFR)	33 mL/min/1.73m²	Greater than or equal to 90 mL/min/1.73m²

Table 2. Test results for the complete metabolic panel (CMP), along with the normal ranges. Only abnormal values and lipase are listed.

Table 3. D-Dimer and BNP Tests

Lab Test	Patient's Values	Reference Values
D-dimer	1287 ng/mL	Less than 500 ng/mL
Brain Natriuretic Peptide	70 pg/mL	0-100 pg/mL

Table 3. Test results for the D-dimer and BNP tests, along with the normal ranges.

Based on the CBC in Table 1, the patient's WBC and neutrophil counts are high, while the lymphocyte, monocyte, and eosinophil counts are low. The CMP in Table 2 shows that the patient's serum glucose, urea nitrogen, and creatinine measurements are high. The calcium levels are slightly decreased, while the lipase levels are within normal ranges. The estimated glomerular filtration rate is lower than normal. In Table 3, the D-dimer measurement is increased compared to reference values, and the patient's BNP is within normal limits.

Chest CT results indicate a pericardial effusion along the apex of the heart, and pacemaker leads are visible (Figure 1). Electrocardiogram (EKG) reveals an atrial paced rhythm, along with abnormal ST interval and T waves that may indicate anterolateral ischemia (Figure 2).

On September 8th, the patient was brought to the catheter lab for repositioning of his pacemaker leads. The course was then complicated by atrial fibrillation with a rapid ventricular response, which was resolved with 1000 cc of intravenous fluids (IVF). Patient was discharged on September 18th with prescription of 10 mg prednisone, 0.6 mg colchicine, 10 mg empagliflozin, 0.4 mg tamsulosin, and albuterol. Patient was discontinued on simvastatin and apixaban.

A repeat limited transthoracic echocardiogram (TTE) was ordered, which was taken on September 26th. TTE revealed a LVEF of about 80% and an intracavitary gradient of 33mmBar with Valsalva maneuver, indicating an obstruction. There was no regional left ventricular wall hypokinesis nor pericardial effusion. The bubble study was negative for the presence of a shunt.

February 2nd, 2024 - Follow Up

Patient was seen again and a transthoracic CT was ordered. Comparing chest CT from Sept 23, 2023, CT scan revealed pulmonary emboli in the segmental branches of the right lower pulmonary artery and in the leg segmental branches. Prominent central pulmonary arteries suggest pulmonary hypertension. There was no evidence of a right heart stream, pleural effusion, nor cardiac effusion. The dual chamber pacemaker was in place and visible on CT scan. There is a left ventricular outflow tract (LVOT) gradient, indicating obstruction. There was only a trivial fusion seed.

One month later on March 12th, 2024, the patient was seen again for cardiology follow up. At this time, the patient's assessment disclosed a history of multiple, recurrent DVTs with PEs, despite treatment with an inferior vena cava (IVC) filter. For the patient's pulmonary hypertension, he was asked to continue apixaban 2.5 mg twice a day. Patient was asked to follow up to the electrophysiology clinic in 1 month and return to the clinic in 6 months.

Figure 1. Posteroanterior Chest CT

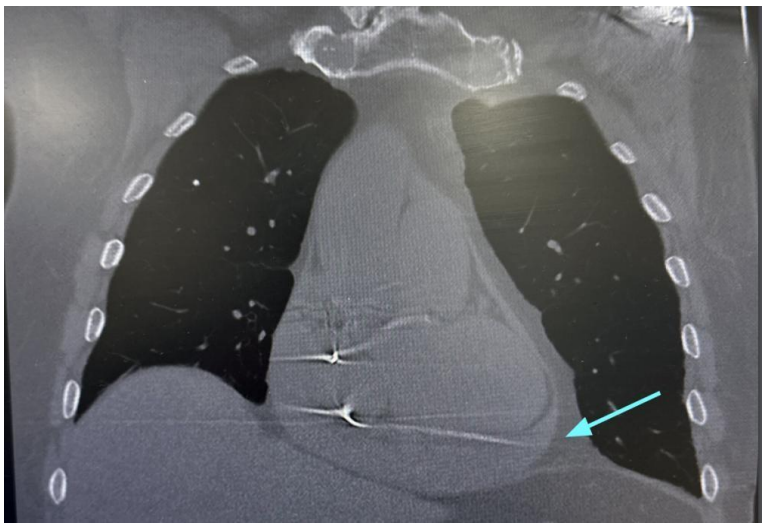

Figure 1. Blue arrow indicating site of pericardial effusion at heart apex. Taken and assessed September 5th, 2023.

Figure 2. Electrocardiogram (ECG)

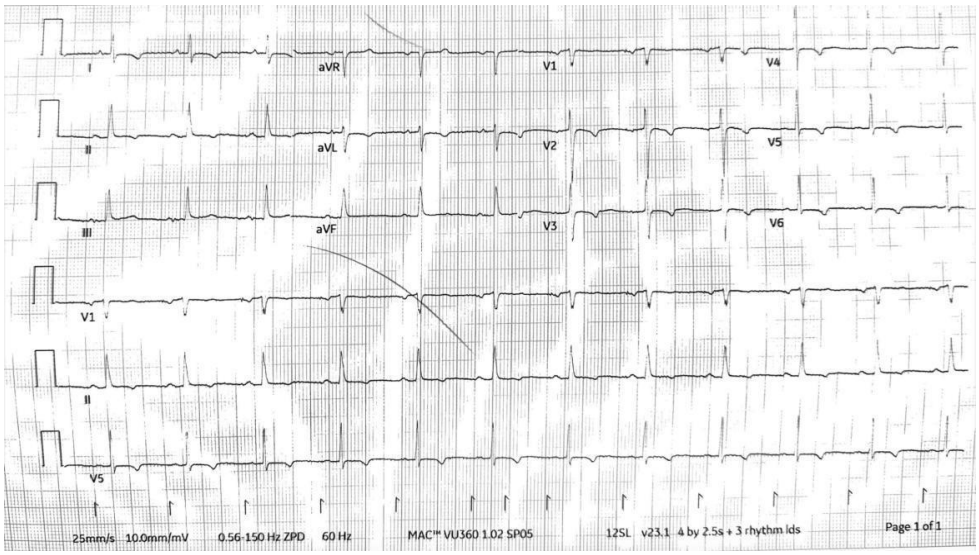

Figure 2. ECG has a regular rate of approximately 75 beats per minute. QRS complexes have normal width, indicating regular sinus rhythm. Leads V1 through V6 indicate ST and T wave abnormalities, either with T wave depressions or missing T waves. Taken and assessed September 5th, 2023.

Discussion

Considering the patient's history and symptoms, a diagnosis of **post-cardiac injury syndrome** (PCIS) is likely. PCIS is a group of autoimmune-mediated conditions that cause inflammation to either the pericardium, epicardium, or myocardium (Sasse & Eriksson, 2017). In this case, PCIS may have resulted from the implantation of a pacemaker, which led to the development of pericarditis and pericardial effusion. With regards to the patient's medical history, the pacemaker was indicated due to the patient's longstanding history of bradycardia (Mulpuru et al., 2017). Although the exact pathogenesis of PCIS is still under investigation, it is currently suspected that antimyocardial antibodies may be involved, especially as these antibody levels are increased following cardiac surgery, or in the case of this patient, pacemaker implantation (Imazio & Hoit, 2013). These elevated antibodies are associated with increased differentiation of heart-localized CD4+ T cells, triggering the rise of inflammatory markers within the heart, particularly around the pericardium and endocardium.

Diagnostically, PCIS is generally characterized as having increased D-Dimer levels and signs of inflammation, which include an increased white blood cell count as well as leukocyte count and elevated C-reactive protein levels. Furthermore, a decreased estimated glomerular filtration rate (eGFR) has been associated with cardiovascular impairment, injury, or surgery as a result of increased oxidative stress and the subsequent dysregulation of the renin-angiotensin-aldosterone system (RAAS) (Ravarotto et al., 2022). Symptomatically, patients suffering from PCIS may also experience chest pain, fever, and pericardial effusion.

Factors contributing to the development of recurrent pericardial effusion due to PCIS may include anticoagulation therapy. Blood thinners are used to interfere with normal clotting processes to prevent coagulation. In this case, the patient was on apixaban, an inhibitor of factor Xa, and warfarin, an inhibitor of clotting factor activation. A study investigating the role of warfarin in late pericardial effusion after cardiac surgery found that large pericardial effusions are more likely in anticoagulated patients, especially if they were given an excessive dosage (Malouf et al., 1993). In addition, a case report with similar presenting symptoms showed a 66-year-old male on warfarin with recurrent pericardial effusion caused by pacemaker lead perforation (Nakanishi et al., 2012). A right auricular appendage wall perforation was found, and it was theorized that warfarin-induced anticoagulation might have dissolved a dislodged fibrin clot leading to the pericardial effusion. Another study hypothesized that warfarin administration could contribute to the recurrent pericardial effusions as the anticoagulative effects predispose the patient to bleeds, including bleeding into the pericardial sac (Limdi et al., 2009). In this case report, the patient's use of anticoagulants may have predisposed him to the recurrent pericardial effusions.

Additionally, the patient's diabetes mellitus (DM) may have contributed to their eventual PCIS complication. T2DM has been associated with greater baseline levels of inflammatory markers, including pro-inflammatory cytokines IL-1β, IL-18, TNF-α, and acute phase reactants like IL-6 and C–reactive protein (CRP) (Lempesis & Georgakopoulou, 2023). While DM has been associated with increased risk for developing pericardial effusion post-chemoradiotherapy for Stage I esophageal cancer (Tamari et al., 2014), there has been limited research on how DM leads to pericardial effusion after cardiac interventions. Therefore, there is little direct evidence demonstrating that DM plays a role in PCIS and pericardial effusion after pacemaker placement. Future studies could investigate DM as a risk factor for pericardial effusion and PCIS to elucidate the underlying mechanisms for such possible connections.

The patient in our case also has a history of chronic kidney disease (CKD). His elevated creatinine at 1.9 mg/dL and low eGFR at 33 upon ER admission indicate continued poor kidney function. CKD may play a role in recurrent pericardial effusions by increasing risk of cardiovascular disease (CVD). Not only do CKD and CVD share similar risk factors like DM and

hypertension, but they also share pathophysiological processes like volume overload and proinflammatory states (Matsushita et al., 2022). Pericarditis may be a complication of uremia secondary to CKD. In uremic pericarditis, the kidneys lose their ability to filter out toxins in the blood, which can cause pericarditis. It generally presents with exudative fluid, with increased LDH levels and decreased lymphocytes. A study conducted by Dad & Sarnak (2016) noted cases of dialysis-associated patients with reduced glomerular clearance and episodes of acute access clotting prior to pericarditis. Additionally, CKD can increase the risk for pericarditis through volume overload. With CKD, decreased kidney function and impaired sodium-water excretion can cause fluid to accumulate in different parts of the body like the pericardium (Rehman et al. 2017). CKD can also cause hypoalbuminemia that lowers oncotic pressure, promoting leakage of fluid into the pericardial space. Eslami et al. (2024) found that hypoalbuminemia and neutrophilia were strongly predicted of pericardial effusions in patients with CKD. In our case, the patient's long-standing kidney disease may have influenced his readmissions to the ER for pericardial effusions and pericarditis.

Ultimately, the combination of the patient's anticoagulation medications, DM, CKD, and pacemaker implantation may have contributed to inflammation of the cardiac region and eventual pericardial effusion. Not only does the RAAS system contribute to the proinflammatory state of CKD, but T2DM also further exacerbates proinflammatory states due to abnormal glucose metabolism and production of reactive oxygen species (Bell et al., 2023). Unfortunately, PCIS is relatively rare, with one study estimating the incidence to be at 3% in acute MI patients (Malik et al., 2021). Thus, it is important to continue studying PCIS cases to further elucidate risk factors and minimize the complications of future pacemaker implantations.

Conclusion

Within two months of pacemaker placement, our patient with a history of previous pericardial effusion four weeks prior presents to the ER with an elevated D-dimer and pericardial effusion. In addition to his recent history of pacemaker placement, our patient exhibits a myriad of risk factors including anticoagulant use, CKD, DM, and previous DVT with PEs that may have exacerbated cardiovascular strain. Based on the patient's history and presentation, the primary differential diagnosis is post-cardiac injury syndrome (PCIS). PCIS is a heterogeneous group of autoimmune-mediated conditions that cause inflammation to either the pericardium, epicardium, or myocardium (Sasse & Eriksson, 2017). This case is unique because the patient has a significant set of comorbidities whose pathophysiology may exacerbate that of another. More specifically, the combination of DM and CKD contributes to a hyper-inflammatory state that can predispose the patient to PCIS and increase cardiovascular strain leading to pericardial effusions (Bell et al., 2023).

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