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RECURRENT CARDIAC TAMPONADE AS A COMPLICATION OF PROLONGED LEFT BUNDLE BRANCH AREA PACING

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ABSTRACT

Post-cardiac injury syndrome (PCIS) is one of the complications of cardiac interventional procedures. This condition has been postulated to be due to an immune reaction, especially with pacemaker lead implantation. Patients with PCIS can have uncomplicated pericardial effusion until they develop cardiac tamponade. The management of PCIS, which can be conservative management or require pericardiocentesis or surgery depends on the patient's clinical symptoms. As the left bundle branch area pacing (LBBAP) implantation technique and technology are still new, it is associated with longer procedural time than conventional pacemaker implantation. Herein, we report a case of PCIS in a patient who had a prolonged LBBAP procedure due to difficult anatomy and presented with recurrent cardiac tamponade.

KEYWORDS

Radiation pericarditis, cardiac implantable electronic device, cardiac tamponade, corticosteroid therapy, left bundle branch area pacing

LEARNING POINTS

- Prolonged fluoroscopy in cardiac procedures can potentially lead to post-cardiac injury syndrome.
- Active pacemaker lead fixation is one of the risk factors for post-cardiac injury syndrome due to immune reaction.
- Left bundle branch area pacing in adults with congenital heart disease is challenging because of anatomical differences.

INTRODUCTION

We report the case of an adult female with congenital heart disease who presented with non-ischemic dilated cardiomyopathy secondary to intermittent complete heart block with junctional escape rhythm. She underwent a complex and prolonged left bundle branch area pacing (LBBAP) procedure due to difficult anatomy and septal fibrosis. Her condition was complicated by post-cardiac injury syndrome (PCIS), which manifested as recurrent cardiac tamponade and responded to aspirin, colchicine, and prednisolone.

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CASE DESCRIPTION

A 20-year-old woman with a known history of patent ductus arteriosus (PDA) and atrial septal defect (ASD) underwent transcutaneous closure of the PDA in 2005 and the ASD in 2014, respectively. She came to our centre complaining of reduced effort tolerance and intermittent chest pain for 1 month. During her initial admission, her blood pressure was 106/76 mmHg, and her pulse rate was 85 bpm, with a displaced apex beat. Her body weight was 35 kg, and her height was 153 cm, giving her a surface area of 1.22 m^2. Other clinical examinations were unremarkable. Her electrocardiogram (ECG) revealed sinus rhythm with intermittent atrioventricular (AV) conduction with junctional escape rhythm *(Fig. 1)*, and the transthoracic echocardiogram (TTE) demonstrated a left ventricular ejection fraction of 38%, dilated left atrium and ventricle, with no residual ASD/PDA. Further investigation using cardiac magnetic resonance imaging (MRI) revealed fibrosis of the interatrial septum and the walls of the right and left atria, with dilation of the left atrium and ventricle *(Fig. 2)*.

The patient was subsequently diagnosed with non-ischemic dilated cardiomyopathy secondary to intermittent complete atrioventricular block. Because she was symptomatic, an implantation of a dual-chamber permanent pacemaker was planned. The initial strategy was to implant right ventricular pacing at the LBBAP to achieve physiological cardiac pacing. Her procedure was performed under conscious sedation, using a Philips Azurion 7 Biplane system (Philips, USA) with a low frame rate (7.5 frames/seconds). We decided to use the Abbott Agilis HisPro™ Steerable catheter with the style-driven lead system for LBBAP.

During the implantation procedure, we encountered difficulty achieving an optimal threshold at LBBAP due to a high pacing threshold, which is most likely due to septal fibrosis. Apart from that, despite using the steerable catheter, we had difficulty stabilizing the catheter while screwing the lead into the septum. After 5 hours of searching for the optimal location of LBBAP and several modifications of the catheter curve with a lesser primary curve angle, we eventually managed to implant at the right ventricle low septal area with narrow QRS complex with a negative deflection in lead II and III during pacing ECG *(Fig. 3)*. The total cumulative fluoroscopy dose area product (DAP) was 45.502 Gy×cm^2, total air Kerma 354 mGy with a fluoroscopy time of 69 min. The patient was discharged after 1 week of post-procedural observation.

Two weeks later, she experienced recurrent chest pain and shortness of breath. Vital signs were blood pressure 98/78 mmHg, pulse rate 100 bpm, afebrile. A cardiovascular examination showed distended neck veins and muffled heart sounds. No mucocutaneous sign suggestive of radiation exposure side effects was noted. The ECG showed ventricular pacing rhythm. Urgent TTE revealed a global massive pericardial effusion measuring 3 cm with evidence of cardiac tamponade. Urgent pericardiocentesis was performed, revealing an exudative pericardial effusion

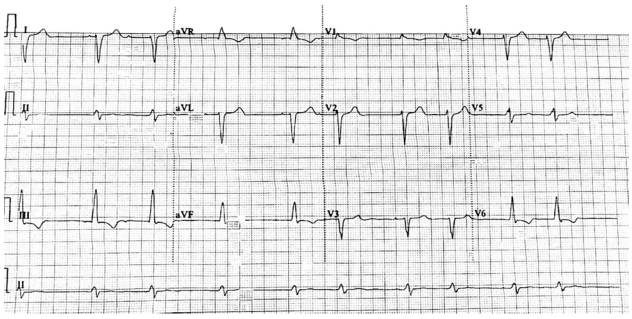


Figure 1. Electrocardiogram showing sinus rhythm with intermittent atrioventricular conduction and presence of junctional escape rhythm.

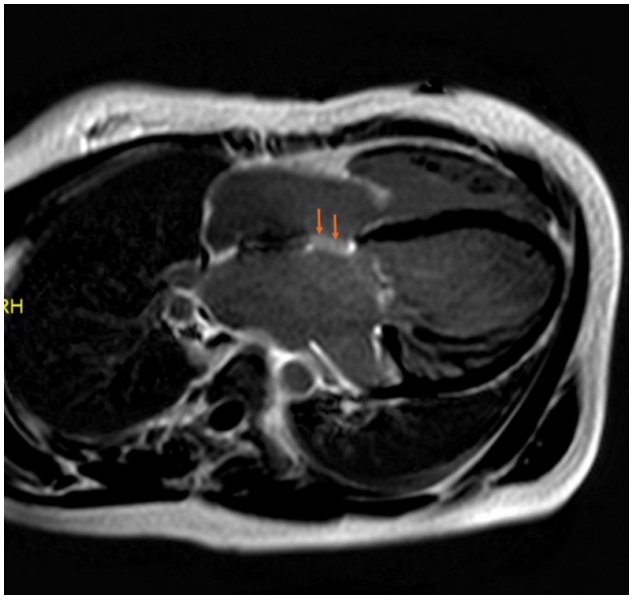


Figure 2. Cardiac magnetic resonance imaging during late gadolinium phase showed enhanced and thickened interatrial septum surrounding the atrial septal defect occluder (red arrow). There is no enhancement in right and left ventricular wall.

without malignant cells. Inflammatory markers, such as C-reactive protein, were negative in two readings, and the erythrocyte sedimentation rate was 7 mm/hr, which is not elevated. Connective tissue disease markers such as antinuclear antibodies (ANA), anti-double-stranded DNA (anti-dsDNA) antibodies, and rheumatoid factor were negative with normal complements levels. Following the recent procedure, the patient was diagnosed with PCIS. We started her on aspirin 150 mg once daily and colchicine 0.5 mg twice daily. The TTE at discharge showed improvement in the pericardial effusion.

However, a week later, she was admitted again with similar symptoms. TTE showed worsening pericardial effusion around the right atrium and left ventricle. During this admission, testing for inflammatory markers was repeated and the results were negative. Cardiac computed tomography (CT) scan showed no evidence of cardiac perforation except a pericardial effusion at the basal inferior wall measuring 1.6 cm *(Fig. 4)*. The patient was started on prednisolone 0.5 mg/kg once daily, which was tapered down weekly with TTE monitoring. Her cardiac failure symptoms improved with serial TTE consistent with minimal pericardial



Figure 3. Cardiac magnetic resonance imaging showing low density pericardial effusion at the left ventricular free wall and right atrium (blue arrow). There was no contrast leak into pericardium noted. Presence of the atrial septal defect occluder with pacing electrode in the right cardiac chamber with streak artefact.

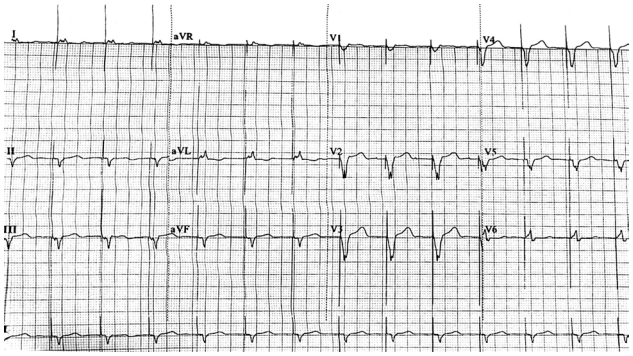


Figure 4. Post procedural electrocardiogram showing paced ventricular rhythm from the right low septal area with a small P wave suggestive of a diseased atrium.

effusion of about 1cm and prednisolone 10 mg once daily was continued for 1 month. Subsequently, we were able to reduce the dose to prednisolone 5 mg once daily for another 2 weeks. TTE showed minimal pericardial effusion of 0.5 cm and was able to stop prednisolone after 1 month. Serial TTE showed minimal pericardial effusion less than 0.5 cm, and she remains stable under outpatient monitoring.

DISCUSSION

PCIS is also known as pericarditis after pericardium injury that causes inflammation to either the pericardium, epicardium, or myocardium. The incidence of PCIS has been reported to be 1–5% after implantation of intra-cardiac devices, with an incidence of cardiac tamponade ranging from 0.1 to 0.8%[1]. The onset of PCIS can vary from hours to days and can occur up to 5–56 days after the procedure. Diagnosis of PCIS is usually based on excluding other possible causes of pericarditis[2].

The clinical presentation of PCIS is variable, including pleural effusion, pericardial effusion, or even acute pericarditis leading to a severe form of cardiac tamponade following invasive cardiac procedures such as coronary angioplasty or transvenous pacemaker implantation, especially in dual chamber mode with active atrial fixation leads[3]. Pericardial effusion can resolve on its own without any treatment in asymptomatic patients. In symptomatic PCIS, the first-line treatment includes aspirin 750 mg every 8 hours for 2 weeks and colchicine 0.5 mg every 12 hours for 6 months. For refractory or recurrent cases, prednisolone at 0.5 mg/kg/day for 4 weeks, followed by a very slow taper, is another option[4]. Surgical intervention with pericardiectomy can also be considered if refractory to medical therapy[5].

The main reason this patient developed PCIS is likely due to prolonged procedure and multiple reattempts of right ventricular lead implantation in the LBBAP. We decided on LBBAP for her intending to achieve cardiac physiological pacing and correct her left bundle branch block. However, it was not successful because of the presence of septal fibrosis as a result of previous ASD device closure.

Interestingly, this patient developed cardiac tamponade after 2 weeks from the initial long pacemaker procedure, and it recurred after 1 week of initial treatment; hence, a cardiac CT scan was done to look for lead perforation. Our patient was placed on aspirin and colchicine as initial therapy. However, she failed to show adequate improvement, as evidenced by recurrent pericardial effusion. Subsequently, she was treated with a course of corticosteroids, which resulted in a marked improvement in her condition. Therefore, it is crucial to evaluate patients regularly at follow-up visits for 1–3 months after the cardiac procedure to avoid missing PCIS and anticipate complications, even though the clinical manifestations may be non-specific[6].

Regular echocardiography and clinical assessments can help detect early signs of recurrent pericardial effusion, allowing for prompt intervention and management. The diagnosis of PCIS following prolonged cardiac implantable electronic device (CIED) implantation procedure was made only after excluding other common aetiologies: infection, autoimmune disease, radiation exposure side effects, or malignancy.

CONCLUSION

This case highlights the challenges in early diagnosis of PCIS due to overlapping symptoms with other conditions like infection and radiation pericarditis. Our case also demonstrates the effectiveness of corticosteroid therapy in treating recurrent pericardial effusion as a complication from a prolonged CIED implantation procedure. It is crucial to explore alternative second-line treatments if the initial therapy fails to prevent severe complications, ensuring timely and effective management. Apart from that, shortening the procedure and fluoroscopy time is one of the important ways to reduce the risk of PCIS.

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