

CASE REPORT

Postcardiac injury syndrome and stroke following permanent pacemaker insertion

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SUMMARY

An 80-year-old woman initially presented with an episode of pleuritic chest pain 10 days after implantation of a dual chamber permanent pacemaker. She returned to hospital a day later with vomiting and fever. She was found to have new atrial fibrillation in addition to right-sided weakness and dysarthria. An infarct in the left anterior inferior cerebellar artery territory was later confirmed on CT. She continued to have recurrent febrile episodes associated with vomiting and dyspnoea. Extensive investigations for infection were negative, and her symptoms were initially attributed to aspiration pneumonia. The patient gradually deteriorated despite antibiotics and became progressively short of breath, with development of large pleural and pericardial effusions. A diagnosis of postcardiac injury syndrome was made after exclusion of other differentials. The patient recovered well after pleurocentesis, pericardiocentesis and a pericardial window, with resolution of symptoms without further medical therapy.

BACKGROUND

Postcardiac injury syndrome (PCIS) is a well-recognised syndrome characterised by pericarditis following injury to the pericardium.¹ Classically, it has been associated with myocardial infarction (Dressler syndrome), cardiac surgery (postpericardiotomy syndrome) or trauma (post-traumatic pericarditis).¹ While there have been reports of PCIS following minimally invasive procedures such as percutaneous coronary intervention or permanent pacemaker (PPM) insertion, this is rare (<1% to 5%).¹⁻⁴ We report a case of PCIS following dual chamber PPM insertion that presented with an embolic stroke secondary to new atrial fibrillation. The patient later developed recurrent pleural effusions and a large pericardial effusion. The case highlights an unusual presentation of PCIS and emphasises the importance of considering this diagnosis following endovascular procedures.

CASE PRESENTATION

An 80-year-old woman presented to a rural hospital with an episode of right-sided pleuritic chest pain. Ten days earlier, she had undergone transvenous insertion of a dual chamber PPM with active fixation leads at a tertiary centre. This was done to manage episodes of syncope associated with sinus bradycardia. She had been otherwise well following the procedure, with no immediate complications. Her medical history was unremarkable, with only

hypertension and hyperlipidaemia. The chest pain was investigated with serial troponins, which were negative. A chest X-ray revealed only mild cardiomegaly, and did not demonstrate lead malposition. An ECG showed that she was in normal sinus rhythm. After a period of observation, the patient was discharged home with analgesia.

The following day, she returned by ambulance after an episode of vomiting associated with altered conscious state. She was noted to be febrile and on examination was found to have new neurological deficits including a right-sided facial droop, arm and leg weakness, as well as dysarthria. An ECG revealed new atrial fibrillation, and she was anticoagulated with low molecular weight heparin to prevent further embolic strokes.

Investigations to determine the cause of her fever and atrial fibrillation were performed, repeated cultures of blood and urine were unremarkable and an ultrasound of the pacemaker site showed only normal postoperative changes. A chest X-ray showed only a small left-sided pleural effusion. Thyroid function was within normal limits. The pacemaker was functioning normally on interrogation. Subsequent CT brain angiography confirmed an infarct in the territory of the left anterior inferior cerebellar artery.

Over the following weeks, the patient was unable to progress with rehabilitation. She continued to have intermittent episodes of fever associated with nausea, vomiting, dyspnoea and rapid atrial fibrillation. The episodes each lasted several days and were associated with raised inflammatory markers (C reactive protein). They were presumed to be recurrent aspiration pneumonia due to her stroke and impaired swallowing and appeared to respond to courses of antibiotics with piperillin/tazobactam.

Nevertheless, the patient gradually deteriorated and became progressively short of breath. Serial chest X-rays showed accumulation of a large left-sided pleural effusion (figure 1). Two litres of straw-coloured pleural fluid was drained using ultrasound-guided pleurocentesis, and this was found to be exudative by Light's criteria. Cytology was non-diagnostic; fungal and bacterial cultures were unremarkable.

Unfortunately, her symptom improvement was short-lived, and within 1 week, she had rapidly reaccumulated bilateral pleural effusions. Sequential bilateral ultrasound-guided pleurocentesis was performed. During the first of these procedures, the patient was noted to have a large pericardial effusion. CT of the chest confirmed that this effusion



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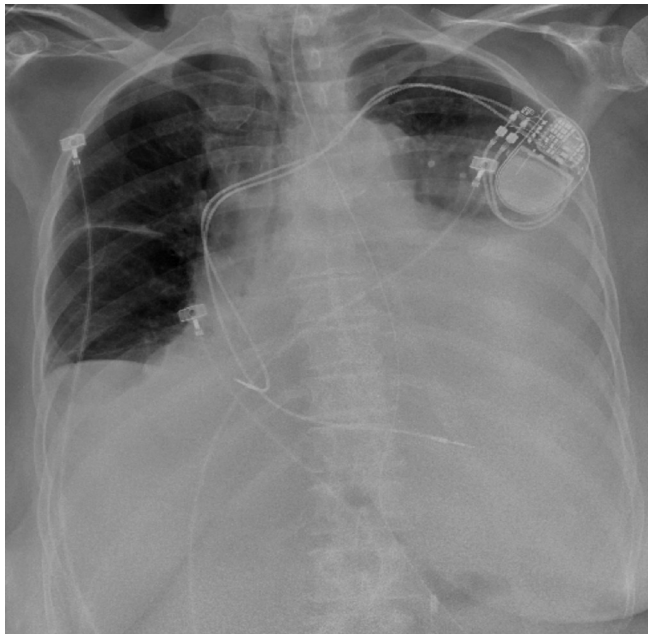


Figure 1 Chest X-ray 1 month into admission demonstrating development of a large left-sided pleural effusion.

was circumferential and 4 cm in depth (figure 2). Importantly, there were no clinical signs of cardiac tamponade. The patient was transferred to a tertiary centre for echocardiography and urgent management.

At the tertiary centre, a transthoracic echocardiogram confirmed the presence of a circumferential pericardial effusion, 4 cm in depth, with no features of tamponade (figure 3). The pacemaker leads were in position, and there were no vegetations suggestive of infective endocarditis. A pericardiocentesis and a pericardial window were performed, draining more than 400 mL of blood-stained pericardial fluid. Significantly, further investigations for an underlying cause were negative. The pericardial fluid was exudative by Light's criteria, cytology was unremarkable, and bacterial, mycobacterial and fungal cultures were negative. Histology from the pericardial tissue showed features of mild acute on chronic reactive fibrous pericarditis with overlying patchy fibrinous exudate. Her autoimmune markers (antinuclear antibody, antineutrophil cytoplasmic antibody, rheumatoid factor and complement C3/4 levels) were within normal limits. A diagnosis of PCIS was reached given other possibilities had been excluded.

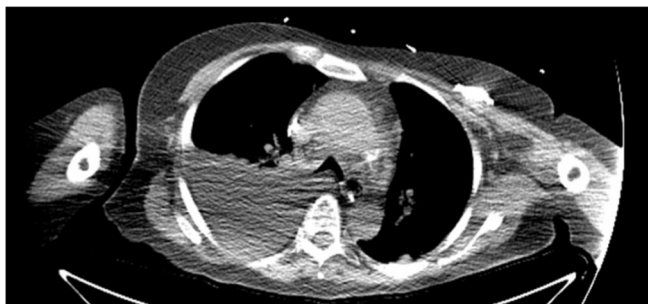


Figure 2 CT after left-sided pleurocentesis. There is a large circumferential pericardial effusion, 4 cm in depth. A large right-sided pleural effusion is also present.



Figure 3 Transthoracic echocardiography confirming the presence of a larger circumferential pericardial effusion, 4 cm in depth.

INVESTIGATIONS

See the Case presentation section.

DIFFERENTIAL DIAGNOSIS

- ▶ Pacemaker lead malposition
- ▶ Pacemaker pocket infection
- ▶ Infective endocarditis

TREATMENT

See the Case presentation section.

OUTCOME AND FOLLOW-UP

Following the pleurocentesis, pericardiocentesis and pericardial window, medical therapy with non-steroidal anti-inflammatory drugs was not immediately commenced in view of the risk of bleeding in the setting of anticoagulation for atrial fibrillation. Her inflammatory markers remained within the normal range. The patient was returned to our facility and carefully observed for signs and symptoms of recurrent pericarditis, with a plan to introduce medical therapy if required. Nevertheless, her symptoms had resolved spontaneously, and over the subsequent 2 months, she had no further episodes of fever, chest pain, dyspnoea or recurrence of effusions. The patient subsequently recovered well and was discharged home after a period of physical rehabilitation.

DISCUSSION

The pathophysiology of PCIS is not completely understood.^{1 5} It is thought to be an autoimmune inflammatory response, triggered by autoantigen release after injury of pericardial and/or pleural mesothelial cells.¹ While most commonly associated with major cardiac surgery (10%–40%),¹ recently there have been increasing reports of the condition occurring after minimally invasive endovascular procedures (<1%–5% incidence).¹

PCIS after permanent electronic cardiac device insertion is rare, with previous reports estimating this incidence at less than 5%.^{2–4} Significantly, our case is consistent with previous studies that suggest female sex, advanced age and the usage of active fixation leads are potential risk factors.^{3 4 6} Active fixation screw-in leads are thought to have a greater propensity to protrude through the thin atrial wall causing pericardial inflammation, and studies where their use has been limited have shown extremely low incidence of PCIS (<1%).⁴

The diagnosis of PCIS is typically one of exclusion and in the context of recent electronic device implantation, other differentials such as device site infection and infective endocarditis need to be ruled out.¹ In particular, lead malposition needs to be considered as a possibility in patients that develop subsequent stroke.^{7,8}

The clinical manifestations of PCIS are non-specific, and the diagnosis is frequently delayed. PCIS may occur days to months following a procedure, and the most common symptoms are pleuritic chest pain (90%), low-grade fever (60%–70%) and dyspnoea (50%–60%).¹ ECG changes associated with pericarditis may be evident in more than 20%.¹ However, only two previous cases of new atrial fibrillation have been reported,³ with none accompanied by embolic stroke. The development of pleural (>60%) and pericardial (>80%) effusions is common, and rarely this can be large and associated with haemodynamic compromise due to tamponade.¹

While up to 60% of PCIS is self-limiting, early recognition and pharmacological management has been demonstrated to increase the rate of remission and reduce the incidence of recurrence.⁹ This may avoid the development of massive pleural or pericardial effusions that require additional surgical management, as occurred in the present case. Non-steroidal anti-inflammatory drugs such as aspirin or ibuprofen, given over a period

of 1–2 weeks and tapered, have been found to be effective.⁹ Colchicine may be added to reduce the risk of recurrence,¹⁰ and corticosteroids can be useful when other therapies fail or are contraindicated.¹¹ While the long-term prognosis is generally benign, follow-up is recommended as there is an intermediate risk (2%–5%) of developing constrictive pericarditis.¹²

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Contributors CB and EW wrote the case study. NU was the clinician involved for the patient care, reviewed the case and supervised the presentation and edited the case study.

Competing interests None declared.

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Learning points

- ▶ Postcardiac injury syndrome should be considered as a cause of symptoms after minimally invasive procedures such as transvenous permanent pacemaker insertion.
- ▶ The condition is typically associated with pleuritic chest pain, fevers and dyspnoea with pleural and/or pericardial effusions. However, unusual presentations such as atrial fibrillation with secondary embolic stroke can occur.
- ▶ The diagnosis is one of exclusion, and investigations are needed to rule out other potentially life-threatening differentials.
- ▶ Early identification and prompt medical management of PCIS reduces the incidence of recurrence and may preclude the need for subsequent surgical intervention.

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