

Recurrent pericardial effusion and tamponade after epicardial pacemaker lead placement: a case report

Danh Q. Nguyen ^{1,2}, Geoffrey D. Huntley^{1,2}, Timothy A. Zaki^{1,2}, and Darren K. McGuire ^{2,3*}

¹Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, TX, USA; ²Parkland Health and Hospital System, Dallas, TX, USA; and ³Division of Cardiology, Department of Internal Medicine, University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75235-8830, USA

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Background

Epicardial pacemaker placement is often necessary in pacemaker-dependent patients with ongoing device pocket infection or lack of venous access. Pericardial effusion and tamponade are rare but serious complications of this procedure.

Case summary

A 38-year-old woman presented with nausea, diaphoresis, and hypotension 7 days after epicardial lead placement. Echocardiography revealed a large pericardial effusion with signs of tamponade. Despite initial improvement after pericardiocentesis, she continued to develop symptomatic pericardial effusions. The patient ultimately underwent pleuro-pericardial window surgery, which resulted in sustained resolution of effusion recurrence.

Discussion

Cases of recurrent pericardial effusion and tamponade following epicardial lead placement have been reported in the literature, although they are rare. While extensive partial pericardiectomy or total pericardiectomy was required to achieve adequate control of fluid accumulation in prior case reports, our patient was successfully managed with a pleuro-pericardial window.

Keywords

Pacemaker • Tamponade • Pericardial effusion • Atrial fibrillation • Systolic heart failure • Electrophysiology • Case report

ESC Curriculum

2.2 Echocardiography • 5.3 Atrial fibrillation • 5.11 Cardiac resynchronization therapy devices • 6.2 Heart failure with reduced ejection fraction • 7.1 Haemodynamic instability

Learning points

- A high index of suspicion is needed to promptly recognize pericardial effusion and tamponade as potential complications of epicardial pacemaker lead placement.
- A variety of pharmacologic, procedural, and surgical treatment modalities should be considered in the management of recurrent symptomatic pericardial effusions.

* Corresponding author. Tel: +1 214 645 7500, Email: Darren.McGuire@UTSouthwestern.edu

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Timeline

Time	Events
Day 0	Admitted to hospital with bacteraemia from right-sided device pocket and lead infection
Day 1	Intravenous antibiotics started Extraction of device generator and transvenous leads from the right chest
Day 9	Debridement of device pocket Blood cultures negative for growth
Day 18	New device generator placed into new subxiphoid pocket
Day 25	Biventricular epicardial lead placement 1st episode of cardiac tamponade Pericardiocentesis with the removal of 1 L of fluid
Day 28	Colchicine 1.2 mg started 2nd episode of cardiac tamponade Repeat pericardiocentesis with the removal of 1.5 L of fluid Prednisone 60 mg and ibuprofen 600 mg started
Day 31	Pericardial window with small-bore chest tube placement
Day 38	Discharge home from hospital with chest tube in place
Day 51	Intravenous antibiotics completed
2 months post-discharge (clinic)	Repeat echocardiogram showed no pericardial effusion Chest tube removed
4 months post-discharge (clinic)	Repeat echocardiogram showed no pericardial effusion

Introduction

Cardiovascular implantable electronic device infection is a challenging problem among pacemaker-dependent patients. Prompt transvenous/endocardial pacemaker re-implantation is often contraindicated due to ongoing infection (e.g. device pocket infection, endocarditis) or limited venous access (e.g. repeated prior infections and re-implantations).¹ An alternative option in such scenarios is surgical implantation of an epicardial pacing system via a subxiphoid approach.² Pericardial effusion/tamponade is an uncommon complication associated with epicardial pacing, and has been scarcely described in prior literature.^{3–5} Herein, we present a rare case of recurrent pericardial effusion after epicardial lead placement that was successfully treated with a surgical pleuro-pericardial window.

Case presentation

A 38-year-old woman who presented to the hospital with 1–2 days of fever and nausea was admitted to our inpatient cardiology service

with severe sepsis and methicillin-sensitive *Staphylococcus aureus* (MSSA) bacteraemia from an infection of her right-sided cardiac re-synchronization therapy with defibrillator (CRT-D) device. Four months prior to this admission, the patient was admitted for left-sided CRT-D pocket infection with MSSA bacteraemia requiring device extraction and re-implantation onto the right side. Her past medical history was also notable for permanent atrial fibrillation (AF) status post-atrioventricular junction (AVJ) ablation, heart failure with reduced ejection fraction [nadir left ventricular (LV) ejection fraction of 15%, which recovered to 35% after ablation] secondary to non-ischaeamic cardiomyopathy (presumed peripartum cardiomyopathy after the birth of her son 21 years prior), Type 2 diabetes, obesity, and hyperlipidaemia. Her outpatient medications included lisinopril 2.5 mg daily, metformin 1000 mg twice daily, metoprolol succinate 12.5 mg daily, rivaroxaban 20 mg daily, spironolactone 25 mg daily, and torsemide 150 mg daily.

On admission, the patient was empirically treated with vancomycin and piperacillin-tazobactam, subsequently narrowed to cefazolin monotherapy based on culture susceptibilities. Transesophageal echocardiogram (TEE) did not reveal any vegetations. Cardiovascular thoracic surgery (CVTS) extracted the device generator and its leads and debrided the infected pocket. Cultures of the device generator and both ventricular leads grew MSSA. Given that this was her second admission for device infection with MSSA bacteraemia, we decided to treat for suspected endocarditis despite the absence of vegetations seen on TEE.

After device removal, the patient remained normotensive and asymptomatic in AF with complete heart block and a junctional escape rhythm generating heart rates ranging 40–60 beats per minute. Bridge therapy with temporary transvenous pacing was deferred given her ongoing bacteraemia and haemodynamic stability. She awaited CRT-D re-implantation in the intensive care unit with transcutaneous pacer pads in place and dopamine readily available should her clinical status deteriorate.

Blood cultures cleared with no growth on Day 9 of antibiotic treatment. After confirming that blood cultures had remained negative for more than 72 h, a replacement device generator was eventually placed into a new subxiphoid pocket. A subxiphoid pocket with biventricular epicardial leads was utilized instead of transvenous leads due to the lack of vascular access resulting from prior pocket infections in the chest wall bilaterally. Transthoracic echocardiogram (TTE) performed after device re-implantation showed a trivial (not haemodynamically significant) pericardial effusion.

On the morning of planned discharge (post-operative Day 7), the patient acutely developed nausea, dizziness, diaphoresis, and malaise. Though she could not characterize her symptoms precisely, she emphasized that she felt as though she was going to die. She was afebrile with a blood pressure of 79/47 mmHg, paced heart rate of 60 beats/min (pacemaker-dependent in permanent AF status post-AVJ ablation), respiratory rate of 20 breaths/min, and oxygen saturation of 98% on 2 L/min of supplemental oxygen. She appeared ill and in acute distress. Her lungs had clear breath sounds on auscultation bilaterally. Jugular venous pressure was elevated from baseline without evident Kussmaul's sign. Her heart rate was regular with distant heart sounds with no appreciable rubs or murmurs. She had a trace pedal and pretibial pitting oedema bilaterally. The remainder of the exam was unremarkable.

Initial laboratory workup was significant for an elevated, up-trending leucocyte count ($19.08 \times 10^9/L$ from $10.33 \times 10^9/L$ the day prior) and stable haemoglobin (10.3 g/dL from 9.7 g/dL the day prior). High-sensitivity troponin T demonstrated a flat trend upon serial measurement (9 and 11 ng/L). Inflammatory markers were elevated with a C-reactive protein of 12.1 mg/dL [reference range (RR): $0.0\text{--}0.5 \text{ mg/dL}$] and an erythrocyte sedimentation rate of 32 mm/h (RR: $0\text{--}20 \text{ mm/h}$). Anti-nuclear antibody, C3 complement, C4 complement, and thyroid-stimulating hormone levels were within normal limits. Electrocardiography revealed AF with complete heart block and 100% ventricular pacing at 60 beats/min. Chest radiograph showed marked enlargement of the cardiac silhouette, which was unchanged from prior (see [Supplementary material online, Figure S1](#)). Immediate point-of-care ultrasound revealed a large pericardial effusion with right ventricular (RV) collapse. Emergent TTE demonstrated an LVEF of 30–35% and confirmed a moderate to large (1.8 cm posteriorly) circumferential pericardial effusion with diastolic RV collapse and systolic right atrial (RA) collapse concerning tamponade ([Figure 1](#) and [Video 1](#)).

The patient received a 1 L bolus infusion of lactated ringer's and the cardiac catheterization lab was activated for emergent pericardiocentesis. Approximately 1 L of serous fluid was removed and a pericardial drain was left in place. Her blood pressure immediately normalized to 117/76 mmHg and her symptoms resolved. Analysis of the pericardial fluid showed a normal nucleated cell count ($1075/\mu\text{L}$; RR: $\leq 1999/\mu\text{L}$) with a neutrophilic predominance (51%; RR: 25–36%). Pericardial fluid protein was 5.6 g/dL, while serum protein was 5.5 g/dL. Cultures of the pericardial fluid did not grow bacteria or fungi. In hopes of preventing further pericardial fluid accumulation, the patient was started on twice daily colchicine (1.2 mg). Because a large pericardial effusion could cause lead dislodgement, we obtained a computed tomography scan of the chest,

which confirmed the appropriate position of five epicardial electrodes: one bipolar/bifurcated lead and one unipolar back-up lead remained secured to the LV, and one bipolar/bifurcated lead remained secured to the RV.

Three days after pericardiocentesis, output from the pericardial drain ceased and the drain was removed. Unfortunately, later that night the patient again became nauseous and hypotensive with a blood pressure of 85/59 mmHg. Emergent TTE demonstrated re-accumulation of the pericardial effusion with early systolic RA invagination and late diastolic RV collapse, suggestive of elevated intracardiac filling pressures and impending tamponade ([Figure 2](#) and [Video 2](#)). Another emergent pericardiocentesis was performed with removal of $\sim 1.5 \text{ L}$ of serous fluid and placement of a pericardial drain. Her symptoms and hypotension again resolved immediately during the procedure. Because the effusion continued to re-accumulate on colchicine monotherapy, we added prednisone (60 mg daily) and ibuprofen (600 mg daily) to the patient's medical regimen.

Transthoracic echocardiogram performed on the next day showed an interval increase in the size of the pericardial effusion but no signs of tamponade. Given the continuous re-accumulation of pericardial fluid and recurrent episodes of tamponade, CVTS created a transthoracic pleuro-pericardial window and placed a small-bore chest tube into the pericardial and left pleural spaces. Drainage from the chest tube gradually declined over the following week. Transthoracic echocardiogram performed the day before discharge did not show a significant pericardial effusion ([Video 3](#)). The patient was discharged home in stable condition with instructions to evacuate fluid from her chest tube every other day. Her medications at discharge included a 3-month course of colchicine (1.2 mg twice daily), a 1-month course of ibuprofen (60 mg daily), and a prednisone taper. Intravenous cefazolin was continued at home until 6 weeks after blood cultures were first negative.

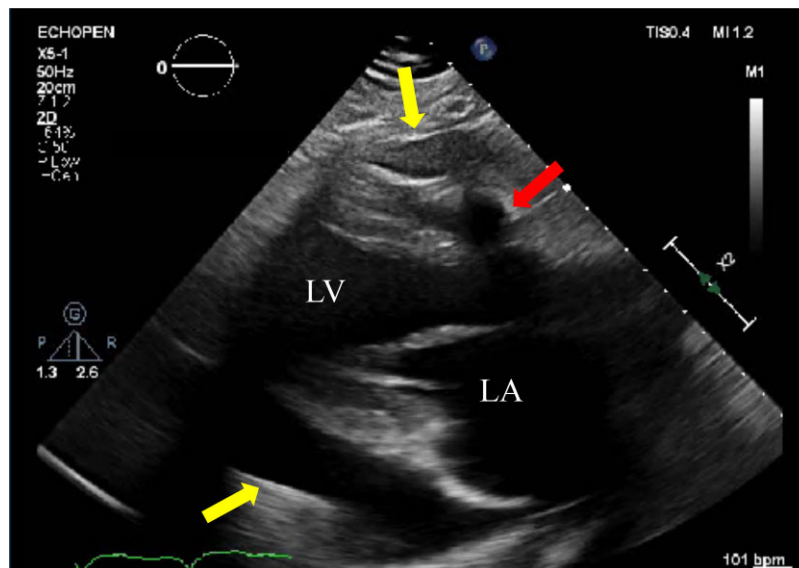
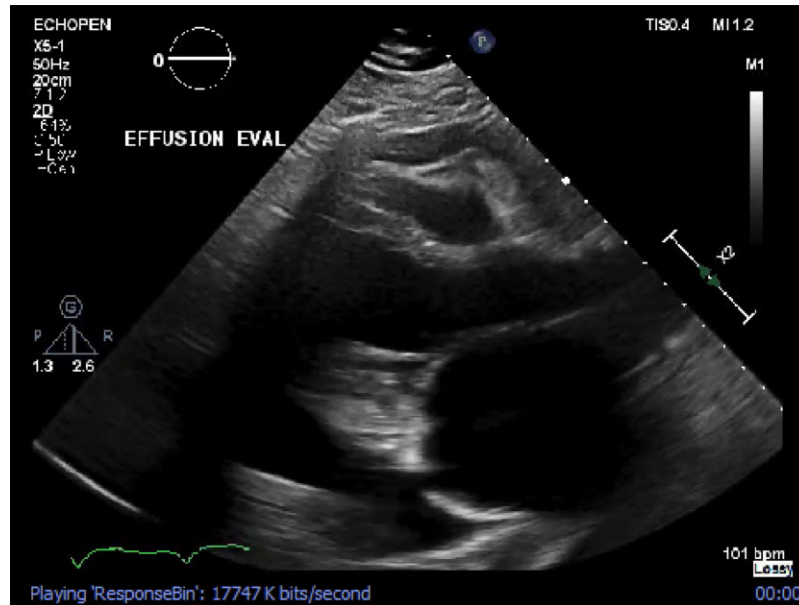


Figure 1 Pericardial effusion and tamponade following epicardial lead placement. Transthoracic echocardiogram in parasternal long axis demonstrates circumferential pericardial effusion (top and bottom arrows) and right ventricular collapse (middle arrow) in early diastole. LA, left atrium; LV, left ventricle.



Video 1 Pericardial Effusion and Tamponade Following Epicardial Lead Placement. Transthoracic echocardiogram in parasternal long axis demonstrates circumferential pericardial effusion and right ventricular collapse in early diastole.

At 1-month follow-up in CVTS clinic, the patient was stable, asymptomatic, and was tolerating her chest tube, which had minimal drainage (<25 mL per week). She reflected on how frightening her experience in the hospital had been and expressed gratitude that her acute illness had largely resolved. At 2-month follow-up appointment, repeat TTE

did not show effusion recurrence, and the chest tube was removed. At 4-month follow-up, the patient reported feeling well and surveillance TTE was again negative for pericardial effusion recurrence (see [Supplementary material online, Videos S1 and S2](#)). Epicardial lead settings were kept the same, with RV and LV amplitudes set to 3 V.

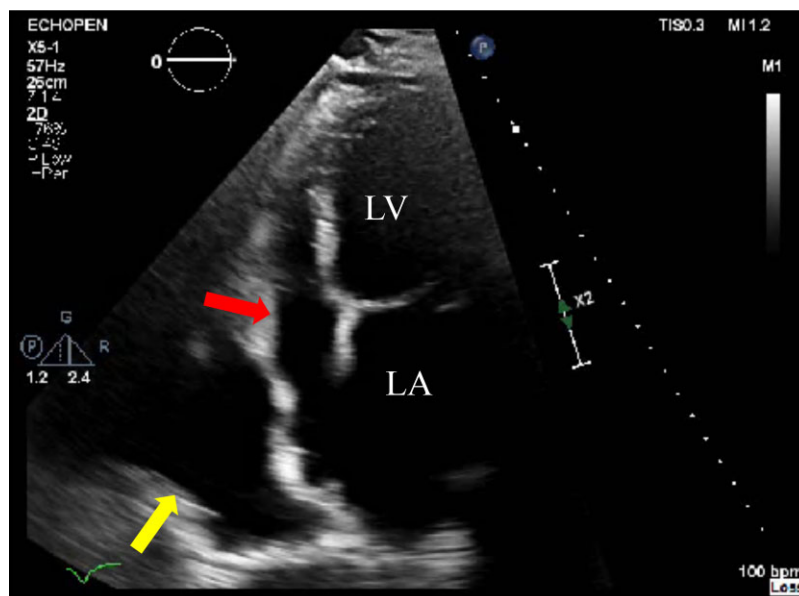
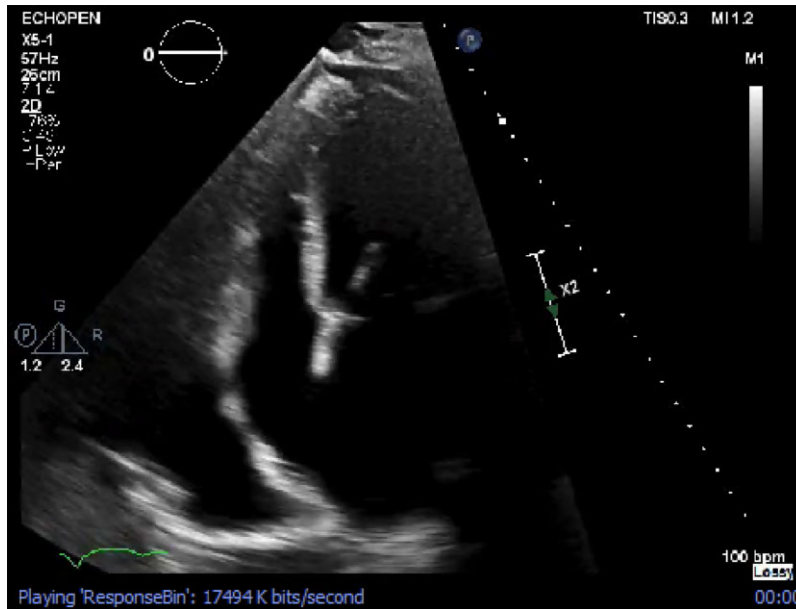


Figure 2 Re-accumulation of pericardial effusion after pericardial drain removal. Transthoracic echocardiogram in apical four chamber view demonstrates posterior pericardial effusion (bottom arrow) and right atrial invagination (top arrow) during early systole. LA, left atrium; LV, left ventricle.

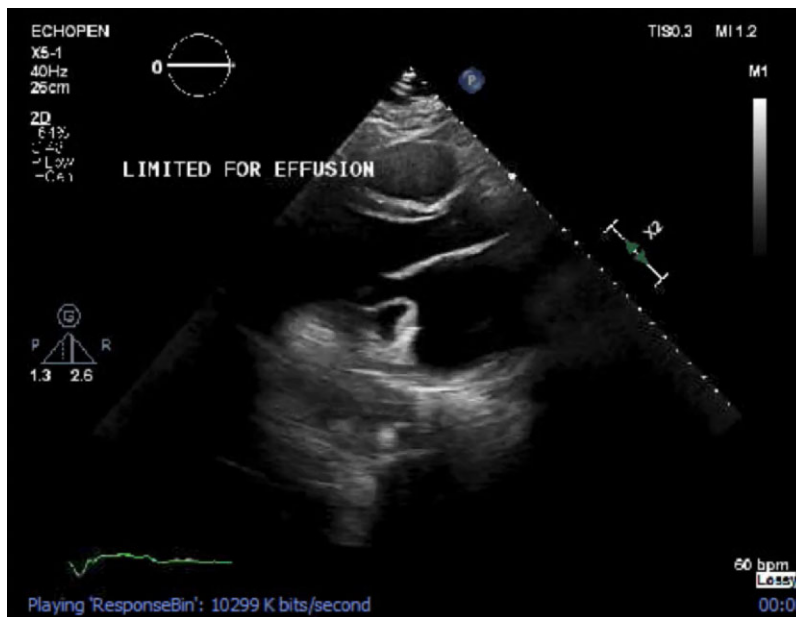


Video 2 Transthoracic echocardiogram in apical four chamber view demonstrates posterior pericardial effusion and right atrial invagination during early systole.

Discussion

This case illustrates the high index of suspicion needed to quickly identify and treat cardiac tamponade following epicardial pacemaker lead placement. What made the diagnosis of tamponade unclear in this patient were the competing, already established diagnoses that warranted her hospital admission and epicardial device placement

in the first place: sepsis and decompensated heart failure. Further confounding our initial clinical assessment was the absence of tachycardia as is usually present with tamponade, but this patient had AF/AVJ ablation and pacemaker dependence. Moreover, pericardial effusion and tamponade as a complication of epicardial lead placement have been rarely described—to our knowledge, only three cases of this uncommon phenomenon have been reported.³⁻⁵



Video 3 Transthoracic echocardiogram in parasternal long axis without evidence of pericardial effusion.

We considered several mechanisms by which these effusions may have occurred. Serum and pericardial fluid laboratory parameters were not suggestive of malignancy, uraemia, myocardial infarction, haemopericardium (e.g. from cardiac perforation by a lead), connective tissue disease, infection, or hypothyroidism as underlying aetiologies. We hypothesize that this presentation of epicardial pacemaker lead-associated pericardial effusion represents a case of post-pericardiectomy syndrome (PPS). Post-pericardiectomy syndrome is characterized by a provoking injury to or invasion of the pericardium with subsequent development of acute pericarditis and/or pericardial effusion.⁶ To explore this possibility further, we obtained a serum anti-myocardial antibody screen, which can be elevated in PPS.⁷ Although the patient's antibody screen was negative, the presence of elevated inflammatory markers and onset of effusions 7 days after pericardiectomy and epicardial lead placement are highly suggestive of this diagnosis.

We elected to treat empirically with colchicine, ibuprofen, and prednisone based on evidence extrapolated from patients with acute pericarditis and PPS.^{8,9} However, given the relatively short time frame between initiation of these medications and final surgical intervention, it is unclear what effect, if any, these medications had on the patient's condition in the acute setting. We prescribed a prolonged course of colchicine for the patient upon discharge since it has been shown to help prevent post-pericardiectomy pericarditis, though its effect on preventing post-pericardiectomy effusion remains inconclusive.¹⁰

In prior case reports of epicardial lead-associated pericardial effusion/tamponade, either extensive partial pericardiectomy or total pericardiectomy was required to achieve adequate control of fluid accumulation.^{3–5} While total pericardiectomy was considered as a definitive therapy to treat this patient's recurrent pericardial effusions, an alternative option was the creation of a pleuro-pericardial window, in which a small fenestration in the pericardium is created to allow fluid to drain into the thorax. Prior studies have shown that pericardial windows are associated with a lower rate of effusion recurrence when compared with pericardiocentesis alone.¹¹ Due to the greater morbidity associated with total pericardiectomy, CVTS decided to attempt a pericardial window first, which ultimately proved to be sufficient. This case report supports the notion that less invasive measures (e.g. pericardiocentesis and medical management, followed by pericardial window if necessary) should be attempted prior to consideration for total pericardiectomy.

How might we avoid this complication in the future? For patients with an indication for CRT who lack upper extremity venous access (as was the case with our patient), there is a novel alternative to epicardial pacing that circumvents the need for pericardiectomy to altogether: totally leadless CRT, which involves endocardial pacing through wireless electrodes implanted directly into each ventricle.¹² However, an important limitation to totally leadless CRT currently is a lack of availability in most health systems (including ours).

Conclusion

We report a rare case of recurrent pericardial effusion and cardiac tamponade following epicardial lead placement, which was most likely due to PPS. Clinicians should consider tamponade in any hypotensive patient with recent pericardial manipulation and proceed with pericardiocentesis after confirming the diagnosis. A variety of medical

and surgical treatment modalities are available to help prevent effusion recurrence.

Lead author biography



Danh Nguyen earned his medical degree at the University of Texas Southwestern Medical Center (US), where he is currently a first-year resident physician training in internal medicine.

Supplemental material

Supplementary material is available at *European Heart Journal—Case Reports* online.

Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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