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Cardiac tamponade and constrictive pericarditis due to *Actinomyces meyeri* bacterial pericarditis: a case report

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Background	Purulent bacterial pericarditis (PBP) is a highly lethal infection of the pericardial space that arises as a complication of infective illnesses. Purulent bacterial pericarditis remains a diagnostic challenge given its non-specific clinical and investigative features and carries exceedingly high mortality rates due to fulminant sepsis and morbidity including constrictive pericarditis in survivors. We present our management of cardiac tamponade and subsequent constrictive pericarditis due to *Actinomyces meyeri* PBP.
Case summary	A 53-year-old Caucasian male presented with acute New York Heart Association Class IV dyspnoea and chest discomfort, in the context of multiple hospital presentations over the preceding 8 weeks due to presumed recurrent viral pericarditis. On this admission, initial transthoracic echocardiography (TTE) demonstrated a large asymmetric pericardial effusion for which he underwent urgent pericardiocentesis. Serial TTE post-pericardiocentesis, however, demonstrated effusion re-accumulation and effusive-constrictive pericarditis, confirmed on cardiac magnetic resonance imaging. Fluid culture was positive for *A. meyeri*. He was diagnosed with PBP, but his condition deteriorated despite appropriate intravenous antibiotic therapy, necessitating semi-urgent surgical pericardiectomy. He recovered well and was discharged on Day 10 post-operatively.
Discussion	Unlike uncomplicated acute viral or idiopathic pericarditis, PBP portends a very poor prognosis if unrecognized and untreated. Diagnostic challenges persist given its rarity in modern clinical practice; however, PBP should be considered in cases of seemingly recurrent pericarditis. Multi-modal cardiac imaging and careful analysis of pericardial fluid including cultures and lactate dehydrogenase/serum ratios may assist in earlier recognition. In this case, source control and symptom relief were achieved only with combined intravenous antibiotics, surgical evacuation, and pericardiectomy.
Keywords	Tamponade • Constrictive • Cardiomyopathy • Chest pain • Echocardiography • Pericardial effusion • Case report
ESC Curriculum	2.3 Cardiac magnetic resonance • 2.2 Echocardiography • 6.6 Pericardial disease • 6.7 Right heart dysfunction • 7.5 Cardiac surgery

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Learning points

- Purulent bacterial pericarditis (PBP) is an uncommon cause of pericarditis that should be considered in cases of seemingly recurrent viral pericarditis, and when effusive-constrictive pericarditis is suspected.
- There should be early suspicion for PBP prior to culture results if lactate dehydrogenase (LD) and fluid/serum LD ratio are elevated on pericardial fluid biochemistry.
- Percutaneous pericardiocentesis is likely to be ineffective. Combined antibiotic therapy and surgical pericardiectomy is likely required.

Introduction

Purulent bacterial pericarditis (PBP) is a highly lethal infection of the pericardial space that arises as a complication of infective illnesses.[1] Whilst the use of antibiotics in the modern era has reduced its incidence, PBP continues to have high associated mortality due to its rapidly progressive nature compounded by delayed treatment initiation because of its indolent nature and non-specific presenting symptoms including chest discomfort, dyspnoea, malaise, and fevers, thus limiting clinical recognition. Even with appropriate antimicrobial treatment, PBP can precipitate complications including effusive-constrictive pericarditis, a syndrome caused by pericardial effusion constricted by the visceral pericardium[2] and cardiac tamponade.

Common causative organisms of PBP include *Staphylococcus*, *Streptococcus*, *Haemophilus*, and *Mycobacterium tuberculosis*.[3] Purulent bacterial pericarditis as a result of *Actinomyces meyeri*, a commensal organism of gastrointestinal and urogenital tracts, is rare with only three reported cases of Actinomyces PBP with resultant tamponade.[4–6] We present our management of a patient with *A. meyeri* bacterial pericarditis-induced cardiac tamponade and constrictive pericarditis. Combined antibiotic therapy, surgical evacuation, and pericardiectomy was ultimately required for definitive control.

Timeline

Date	Clinical event
18 September 2020	The patient was admitted under Cardiology after presenting to ED with 2 weeks of acute NYHA IV dyspnoea and chest discomfort, tachypnoea respiratory rate (24 breaths/min), tachycardia (heart rate 112 beats/min), and elevated inflammatory markers (CRP 105.8 mg/L). Multiple hospital attendances in the preceding 8 weeks for similar symptoms were attributed to recurrent viral pericarditis
	Admission TTE demonstrated large (2.4 cm) asymmetric pericardial effusion around the RV, with borderline significant respiratory inflow variation and low-normal LV function, in keeping with cardiac tamponade
	Urgent pericardiocentesis was performed, draining 150 mL haemoserous fluid with immediate symptom relief and haemodynamic improvement (heart rate 90 beats/min, respiratory rate 18 breaths/min)
	Pericardial fluid was xanthochromic, with abundant neutrophils. No malignant cells or lymphoid population were detected
	Biochemistry demonstrated a protein level of 46 g/L, cholesterol 1.7 mmol/L, and lactate dehydrogenase 3584 U/L
19–20 September 2021	The patient remained clinically stable. A small-volume straw-coloured fluid and some debris were noted to be draining from the pericardial drain
	Repeat TTE demonstrated an echodense space (1.3 cm) adjacent to a non-collapsing RV; however, the RV free wall appeared adhered/akinetic. The patient was noted to have tricuspid inflow variation ~35% and mitral inflow variation ~23%, with features of constriction including lateral E′ < septal E′, ventricular interdependence, and dilated IVC with very limited collapse
	The patient's pericardial drain was removed. The clinical team was subsequently notified later that day about the identification of gram-positive bacillus in pericardial fluid culture. The administration of i.v. vancomycin antibiotic commenced
	On further history, the patient reported intermittent nocturnal fevers and sweats for the preceding month, which had been attributed to viral upper respiratory tract infection
21–22 September 2021	The patient reported mild dyspnoea but no further chest pain, fevers, chills, or rigours
	Septic screening including three sets of blood cultures, urine culture, and orthopantomogram showed negative results. Inflammatory markers showed improvement—WCC 7.84×10^9/L, neutrophils 5.3×10^9/L, CRP 94.6 mg/L
	A repeat TTE demonstrated an unchanged echodense space adjacent to RV, with persistent ventricular interdependence but worsening mitral inflow variation (now 35%)
	Cardiac magnetic resonance (CMR) imaging demonstrated pericardial effusion adjacent to the RV free wall, with the highest dimension of 2.6 cm, and associated marked thickening and fibrosis of the pericardium, in keeping with constrictive effusive pericarditis

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Date	Clinical event
23 September 2021	A repeat TTE was done for increasing pericardial effusion (1.8 cm) around RA/RV, with a septal shift in the presence of the adhered right ventricular free wall. IVC was fixed and dilated
	The clinical team notified the patient about the identification of *Actinomyces meyeri* in pericardial fluid. Antibiotics was changed to targeted i.v. benzylpenicillin
	Given concerns regarding constrictive pericarditis and worsening TTE findings, referral was made to the Cardiothoracic Surgery Department regarding the role and timing of surgical pericardiectomy and drainage
24–25 September 2021	The patient remained clinically good with a down-trending CRP of 52.9 mg/L
	Discussions were held at a multidisciplinary meeting on surgical timing and pre-surgery antibiotic duration due to the risk of sternal wound contamination. A further 72 h of monitoring and the establishment of long-term peripheral cannula were planned; also outpatient surgical management was planned if the patient remained in good condition
26–28 September 2021	The patient reported worsening significant exertional dyspnoea (oxygen saturation 93–94% on room air), with an up-trending CRP 90.4–113.1 mg/L. The case was rediscussed with the Cardiothoracic Surgery Department and inpatient surgical intervention was planned
29 September–3 October 2021	The patient underwent surgical pericardiectomy and a washout of pericardial collection and was transferred to the ICU post-operatively; he was extubated on Day 2 post-operatively.
	Surgical culture aspirate culture: No growth on aerobic and anaerobic incubation was seen after 10 days
	Surgical culture aspirate microscopy: No bacteria was seen.
	Surgical pathology report: The report showed organizing fibrous and fibrinous pericarditis. The pericardium was thickened by a rind of fibrous tissue that merged with a surface of organizing granulation tissue with overlying fibrinous exudate. Reactive stromal cells and endothelial cells were noted, but there was no evidence of malignancy
	Surgical tissue acid-fast bacilli culture: no growth after 6 weeks
	Surgical tissue acid-fast bacilli microscopy: not detected—negative smear
	Blood, urine, and sputum cultures: negative
4 October 2021	The patient was transferred to the Cardiothoracic Surgery ward. He remained clinically stable with a down-trending CRP of 99.2 mg/L
5–8 October 2021	The patient experienced febrile episodes (temperature 38.2°C) but had a stable CRP 105 mg/L. He was reviewed by Infectious Diseases—there was no overt septic source or any evidence of drug fever, and, therefore, no changes were made to antibiotic therapy
	Three sets of blood cultures, urine culture, and chest drain tip culture were found to be negative
	A repeat TTE demonstrated a small pericardial space with no obvious effusions or vegetations, a normal RV size, and a preserved LV function
9th–10th October 2021	No further febrile episodes were found and CRP improved (58.1 mg/L). The patient was discharged with a plan for administering more i.v. and p.o. antibiotics

WCC, white cell count ($\times 10^9$/L); CRP, C-reactive protein (mg/L); NTproBNP, N-terminal pro-B natriuretic peptide (ng/L); LD, lactate dehydrogenase (U/L); TTE, transthoracic echocardiogram; CTPA, computed tomography pulmonary angiogram; RA, right atrium; RV, right ventricle; LV, left ventricle; IVC, inferior vena cava; i.v., intravenous; ICU, intensive care unit; NYHA, New York Heart Association.
Normal reference ranges: respiratory rate, 12–20 breaths/minute; heart rate, <100 beats/min; CRP, 0–8 mg/L; pericardial fluid total protein, <30 g/L; pericardial fluid cholesterol, <1.2 mmol/L; pericardial fluid lactate dehydrogenase ratio, <0.6; pericardial fluid lactate dehydrogenase, <300 U/L; white cell count, 4–11×10^9/L; neutrophils, 1.8–7.5×10^9/L; oxygen saturation, >94% on room air; temperature, ≤37°C.

Case presentation

A 53-year-old Caucasian male represented to hospital with a 2-week history of acute New York Heart Association Class IV dyspnoea, reduced exercise tolerance, and intermittent chest pain. Of note, he had multiple hospital attendances in the preceding 8 weeks, for similar symptoms attributed to recurrent viral pericarditis in the context of a recent viral illness. He had been managed with non-steroidal anti-inflammatories, colchicine, and 4 weeks of prednisolone for this. His medical history included asthma with infrequent exacerbations, allergic rhinitis, hypertension, hypercholesterolaemia, obesity, and treated obstructive sleep apnoea. There was no history of recent surgery, travel, ill contacts, or active dental infection.

On examination, he was afebrile, normoxic, and normotensive, but was tachycardic (heart rate 112 beats/min) and tachypnoeic (respiratory rate 24). Cardiorespiratory examination revealed soft dual heart sounds and decreased breath sounds at bilateral lung bases, but no pulsus paradoxus. Jugular venous pressure was not elevated. Oral cavity examination noted multiple previous fillings, but overall dental health was adequate with no over gingival inflammation or dental abscesses or decay.

Electrocardiogram demonstrated sinus tachycardia with no diffuse changes or electrical alternans. Blood investigations revealed normal white cell count [7.32×10^9/L (reference range: 4–11×10^9/L)] but
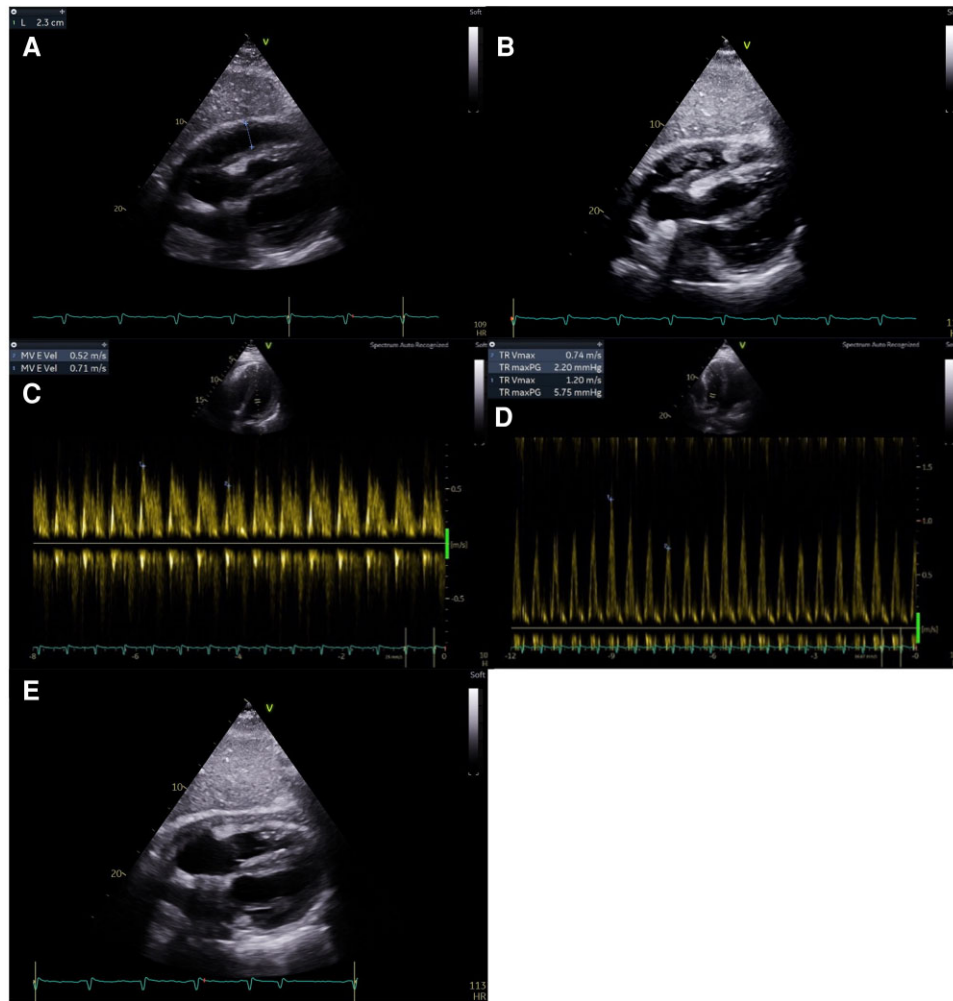
Figure 1 Admission transthoracic echocardiography demonstrating tamponade pre-pericardiocentesis. (*A*) Large pericardial effusion around the right ventricle, (*B*) loculated collection with significant mitral and tricuspid inflow variation (*C* and *D*). (*E*) Post-pericardiocentesis transthoracic echocardiography demonstrating a significantly reduced effusion size.

elevated C-reactive protein [CRP; 106 mg/L (reference range: 0–8 mg/L)]. Chest X-ray, N-terminal pro-B-type natriuretic peptide (NTproBNP), and autoimmune screens were unremarkable.

Initial transthoracic echocardiogram (TTE) revealed mild left ventricular (LV) dysfunction with a large (2.4 cm) asymmetric pericardial effusion containing organized echogenic material adjacent to the right ventricle (RV). There was associated diastolic collapse and significant tricuspid and mitral inflow variation (62 and 27%, respectively). Inferior vena cava (IVC) was dilated and fixed (*Figure 1A–D* and Supplementary material online, *Video S1A*).

Urgent pericardiocentesis was performed. A total of 150 mL of blood-stained fluid was drained with symptom relief (*Figure 1E* and Supplementary material online, *Video S1B*). Pericardial fluid biochemistry revealed lactate dehydrogenase (LD) 3584 U/L (reference range <300 U/L); fluid/serum LD ratio 12 (reference range <0.6); cholesterol concentration 1.7 mmol/L (reference range <1.2 mmol/L). Cultures were sent for testing. A pigtail catheter was placed for further drainage.

The catheter was removed 48 h later due to clinical improvement, minimal output (total 240 mL drained), and improved inflammatory markers (CRP 95 mg/L, 79 mg/L, 53 mg/L 1, 2, and 4 days post-drainage). Notably, purulent debris in the catheter was observed before removal.

Repeat TTE demonstrated only a small residual effusion (1.3 cm). Concerningly, however, the RV free wall now appeared akinetic and adherent, with ventricular interdependence and a minimally collapsing dilated IVC.

Unexpectedly, pericardial fluid cultured gram-positive bacillus 48 h post-drainage. Intravenous (i.v.) vancomycin 2 g 12 hourly was initially commenced and changed to targeted i.v. benzylpenicillin 1.8 g 4 hourly on the identification of *A. meyeri* 96 h later. Fungal and acid-fast bacilli cultures were negative. No malignant cells were identified. Extensive workup with serial urine and blood cultures were negative. Orthopantomogram (OPG) demonstrated previous dental restoration and some caries disease, but no periapical lucence, abscess, or periodontal ligament widening.

Repeat TTE, however, demonstrated increasing pericardial effusion (1.8 cm) and worsening tricuspid and mitral inflow variation (35 and 36%, respectively) (*Figure 2A and B* and Supplementary material online, *Video S2A–C*). Further characterization with cardiac
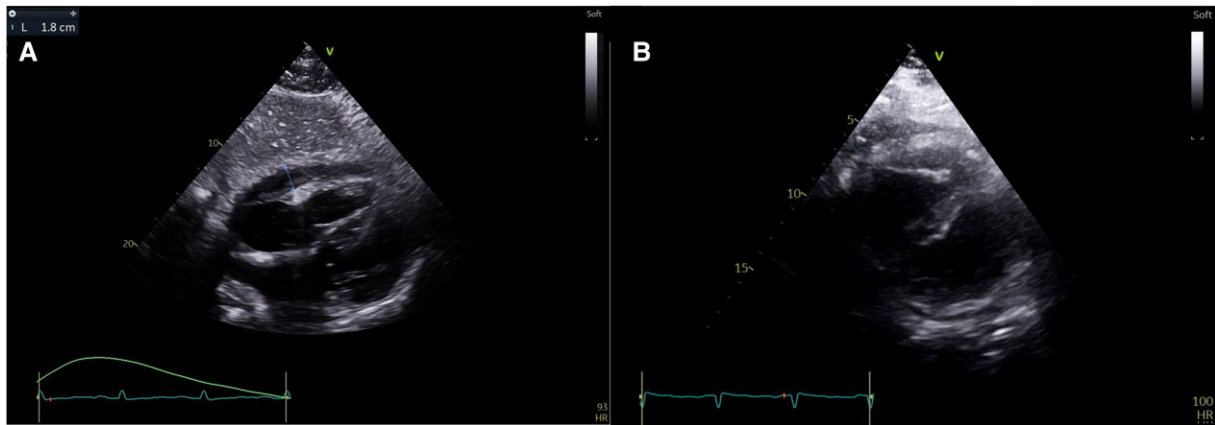
Figure 2 Transthoracic echocardiography 24 h post-drain removal demonstrating (*A*) increasing collection around right ventricle and (*B*) D-shaped flattening suggestive of increased right heart pressure.

magnetic resonance (CMR) imaging confirmed 2.6 cm pericardial effusion adjacent to the RV free wall, with hyperenhancement of the involved markedly thickened pericardium on late gadolinium acquisitions. Free breathing acquisitions demonstrated diastolic septal flattening during expiration consistent with constriction, and there was a mild reduction of LV- and RV end-diastolic volumes and mild biventricular dysfunction (*Figure 3A and B* and Supplementary material online, *Video S3A and B*). There was no evidence of pulmonary infection. He was referred for consideration of surgical source control. Owing to concerns regarding optimal antibiotic duration prior to surgery, his case was also discussed at a multidisciplinary meeting involving Cardiology, Cardiothoracic Surgery, and Infectious Diseases specialists. He was initially deemed suitable for outpatient surgical management. However, clinical deterioration with the development of significant exertional dyspnoea and escalating inflammatory markers despite maximal appropriate antibiotic therapy necessitated semi-urgent inpatient surgery.

Median sternotomy was performed revealing significant collection overlying RV, which was adhered to the thickened pericardium (*Figure 4*). Samples were taken for histopathology and microbiology. The pericardium was incised and a 100–200 mL frank pus was drained, before thorough washing. The sternum was protected from contamination throughout. Pericardium was then resected over the RV whilst preserving the phrenic nerve, around to the LV anterior surface. The right pleura was opened to allow drainage of potential effusions prior to right pleural, pericardium, and mediastinal drain insertions before sternal closure. The drains were removed on Day 5 post-pericardiectomy.

Histopathology confirmed organizing fibrinous pericarditis, with reactive stromal and endothelial cells but no malignant cells. There was no bacterial growth on prolonged aerobic and anaerobic incubation. Day 6 post-operative TTE demonstrated a small pericardial space with no effusion and a preserved LV function (*Figure 5A and B* and Supplementary material online, *Video S4A and B*).
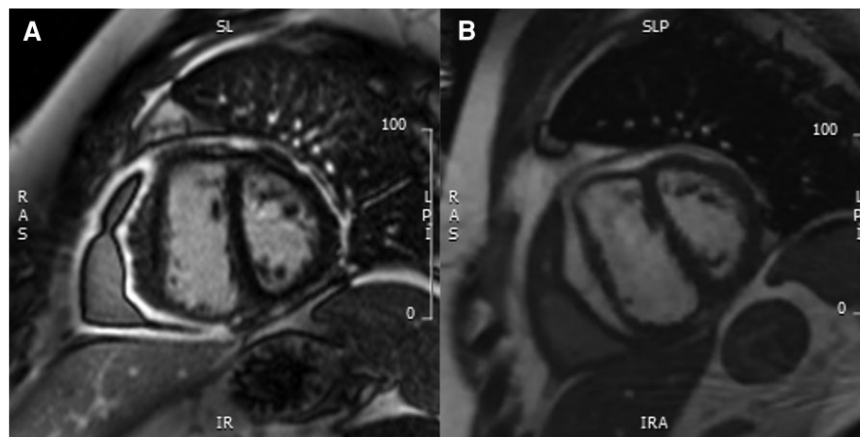
Figure 3 Cardiac magnetic resonance demonstrating residual focal effusion adjacent to the right ventricle and late gadolinium enhancement demonstrating thickened and fibrotic pericardium (*A and B*).
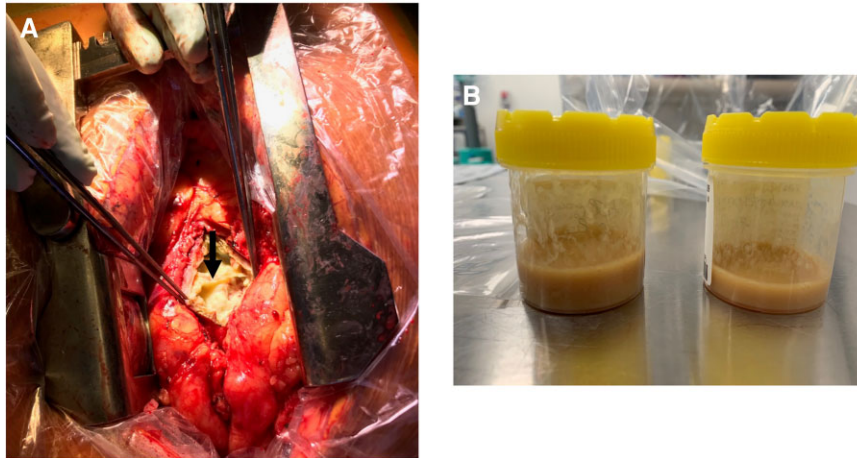
Figure 4 Intraoperative findings. (*A*) Purulent collection enclosed within fibrotic and adhered pericardium and (*B*) surgically evacuated purulent fluid.

The patient was discharged 10 days post-pericardiectomy, with complete symptom resolution. He completed 6 weeks of i.v. benzylpenicillin antibiotic therapy and was reviewed in the Cardiology, Cardiothoracic Surgery, and Infectious Diseases outpatient clinics prior to de-escalation to oral antibiotics. He has since completed 7 of planned 12 months of oral amoxicillin 1 g thrice daily. The patient reports no further episodes of chest discomfort or dyspnoea and is satisfied with his physical recovery. He has, however, experienced low mood as a result of his recurrent hospitalizations and surgery and is currently receiving care from his primary care physician to address this.

Discussion

We present a patient with recurrent pericarditis subsequently found to have *A. meyeri* bacterial pericarditis-induced cardiac tamponade and constrictive pericarditis, who ultimately required pericardiectomy and surgical evacuation for source control.

Purulent bacterial pericarditis is uncommon in the age of antibiotics, especially in immunocompetent adults. It comprises 1–2% of pericarditis[7] but remains a feared entity due to its rapidly progressive nature and 100% mortality rate if untreated.[8] Even with treatment, mortality remains excessively elevated (20–40%),[1,3] contrasting with a 1.1% mortality rate in uncomplicated acute viral or idiopathic pericarditis.[9] Purulent bacterial pericarditis also conveys significant morbidity with a 3.5% risk of developing constrictive pericarditis,[10] compared with <0.5–1.2% risk in uncomplicated acute viral or idiopathic pericarditis.[10,11]

Purulent bacterial pericarditis remains a diagnostic challenge clinically as patients often do not present with archetypal pleuritic chest pain, pericardial friction rub, or diffuse ECG changes.[1,12] Establishing a diagnosis of PBP may be easier if there is pericardial effusion requiring drainage. In addition to fluid assessment for turbidity, the application of Light's criteria to pericardial fluid differentiation has been shown to enhance diagnostic utility.[13] Whilst the diagnostic usefulness of Light's criteria in evaluating pericardial fluid is much less established than in pleural fluid, pericardial fluid/serum LD > 0.6 and cholesterol
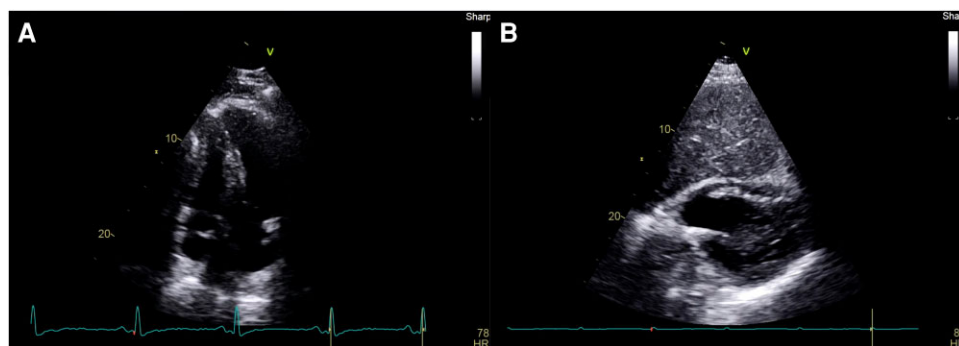
Figure 5 Post-operative transthoracic echocardiography demonstrating minimal pericardial effusion and active right ventricle; (*A*) apical four-chamber view; (*B*) subcostal view.

concentration ≥ 1.2 mmol/L should increase clinical suspicion of PBP, given previous studies demonstrating a diagnostic efficiency of 87 and 83%, respectively, in identifying pericardial exudates.[13]

Purulent bacterial pericarditis without an underlying infection (primary PBP) is exceedingly rare. Secondary PBP is more common, with an underlying infection or cause identified in 78% of cases.[12] Predisposing factors include immunosuppression, dialysis, chemotherapy, malignancy, or chest trauma, increasing susceptibility for infection via haematogenous spread or contiguous spread from intrathoracic or subdiaphragmatic sources.[14] In this case, whilst no underlying bacterial infection was uncovered despite multiple septic screens, a diagnosis of secondary PBP is more likely, given the patient had recent prolonged oral corticosteroid use for treatment of recurrent pericarditis.

Gram-positive pathogens are most implicated; however, incidence of gram-negative, anaerobic, and even fungal infections is increasing.[14] Comprehensive microbiology examination is critical to guide antimicrobial therapy and establish the likely route of infection.

Actinomyces is a commensal organism usually found in the oral cavity, digestive, and genital tracts. Pathogenic actinomycosis can arise when these barriers are compromised, such as in the case of poor dental hygiene, or pneumonia. In this case, *A. meyeri* was isolated in pericardial fluid. The organs that are affected commonly include cervicofacial, abdomino-pelvic, pulmonary, and other soft tissues.[15] *Actinomyces israelii* is the most prevalent pathogenic species; *A. meyeri* infections remain rare, but pericardial involvement has previously been described.[4,5,15] Clinical suspicion for atypical organisms such as Actinomyces is necessary due to the inherent difficulties in establishing microbial diagnosis stemming from Actinomyces' commensal presence in polymicrobial flora, making discrimination between colonization and pathogenicity difficult, as well as its insidious nature requiring up to 20 days of incubation under anaerobic conditions. Actinomyces is susceptible to beta-lactam antibiotics.[15] In this case, the patient was managed with intravenous penicillin G before deescalation to oral amoxicillin following surgical evacuation, with a consensus decision to continue prolonged antibiotic suppression therapy to maximize tissue penetration and ensure adequate treatment.

There are no established PBP management guidelines. The focus of care remains ensuring haemodynamic stability and infectious source control. Pericardiocentesis and antibiotics remain first-line treatment, especially in tamponade, but is unlikely to provide complete or durable resolution particularly in cases of dense adhesions, loculated effusions, recurrent tamponade, or constriction development. Surgical pericardiectomy is often required to prevent cardiac failure sequelae, although optimal timing and duration of pre-surgical antibiotic therapy to reduce sternal contamination risk remains undefined. A multidisciplinary team approach encompassing Cardiology, Cardiothoracic Surgery, Infectious Diseases, and Microbiology input is essential to ensure best outcomes.

Conclusions

Despite its rarity in the modern era of antibiotics, PBP is a highly lethal and morbid condition requiring early recognition and treatment. Percutaneous pericardiocentesis may be utilized initially to achieve haemodynamic stability, identify pathogen, and establish sensitivities. However, surgical pericardiectomy is often required for source control and elimination of constrictive sequelae, especially when there is clinical non-response or rapid deterioration with conservative treatment. A further understanding of surgical timing and antibiotic therapy duration is required to improve outcomes.

Lead author biography

Joanne Eng-Frost is a Cardiology Advanced Trainee at the Flinders Medical Centre in Adelaide, Australia. She completed a Bachelor of Science (Biomedical Science) degree with Honours in Physiology at the University of Adelaide, Australia, before earning a Doctor of Medicine (MD) degree at Flinders University, Australia. Her clinical interests include structural heart disease and interventional cardiology. She is pursuing a Doctor of Philosophy (PhD) to phenotype cardiogenic shock and assess mechanical circulatory supports in cardiogenic shock.

Supplementary material

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

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