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Case Reports

Cardiac Tamponade due to Purulent Pericarditis

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Abstract

Background: Purulent pericarditis, though uncommon in the antibiotic era, remains highly fatal when diagnosis or drainage is delayed. Its presentation often mimics viral hepatitis, sepsis, or parasitic infections—particularly in endemic, low-resource regions—leading to underrecognition. This case reported the development of purulent pericarditis with initial equivocal signs and symptoms, followed by progressive hemodynamic deterioration.

Case Illustration: A 40-year-old previously healthy man presented with fever, dyspnea, stabbing chest and abdominal pain, and dark urine. Examination revealed jaundice, pericardial friction rub, and hepatosplenomegaly. Laboratory tests showed leukocytosis, hyperbilirubinemia, and elevated liver enzymes. Initial echocardiography demonstrated a 2-cm circumferential effusion without signs of tamponade. Two days later, despite stable symptoms, he developed hypotension with new fibrinous effusion and right atrium collapse. Emergency pericardiocentesis drained 1.7 L of thick, purulent fluid. Hemodynamics improved rapidly after drainage. Prednisone and colchicine were initiated once infection control was achieved to limit fibro-inflammatory response and reduce the risk of constriction. Liver function normalized, and follow-up echocardiography showed minimal residual effusion. At follow-up, the patient remained asymptomatic.

Conclusions: This case highlights that purulent pericarditis can occur in immunocompetent individuals without typical risk factors, possibly from overlooked infection in low-resource settings. Hemodynamic collapse may occur even with small increases in pericardial effusion volume, owing to fibrin-induced pericardial stiffness and reduced compliance. Serial echocardiography is therefore critical when symptoms appear stable. Early pericardiocentesis is both diagnostic and therapeutic, reducing bacterial and inflammatory load, while carefully selected adjunctive anti-inflammatory therapy may prevent chronic constrictive sequelae.

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Keywords: Purulent pericarditis, Cardiac tamponade, Constrictive pericarditis.

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Introduction

Purulent pericarditis remains a highly lethal form of pericardial disease if not promptly diagnosed and managed. It is characterized by the accumulation of purulent exudate in the pericardial space, typically resulting from direct extension from adjacent infections, hematogenous spread, or iatrogenic causes such as cardiac surgery or previous pericardiocentesis.[1] Despite being an uncommon entity, the nonspecific and often insidious presentation of purulent pericarditis poses a significant diagnostic challenge.

Common risk factors include immunosuppression, diabetes mellitus, previous thoracic interventions, malignancy, and uremia, and common causative microorganisms include streptococci, staphylococci, Haemophilus spp., and M. tuberculosis.[2-3] In low-resource settings, poor access to healthcare, delayed treatment of primary infections, and high burden of Tuberculosis (TB) and parasitic diseases may contribute to underreported cases.

Before the time of antibiotics, purulent pericarditis had a mortality rate exceeding 90%.[4] Although this number has declined, outcomes remain poor when diagnosis and intervention are delayed. The clinical presentations may mimic viral or tuberculous pericarditis, sepsis, or systemic inflammatory conditions, and echocardiographic findings can initially appear benign. However, the disease may progress rapidly, culminating in cardiac tamponade, constrictive pericarditis, or death.[3] Diagnosis, immediate drainage, and tailored antimicrobial therapy are essential.

Here, we presented a case notable for the development of purulent pericarditis with initial equivocal signs and symptoms, followed by progressive hemodynamic deterioration. It highlights critical management principles: the value of serial echocardiographic assessments, the need for intervention despite seemingly unchanging clinical symptoms, and the role of adjunctive therapies in preventing complications such as constrictive pericarditis.

Case Illustration

A 40-year-old male presented with a 14-day history of fever (38.5°C), chills, dyspnea, stabbing chest and abdominal pain, and dark-colored urine. He had just returned from working as a chef in another border town and denied any recent travel abroad, any animal contact, flood exposure, or known infectious contacts. There was no prior history of significant illness. On physical examination, he was icteric. A pericardial friction rub was heard on auscultation. Hepatosplenomegaly and right upper quadrant tenderness were noted on palpation. Electrocardiogram (ECG) revealed atrial fibrillation with rapid ventricular response (Figure 1).

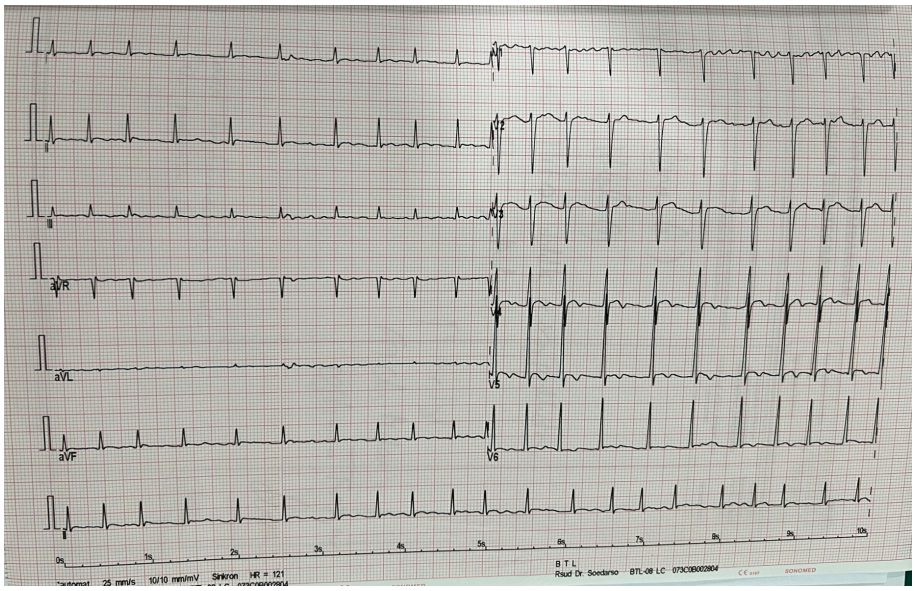

Figure 1. ECG during admission showed atrial fibrillation with rapid ventricular response.

Laboratory findings showed leukocytosis (20,000/mm³) with neutrophilia, hyperbilirubinemia (total 8.7 mg/dL, direct 7.1 mg/dL), and markedly elevated liver enzymes (aspartate transferase [AST] 684 U/L; alanine transferase [ALT] 438 U/L). Renal function and coagulation parameters were within normal limits. Abdominal ultrasonography revealed mild hepatosplenomegaly, minimal ascites, and bilateral pleural effusions. Initial Transthoracic Echocardiography (TTE) demonstrated a 2 cm circumferential pericardial effusion without signs of tamponade (Figure 2). He initially refused pericardiocentesis due to fear. Intravenous (IV) ceftriaxone, furosemide, and hepatoprotector were started.

Two days after admission, the patient became hypotensive (Blood Pressure [BP] 70/50 mmHg), and his rhythm was still atrial fibrillation with a rate ranging from 110 to 130 bpm. His extremities were a little cold to the touch, pulses were still adequate on palpation, and no delay of capillary refill time. He denied worsening chest discomfort and exhibited no overt respiratory distress; he reported feeling slightly better. Repeat echocardiography showed a mildly increased effusion, now fibrinous in appearance, with the right atrium starting to collapse, and exaggerated respiratory variation in mitral and tricuspid inflow (Figure 3). Dobutamine was initiated, and further counseling regarding the urgency and risks of intervention was provided, after which the patient consented.

Emergency pericardiocentesis was performed, draining approximately 500 mL of thick, greenish, purulent fluid initially, followed by production of 1,200 mL of purulent fluid over the next 24 hours (Figure 4). Fluid analysis revealed an extremely high leukocyte count (29,240 cells/mm³), Polymorphonuclear (PMN) predominance (>90%), and a high protein level (5 g/dL). The culture of the pericardial fluid remained sterile, likely due to prior antibiotic exposure in this patient. Tests for TB using GeneXpert (Cepheid, U.S.), malaria, and leptospirosis were negative. The patient showed immediate hemodynamic improvement and resolution of symptoms following pericardial drainage, with BP rising to 100/70 mmHg while

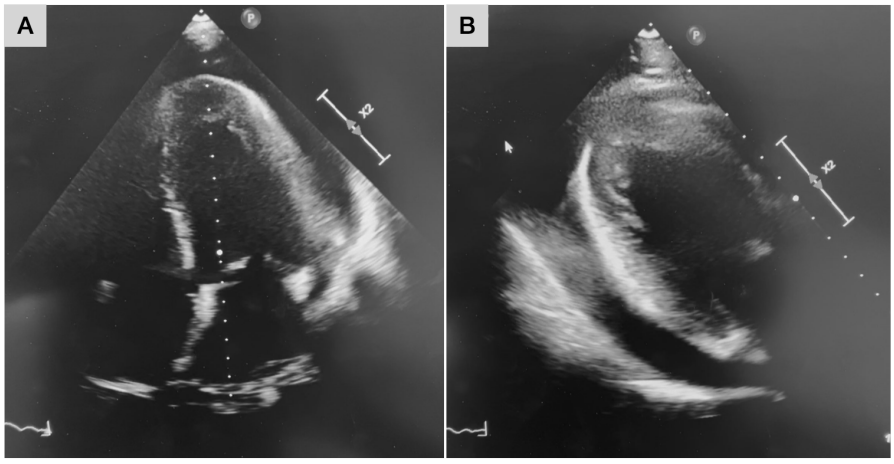

Figure 2. (a) Apical 4-chamber view and (b) parasternal long axis view showing circumferential tamponade.

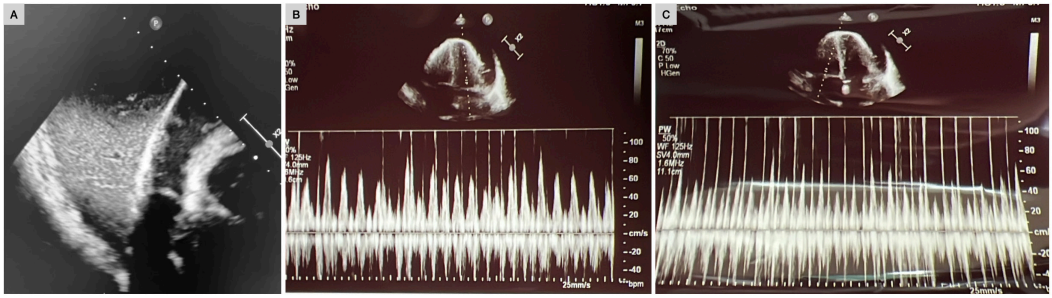

Figure 3. (a) Fibrinous appearance of effusion with exaggerated respiratory inflow variations in (b) mitral and (c) tricuspid.

Figure 4. Pericardial drain production within 24 hours showing greenish purulent fluid.

remaining tachycardic at a heart rate of 100 bpm. Dobutamine was gradually tapered with close hemodynamic monitoring.

By day five, liver enzymes and leukocyte counts had returned to normal, and bilirubin levels had decreased (total 2.8 mg/dL, direct 2.4 mg/dL). Prednisone (1 mg/kg/day), colchicine, and bisoprolol were added to his regimen. By day nine, drain output had decreased significantly, and TTE revealed minimal residual effusion (Figure 5). A respiratory variation >25% in mitral and tricuspid inflow velocities with subtle diastolic interventricular septal shuddering raised early suspicion of constrictive physiology, though medial e' velocity was not greater than lateral e' velocity, and no hepatic vein flow reversal was observed. Patient remained asymptomatic and hemodynamically stable. The pericardial drain was removed, and the patient was discharged on tapering doses of corticosteroids, together with bisoprolol and colchicine. At the 1-month follow-up, he remained asymptomatic, and echocardiography no longer demonstrated diastolic septal bounce, although mitral and tricuspid respiratory variations remained >25%. His rhythm remained atrial fibrillation with a controlled ventricular rate of 80-90 bpm. Bisoprolol was continued for rate control. With a CHADS-VA score of 0, no oral anticoagulant was initiated.

Discussion

This case emphasizes the ambiguous presentation of purulent pericarditis, which masquerades as what was initially thought to be viral hepatitis, sepsis, or parasitic infection like malaria and leptospirosis, which are considered endemic in the region, due to overlapping systemic signs such as jaundice, fever, leukocytosis, and acute liver injury. The absence of classical risk factors does not exclude

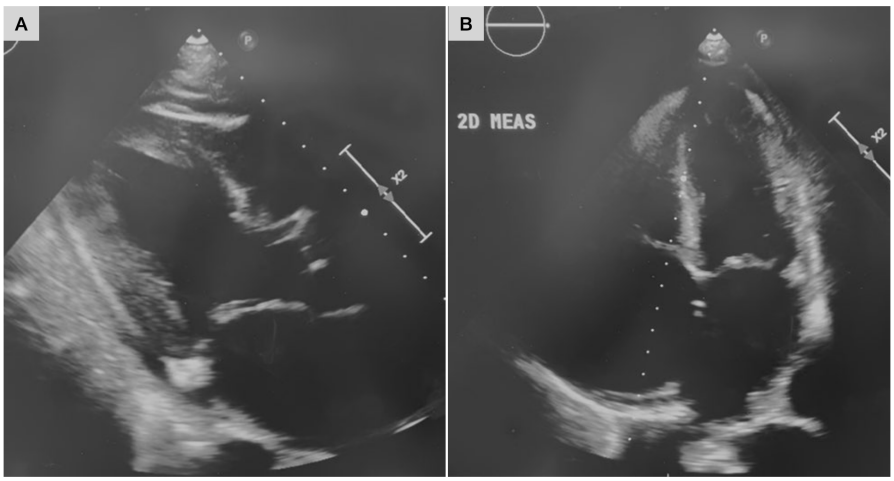

Figure 5. Evaluation of post-drainage TTE showed minimal residual effusion.

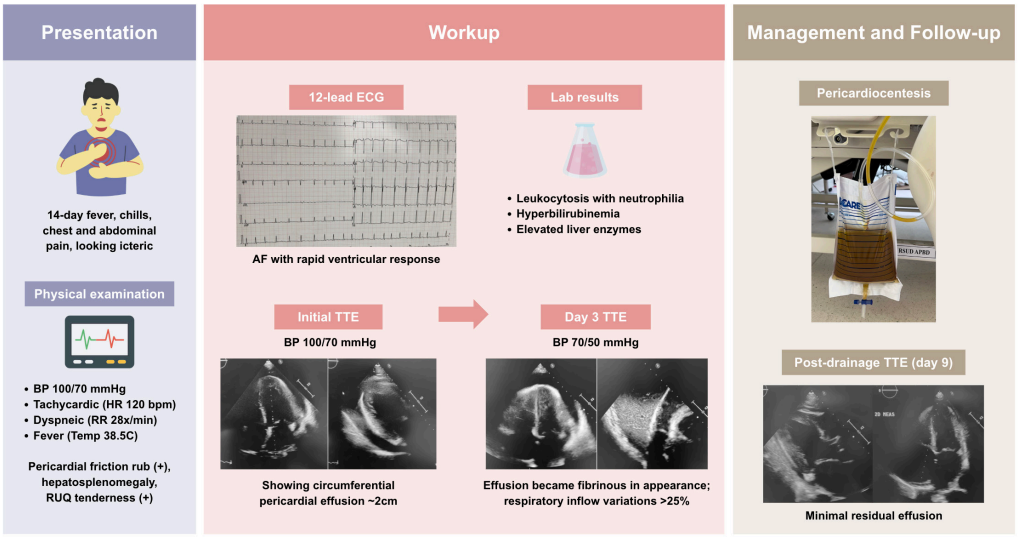

Figure 6. Overall illustration of the case.

the possibility of a preceding infection, given his prior residence in a low-resource border area with limited access to healthcare facilities, which may have increased the likelihood that a minor localized or systemic infection was overlooked or managed symptomatically without professional evaluation.

While TTE remains the cornerstone of diagnosis, its sensitivity for tamponade signs may be limited early in the disease. Thus, serial echocardiographic evaluations are essential, particularly when hemodynamic instability develops in the absence of worsening symptoms. The hemodynamic deterioration despite no symptom progression and a mildly increased effusion illustrates a phenomenon of symptomatically equivocal tamponade, where the inflammation and fibrinous composition might have contributed to the constrictive pericardial physiology, creating a scenario where reduced pericardial compliance amplifies ventricular interdependence that even modest negative intrathoracic pressure during inspiration can precipitate right-sided chamber collapse and a fall in left-sided stroke volume, explaining why decompensation can occur with only slight increases in effusion volume.[5] This shows that imaging can guide decisions when clinical monitoring is insufficient.

Drainage is both diagnostic and therapeutic in purulent pericarditis. Guidelines from ESC and AHA endorse immediate pericardiocentesis for any hemodynamically significant effusion, particularly when a specific or purulent cause is suspected.[6-7] Aside from relieving pressure, drainage is also therapeutic by decreasing bacterial load and removing pro-inflammatory mediators, thereby limiting ongoing pericardial injury, highlighting the importance of early and clear communication about risks and benefits of intervention.

Empirical antibiotic therapy should target common causative agents like Staphylococcus aureus and Streptococcus spp. Third-generation cephalosporins, such as ceftriaxone, are appropriate for community-acquired infections, whereas nosocomial infections or infections in immunocompromised patients may require vancomycin or broader-spectrum agents.[8] The lack of growth in fluid culture is not uncommon and does not exclude bacterial etiology, or suspicion can be raised for other fastidious organisms.[9] Adjunctive corticosteroids remain controversial, especially in infectious pericarditis. However, if inflammatory features are present, such as elevated CRP or pericardial fibrin in our case, they may reduce the risk of constriction by modulating fibroinflammatory responses.[10] Colchicine, proven in idiopathic and recurrent pericarditis, may also benefit post-purulent effusions once infection is controlled.[11] In our case, we favored corticosteroids over Non-Steroidal Anti-Inflammatory Drug (NSAIDs) once infection control was reasonably established (sterile cultures with clinical improvements), to avoid NSAID-related hepatotoxicity in the setting of acute liver injury, to minimize potential renal or platelet effects during recovery, and to achieve a quicker anti-inflammatory effect; colchicine was added as a steroid-sparing adjunct.

One of the dreaded complications of purulent pericarditis is constrictive pericarditis, occurring in 20-30% of cases,[6] which may evolve weeks

to months post-resolution. Signs of constrictive pericarditis include persistent respiratory variation in inflow velocities, abnormal septal motion, medial e' velocity exceeding lateral e' velocity, strain reversus, and diastolic hepatic vein flow reversals.[12] In our case, TTE suggested some constrictive signs, but conservative monitoring was chosen given the absence of clinical signs. Advanced imaging, such as cardiac MRI or CT, may help confirm constrictive physiology in cases of ambiguity. Pericardiectomy remains the definitive treatment for persistent symptomatic constriction.[13] From this case, we learned that:

- Clinical deterioration in purulent pericardial effusion may occur silently and should be suspected. In patients with sepsis and pericardial effusions, serial imaging is vital
- Pericardiocentesis should be promptly considered in suspected purulent pericarditis when there is considerable and accessible fluid accumulation despite the absence of definitive tamponade signs, due to the high risk of rapid decompensation.
- Sterile cultures do not rule out bacterial pericarditis; negative results should not delay or alter treatment in an appropriate clinical context.
- Adjunctive anti-inflammatory agents (e.g., colchicine, steroids, or NSAIDs) play an important role in sequelae prevention when infection is controlled but inflammation persists. Agent selection should consider hepatic and renal profiles, bleeding risk, and the need for rapid control of inflammation.

Conclusion

This case illustrates the deteriorative course purulent pericarditis may take, evolving from sepsis-like symptoms to tamponade within a couple of days. Symptom stability can be misleading, and echocardiographic surveillance plays an essential role in the detection of decompensation. Immediate drainage, empiric antibiotics, and targeted adjunct therapies are key components in management and can significantly improve outcomes. Ongoing monitoring is required to detect complications such as constrictive pericarditis.

List of Abbreviations

AHA	American Heart Association
ALT	Alanine Transferase
AST	Aspartate Transferase
BP	Blood Pressure
BPM	Beats per Minute
CRP	C-Reactive Protein
ECG	Electrocardiogram
ESC	European Society of Cardiology
IV	Intravenous
NSAID	Non-Steroidal Anti-Inflammatory Drug
PMN	Polymorphonuclear
TB	Tuberculosis
TTE	Transthoracic Echocardiography
TV	Tricuspid Valve

Ethical Clearance

Not Applicable.

Publication Approval

All authors consent to the publishing of this manuscript.

Authors Contributions

JAS: conception, case management, data collecting, writing and revision of manuscript; DSP: supervision, case management, revision, approval of final manuscript.

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Conflict of Interest

None.

Availability of Data and Materials

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ChatGPT 5 (OpenAI, GPT-5, 2025) is used

to assist in grammar refinement and phrase adjustment for clarity and improving flow. Content, interpretations, and conclusions were entirely conceived and verified by the authors.

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