ISSN: 3066-7097
Case Report
Journal of Critical Care Research and Emergency Medicine

Septic Pneumopericardium in A Patient with Sepsis Due To Gluteal Cellulitis: A Case Report

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Submitted: 03 September 2025 **Accepted:** 08 September 2025 **Published:** 13 September 2025

https://doi.org/10.63620/MKJCCREM.2025.1050

Citation: *Magazzini, S., Balzarini, B., Betti, M., Cappellini, I., Grassi, I., Longo, M., Matteuzzi, C., & Lai, F. (2025). Septic Pneumopericardium in A Patient with Sepsis Due To Gluteal Cellulitis: A Case Report. J of Cri Res & Eme Med, 4(5), 01-04.*

Abstract

Background: *Pneumopericardium is an uncommon and potentially life-threatening condition defined as the presence of air in the pericardial sac. It is most frequently secondary to trauma, iatrogenic injury, or barotrauma, while infectious etiologies remain rare but highly lethal.*

Case Presentation: *We report a 77-year-old man with type 2 diabetes mellitus, hypertension, and chronic alcohol use disorder who presented to the Emergency Department with fever, dyspnea on exertion, orthopnea, and left gluteal cellulitis. Laboratory investigations revealed leukocytosis, markedly elevated C-reactive protein and procalcitonin, hyperlactatemia, and mixed metabolic–respiratory acidosis. Whole-body CT demonstrated pneumopericardium with bilateral pulmonary infiltrates. The patient was managed according to the Surviving Sepsis Campaign bundle, started on broad-spectrum antimicrobial therapy, and underwent pericardial drainage.*

Conclusion: *Although rare, pneumopericardium should be considered in septic patients with thoracic complaints. Early recognition, rapid imaging, aggressive antimicrobial therapy, and urgent source control are critical for survival.*

Keywords: Pneumopericardium, Sepsis, Gas-Forming Organisms, Cardiac Tamponade, Case Report, Elderly Patient.

Introduction

Pneumopericardium, the accumulation of air within the pericardial sac, is a rare clinical entity most often associated with trauma, thoracic surgery, or barotrauma from mechanical ventilation1,2. Infectious pneumopericardium is less common but carries particularly high mortality, especially in elderly or septic patients with comorbidities3–5. Gas-forming organisms such as Staphylococcus aureus, Streptococcus pneumoniae, Enterobacter ales, Clostridium, and Candida have been implicated6,7. Clinical presentation overlaps with other acute cardiopulmonary syndromes, and progression to tension pneumopericardium with cardiac tamponade portends a grave prognosis8.

Case

An elder 77-year-old male with type 2 diabetes mellitus, hypertension and chronic alcoholism, was seen in the Emergency Room with fever (38,7 °C), progressive dyspnea with orthopnea and asthenia. He had a history of constipation lately and poor food intake. On admission: BP 90/55 mmHg, HR 120/min, RR 30/min, SpO_2 90% in RA. Physical examination revealed muffled heart sounds, coarse breath sounds, distended and diffusely tender abdomen and a warm erythematous target on the left glu-

teal area compatible with cellulitis.

Laboratory findings: WBC 18,500/µL, CRP 27 mg/dL, Procalcitonin 22 ng/mL, Lactate 6.2 mmol/L, ABG: pH 7.18, $PaCO_2$ 50 mmHg, HCO_3^- 18 mmol/L (mixed metabolic–respiratory acidosis), Creatinine 2.4 mg/dL, Urea 92 mg/dL, Na 131 mmol/L, K 5.1 mmol/L, AST 72 U/L, ALT 64 U/L, Bilirubin 2.1 mg/dL, Glucose 298 mg/dL, BNP 910 pg/mL.SOFA score: 10, corresponding to septic shock, and multiorgan failure.

Imaging

Whole-body CT angiography with contrast demonstrated free intrapericardial air encircling the heart (diagnostic of pneumopericardium) with minimal fluid. Bilateral pulmonary consolidations were present, consistent with pneumonia. No evidence of necrotizing fasciitis or gas gangrene was found in gluteal tissues.

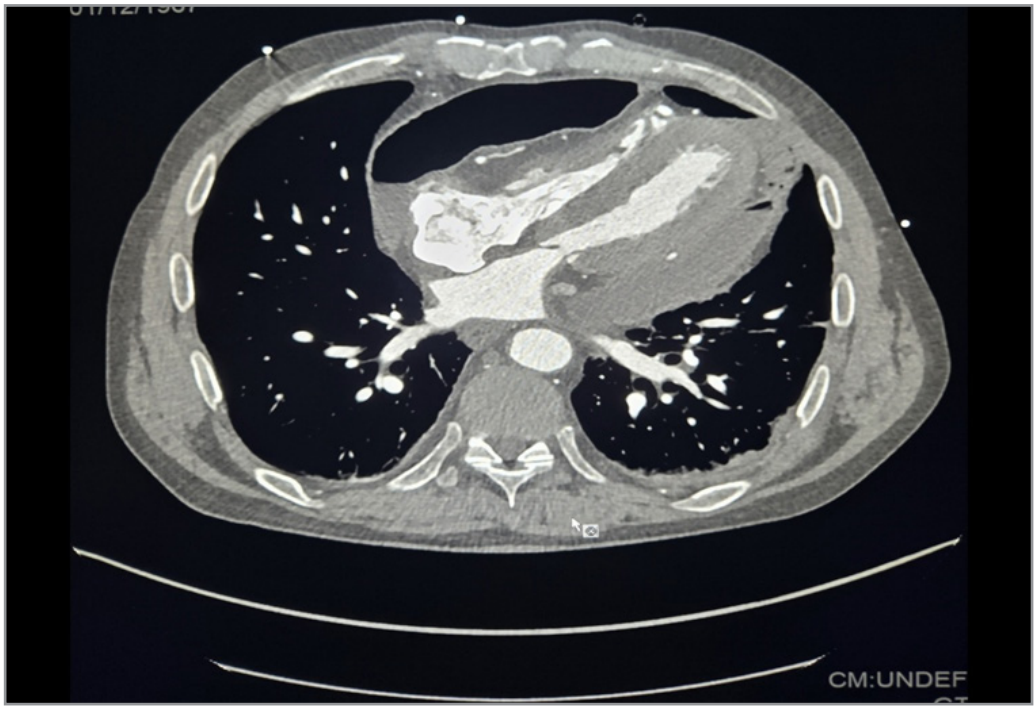

Figure 1: CT scan showing Pneumopericardium

Blood cultures grew an aerobic, gas-forming anaerobe, Clostridium perfringens, after 8 hours. Since microbiology findings and infectious disease service recommendations, the patient's antimicrobial regimen was stepped up from empiric piperacillin/tazobactam 4.5 g IV every 6 hours to meropenem 2 g IV every 8 hours with clindamycin 900 mg IV every 8 hours. Clindamycin was added with the aim of preventing clostridial exotoxin synthesis, which is central to the fulminant course observed in invasive clostridial infections. The patient became severely ill with refractory septic shock and multi-organ failure. Intensive Care Unit management included.

Norepinephrine was gradually titrated up to 0.8 µg/kg/min to maintain the target level of mean arterial pressure of over 65 mmHg. The patient was intubated and started on mechanical ventilation with a lung-protective strategy (tidal volume 6 ml/kg predicted body weight, FiO_2 titrated to maintain $SpO_2 > 92\%$, moderate PEEP titrated for compliance), Hemodiafiltration (CVVHD for severe oliguric AKI and metabolic acidosis. Emergency pericardial drainage drained air and purulent effusion, avoiding the progression to cardiac tamponade. Consecutive surgical debridements of the gluteal necrotizing cellulitis under negative pressure wound therapy until source control is achieved. Despite an early very perturbed trend, there was ultimately amelioration beyond 7 days with reduction in need for vasopressors, normalization of lactate as well as significant improvement in inflammatory serum markers (CRP and procalcitonin). Subsequent echocardiograph also showed the disappearance of the pneumopericardium, and blood cultures taken later were negative.

Sixteen days later, he was weaned off mechanical ventilation and transferred to the surgical ward for further wound care.Although the mortality for septic pneumopericardium has been reported to exceed 70%, this case demonstrates that successful recovery is possible when prompt diagnosis coupled with multidisciplinary care and coordinated critical care are utilized.

Discussion

The occurrence of septic pneumopericardium is an extremely rare clinical entity and infectious etiology represent a minority of the cases, the majority being trauma or iatrogenic. Clostridium perfringens is one of the only pathogens that can lead to the formation of gas in tissue and in the circulation, leading to potentially fatal consequences such as myonecrosis, emphysematous infections, and, in this case, pneumopericardium.

Pathophysiology is via haematogenous spread from a necrotizing soft tissue infection, invasive pericardial infection and gas and exudate collection. This may lead very quickly to tamponade physiology, cardiac arrest and death if not treated.Management is dependent on early diagnosis, prompt empiric antimicrobial coverage, and expeditious source control. In this setting, the pairing of carbapenems (for broad coverage against anaerobes and Gram-negative organisms) with clindamycin (to suppress toxin production) is highly advised. Pericardial drainage can be life-saving to decompress the mediastinum, while aggressive

ICU support using vasopressors, acute dialysis, and lung-protection ventilation may allow the patient to survive the acute phase.

Pneumopericardium results rare as discussed above and the majority of cases are attributed to blunt or penetrating chest trauma (~35%), iatrogenic causes such as intubation or thoracic procedures (~25%), infectious etiologies (~15%), barotrauma (~10%), malignancy-related fistulization (~8%), and spontaneous or idiopathic cases (~7%). Infectious pneumopericardium has the highest mortality, particularly in elderly septic patients [1-5].

Table 1: Epidemiology of Pneumopericardium (based on case series 2005–2022)

Etiology	Percentage (%)
Trauma (blunt or penetrating)	35
Iatrogenic (procedures, ventilation)	25
Infectious (pyogenic, gas-forming)	15
Barotrauma (mechanical ventilation, asthma)	10
Neoplastic fistulization	8
Spontaneous/idiopathic	7

Pathophysiology

Air may reach the pericardial sac via direct communication with the airways, esophagus, or by infection with gas-forming organisms. More than half of reported infectious cases are due to S. aureus and S. pneumoniae. Entero bacterales and anaerobes (Clostridium, Bacteroides) are less frequent but associated with fulminant septic presentations. A "ball-valve" mechanism can cause tension pneumopericardium with hemodynamic compromise [6-10].

Table 2: Pathogens Associated with Infectious Pneumopericardium

Pathogen	Frequency (%)
Staphylococcus aureus	30
Streptococcus pneumoniae	25
Escherichia coli	15
Klebsiella pneumoniae	10
Anaerobes (Clostridium, Bacteroides)	10
Candida spp.	10

Clinical Features

Typical symptoms include dyspnea, orthopnea, chest pain, and fever. The pathognomonic "mill-wheel murmur" may be auscultated. Clinical signs of tamponade—tachycardia, hypotension, muffled heart sounds—signal imminent cardiovascular collapse [11].

Diagnosis

- Chest radiography/CT: reveals intrapericardial air circumferentially surrounding the heart, distinguishing it from pneumomediastinum.
- Echocardiography: may be limited by air artifacts but is critical to evaluate tamponade physiology.
- Laboratory testing: demonstrates systemic sepsis with elevated inflammatory markers and metabolic acidosis.

Treatment

Supportive care with oxygen, fluid administration, norepinephrine for shock. Empirical antibiotics: carbapenem + vancomycin for resistant Gram-positives; antifungal prophylaxis in high-risk patients. Hemodynamic instability or tension pneumopericardium calls for pericardial drainage. The management of superinfections is crucial and aggressive.

- Supportive care: oxygen therapy, fluid resuscitation, norepinephrine for septic shock.
- Empiric antimicrobials: carbapenem plus vancomycin to cover resistant Gram-positives; antifungal therapy (echinocandin) in selected high-risk patients4,5.
- Pericardial drainage: indicated in hemodynamic instability or tension pneumopericardium; lifesaving in tamponade.
- Source control: aggressive management of pulmonary infection and soft tissue cellulitis.

Clinical Scenario	Empiric Regimen	Notes
Community-Acquired, Low Risk	Ceftriaxone 2 g/day + metronidazole 500 mg q8h OR piperacillin–tazobactam 4.5 g IV q6–8h	De-escalate on cultures
High risk / ESBL	Meropenem 1 g q8h (extended infusion) ± amikacin loading dose	Consider colonization, prior antibiotics
MRSA risk	Add vancomycin or linezolid	Nosocomial infections or colonization
Candida Risk	Add echinocandin (e.g., anidulafungin)	Septic shock, abdominal source, positive cultures
Duration Post-Source Control	4 days	STOP-IT trial; shorten if rapid recovery

Prognosis

Mortality in infectious pneumopericardium may reach 50%3–6. Prognosis is determined by early recognition, sepsis bundle compliance, and timely drainage.

Conclusion

Pneumopericardium is a rare but life-threatening complication of infection and sepsis in frail patients. Physicians should maintain a high index of suspicion in elderly septic patients with chest complaints. Rapid imaging, appropriate empiric antimicrobial therapy, and timely pericardial decompression are essential for survival.

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