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Case Report

Diagnostic and therapeutic challenges in managing purulent pericardial effusion with concurrent pneumonia: A geriatric case report

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ABSTRACT

Introduction: Purulent pericarditis is defined as an infection in the pericardial space that produces macroscopically or microscopically purulent fluid. It was a rare but life-threatening condition. It may be primary or secondary to another infectious process. This condition, characterised by an infectious or inflammatory accumulation of fluid in the pericardial cavity, presents significant diagnostic and therapeutic challenges, particularly in the context of multiple comorbidities. The purpose of this case report is to provide descriptive information about rare clinical patient scenario of purulent massive pericardial effusion in elderly.
Case Description: The patient's presentation, complicated by pneumonia and diabetes mellitus, underscores the complexities in diagnosing and managing an 85-year-old male patient with diverse medical backgrounds. Echocardiography confirmed the diagnosis of massive pericardial effusion and showed the purulent fluid from the pericardiocentesis procedure. Nevertheless, despite various efforts to find the origin of the infection and treat it with antibiotics according to the sensitivity test, the patient's outcome with many risk factors, immunocompromised condition, unclear source of infection, aside from septic shock that led to the patient's death during treatment.
Conclusion: Clinicians need to be aware of immunocompromised elderly patients and act quickly to help them. They also need to deal with the diagnostic difficulties of identifying definitive infectious sources, the high risk of death even with modern treatments, and the important role that underlying comorbidities play in prognosis. Clinical evidence shows that purulent pericarditis is still a serious condition that can have adverse outcomes, especially in older patients who already have a lot of health problems.

1. Introduction

Purulent pericarditis represents a focal infectious process within the pericardial cavity, characterised by either macroscopic purulent exudate or microscopic evidence of suppuration (leukocyte count exceeding 20 per high-power field).[1] In modern practice, purulent pericarditis is relatively uncommon, occurring in less than 1% of all pericardial cases, coupled with its high mortality rate of 100% if left untreated, it makes a particularly challenging diagnosis that requires prompt recognition and intervention. In two extensive series of cases of purulent pericarditis during the antibiotic era, direct pulmonary extension accounted for 25% of infections, while haematogenous spread occurred in 22%, perforating injury or surgery in 24%, and myocardial abscess and/or endocarditis in 22%.

Management of purulent pericarditis was the pericardium must be drained, and systemic antibiotic therapy must be started on a whim and then changed based on the results of a microbiological study.[2] In this case, we represent an 85-year-old male who has purulent pericarditis, pneumonia, and type II diabetes. The patient's advanced age of 85 years, long-standing history of uncontrolled diabetes mellitus type II, and chronic smoking history created an immunocompromised state that made him particularly susceptible to severe infections. The patient's history of uncontrolled hypertension may also be influenced by genetic factors, such as polymorphisms in the angiotensinogen gene (AGT) M235T and AGT T174M, which have been linked to essential hypertension.[3] Despite implementing standard therapeutic measures, including urgent pericardiocentesis and broad-spectrum antibiotic therapy, the patient's condition deteriorated into septic shock, ultimately leading to death on the tenth day of hospitalization. The case serves as a valuable learning opportunity for healthcare providers, emphasizing the importance of early diagnostic and treatment of this rare but deadly condition in immunocompromised patients.

2. Case Report

An 85-year-old male patient presented with a complaint of worsening shortness of breath over the course of three days. The shortness of breath occurred throughout the day. It accompanied a productive cough and fever for two weeks. The patient had a history of uncontrolled hypertension and diabetes mellitus (DM) type II. The patient denied experiencing night sweats, weight loss, or a cough with blood in the sputum. He had no prior history of open surgery. He was a smoker for more than 50 years, as much as 1 pack a day. He arrived with a regular heart rate of 108 bpm, a blood pressure of 90/60 mmHg without support, a respiratory rate of 30 bpm, a SpO2 of 96% on a nasal cannula at 3 lpm, a JVP of R+2 cmH2O, coarse rhonchi, and no muffled heart sound or wheezing in both lungs. Electrocardiography showed no low voltage and electrical alternans (Figure 1A); a marker of infection rose in the lab; chest x-rays showed cardiomegaly and pneumonia (Figure 1B); and echocardiography showed massive pericardial effusion without tamponade sign (Figure 1C).

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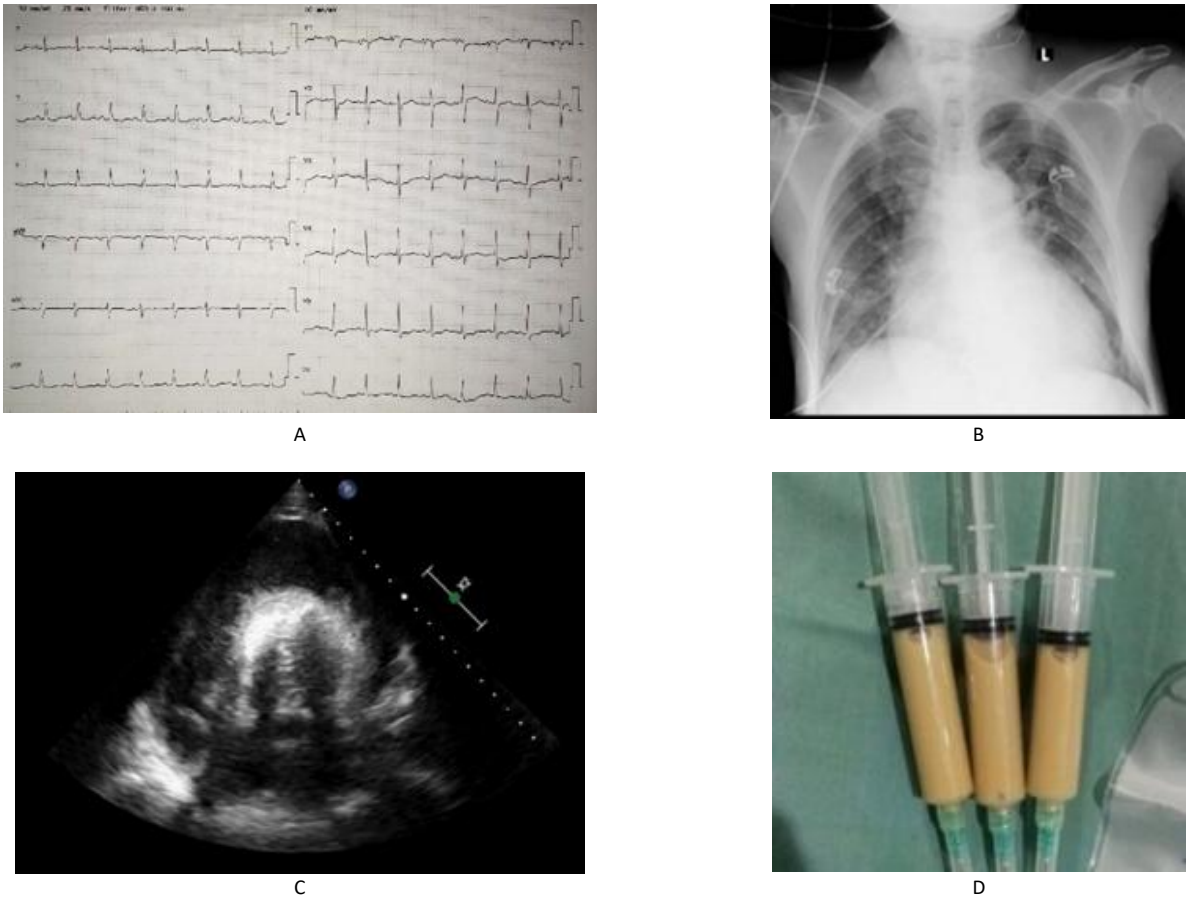

Figure 1. Supportive findings. (A) Electrocardiography showed no low voltage and electrical alternans; (B) Chest x-rays showed cardiomegaly and pneumonia; (C) Echocardiography showed massive pericardial effusion without tamponade sign; (D) Purulent Pericardial fluid sample.

Table 1. Differential Diagnosis of The Specific Forms of Pericarditis

	Viral	Bacterial	Tuberculous	Autoreactive
Cardiotropic microbial agents	Entero-, echo-, adeno-, cytomegalo, Ebstein Barr, herpes simplex, influenza, parvo B19, hepatitis A,B,C virus, HIV	Staphylococci, pneumococci, streptococci, Neisseria, proteus, gram negative rods, Legionella	Mycobacterium tuberculosis	Autoimmune process in the absence of viral and bacterial agents
Etiological evidence by	PCR or in situ hybridisation (evidence level B, indication IIa)	Gram-stain, bacterial culture, PCR for Borrelia and chlamydia pneumoniae (evidence level B, indication I)	Ziehl-Neelsen, auramin 0 stain, culture, PCR (evidence level B, indication I)	Ig-binding to peri- and epicardium, negative PCR for cardiotropic agents, epicarditis (evidence level B, indication IIa)
Incidence (%) Western countries	30	5–10 5 per 100,000 patients	<4 (much more in Africa and South America)	20–30
Male: female ratio	3:1	1:1	1:1	1:1
Predisposition	Unknown	Chronic alcohol abuse, immuno-suppression,	Alcohol abuse, HIV infection	Association to autoimmune disorders
Clinical features	Identical to acute pericarditis, often subfebrile	Spiking fever, fulminant, tachycardia, pericardial rubs	Subfebrile, chronic	Subfebrile, chronic
Effusion size	Variable, mostly small	Variable	Variable, mostly large	Variable
Tamponade	Infrequent	80%	Frequent	Infrequent
Spontan. Remission	Frequent	None	None	Rare
Recurrence rate	30–50%	Rare	Frequent	Frequent; >25%
Aspect of PE	Serous/serosanginous	Purulent	Serosanginous	Serous
Protein content	>3 g/dL	High	High/intermediate	Intermediate
Leukocyte count (PE)	>5000/ml	≫10000/ml	Intermediate >8000	Intermediate <5000
Pericardial fluid analyses	Activated lymphocytes and macrophages (sparse) Adenosindeaminase (ADA)-negative	Granulocytes and macrophages (massive) ADA-negative	Granulocytes and macrophages (intermediate) ADA positive (>40 U/ml)	Activated lymphocytes and macrophages (sparse) ADA-negative
Peri- and epicardial biopsy	Lymphocytic peri-/epicarditis, PCR positive for cardiotropic virus	Leukocytic epicarditis	Caseous granuloma, PCR	Lymphocytic peri-/epicarditis, PCR negative
Mortality if untreated	Depending on agent and tamponade	100%	85%	In untreated tamponade
Intrapericardial treatment	Drainage, if needed, no intrapercardial corticoids	Drainage and rinsing (saline) gentamycin 80 mg i.p.,	Drainage, if needed	Drainage, i.p. triamcinolon (evidence B, indication IIa)
Pericardiotomy/ pericardiectomy	Rarely needed	Promptly needed (evidence level B, indication I)	Rarely needed	Rarely needed
Systemic treatment	I.V. immunoglobulins, IFN (in enteroviral pericarditis) s.c.	I.V. antibiotics	Tuberculostatic + prednisone	NSAIDs, Colchicine, prednisolone/ azathioprin
Constriction	Rare	Frequent	Frequent (30–50%)	Rare

He was assessed with massive pericardial effusion without sign of pericardial tamponade and pneumonia. He planned to perform an urgent pericardiocentesis using an apical approach, which resulted in the production of purulent fluid (Figure 1D), which was suspected to be caused by bacterial infection. The laboratory, anatomical pathology, and microbiology departments evaluated the pericardial fluid. Pericardial fluid cytology had shown negative for the presence of atypical or malignant cells, gram-negative rods, *Escherichia coli,* was revealed from the microbiological culture of the pericardial fluid which sensitive to Levofloxacin and Ceftriaxone.

He began receiving care from the pulmonary departments and received an intravenous injection of antibiotics, anti-inflammatory drugs, and analgesics. We took the epicardial fluid every 6 hours, resulting in a total of 510 mL of purulent fluid on the fourth day of care. The patient continued to receive three types of intravenous antibiotics: moxifloxacin, metronidazole, and ceftriaxone. The patient's response to the medication was poor, leading to the worsening condition of the septic shock. He passed away on the 10th day of hospitalisation. This highlights the challenges in managing septic shock, a condition for which the optimal use of corticosteroids remains a subject of ongoing investigation.[4]

3. Discussion

Purulent pericarditis represents a focal infectious process within the pericardial space, characterized diagnostically by either a macroscopic purulent effusion or microscopic evidence of significant leukocytosis exceeding 20 white blood cells per high-power field.[1,5] This entity demonstrates remarkably low prevalence (<1% of pericardial cases) yet carries significant mortality, with uniformly fatal outcomes in untreated cases.[6,7] Contemporary data indicate a mortality rate approximating 40%, predominantly attributed to three major complications: cardiac tamponade, septic shock, and constrictive physiology.[2,7] The manifestation of the disease in the clinic varies greatly, leading to a delayed diagnosis due to the absence of distinct pericarditis signs and the frequent confusion of symptoms with other infections.[2,5,8] The classic clinical syndrome usually shows up as an acute illness with fever, stiffness, shortness of breath, tachycardia, tussis, chest pain, and the possibility of progressing to cardiac tamponade.[1,9] A definitive diagnosis requires a pericardiocentesis, which can be used for both diagnosis and treatment. The fluid should then be analysed thoroughly, with a macroscopy examination, biochemical profiling showing exudative characteristics, and microbiological studies of both pericardial and systemic specimens (backed by level B evidence, class I indication).[5,9] The patient was an 85-year-old male with a clinical presentation of shortness of breath and fever. The examination shows a massive pericardial effusion with purulent fluid accompanied by pneumonia. Purulent pericarditis usually occurs in immunocompromised patients.[8] The patient is susceptible to purulent pericarditis due to his uncontrolled DM, a pneumonia condition, and his advanced age. The source of infection was suspected from the extension from pneumonia, haematogenous spread, or an extension from a subdiaphragmatic suppurative focus.[2,6] In Western series, the predominant-associated lesions of purulent pericarditis were empyema (50%) or pneumonia (30%).[10] The most common organisms were staphylococci, streptococci, and pneumococci (Table 1). The pericardial fluid culture of this patient shows Escherichia coli. The sputum culture examination explored the suspicion of pneumonia extension, revealing Klebsiella pneumonia. Klebsiella pneumonia and Escherichia coli were both gram-negative organisms, which counted for about one-third of isolated organisms found in purulent pericarditis cases. The suspicion from haematogenous spread was explored by blood culture, but the result was clear. A history of abdominal surgery, cancer, or a subdiaphragmatic abscess (bacterial liver abscess or amoebic liver abscess) was used to look into the possibility of extension from a subdiaphragmatic suppurative focus.[9,11] This patient had no history of abdominal surgery or related manifestations before the onset of disease. He also didn't have a history of malignancy. An imaging examination was scheduled but hasn't been done due to unstable haemodynamics, and the patient passed away right after. The etiopathogenesis of purulent pericarditis in this case might originate from bacterial translocation via subdiaphragmatic route, supported by the isolation of Escherichia coli from pericardial fluid cultures—a microorganism characteristically associated with intra-abdominal and subdiaphragmatic infectious processes, suggesting potential contiguous spread or haematogenous dissemination from an infra-diaphragmatic nidus of infection. The *Escherichia coli* bacterial still might be possibly came as contaminant from the culture laboratory examination procedure. The sample was sent to the culture laboratory when its off, and should wait for the next day to get the sample examined. However, based on the risk factors of purulent pericarditis, the condition primarily stemmed from pulmonary infection, which subsequently extended to the pericardium.[12] The identified risk factors include advanced age, immunocompromised state due to uncontrolled DM and hypertension, and a history of chronic smoking, which are the main vehicles of infection.[8,13] Genetic variations, such as ACE polymorphisms influencing vascular function and potentially increasing susceptibility to cough, may also play a role, particularly in individuals treated with ACE inhibitors.[14] It is mandatory to rinse the pericardial cavity and administer effective systemic antibiotic therapy.[15] The pericardial effusion drainage strategy could be pericardiocentesis, pericardiotomy, or pericardiectomy, based on the patient and technical resources of the institution to achieve complete drainage of the pericardial space.[2,16] Patients who have dense adhesions, loculated and thick purulent effusion, recurrence of tamponade, persistent infection, and progression to constriction need a pericardiectomy as soon as possible.[17]

4. Conclusion

This case presents a rare and true presentation of purulent pericarditis in an 85-year-old male patient with multiple comorbid conditions. The patient experienced substantial difficulties in managing his illness, a common occurrence with this serious and rare condition. The clinical picture progressed to septic shock, culminating in death, despite aggressive treatment measures including pericardiocentesis, antibiotic administration, and other forms of management. Some important points are: early recognition, examination, and treatment of the disease in elderly patients with immunodeficiency; the difficulty in determining and localising the pathogen responsible for the condition; the great possibility of mortality even with standard measures; and the effect of comorbidities. This highlights that purulent pericarditis, despite the rapid advancements in medicine and related technologies, remains a severe disorder with a poor prognosis in most cases, particularly among the elderly population. As with rare oncologic complications in immunocompromised patients, vigilance for atypical presentations is crucial in these complex scenarios.[18]

5. Declaration

5.1 Ethics Approval and Consent to participate
Patient has provided written informed consent prior to involvement in the study.

5.2. Consent for publication
Not applicable.

5.3 Availibility of data and materials
Data used in our study were presented in the main text.

5.4 Competing interests
Not applicable.

5.5 Funding Source
Not applicable.

5.6 Authors contributions
Idea/concept: IV. Design: IV. Control/supervision: SA, CT. Data collection/processing: IV. Analysis/interpretation: IV. Literature review: IV, SA, CT. Writing the article: IV. Critical review: SA, CT. All authors have critically reviewed and approved the final draft and are possible for the content and similarity index of the manuscript.

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