

Purulent Pericarditis and Streptococcal Toxic Shock Syndrome: A Rare Presentation of *Streptococcus pyogenes* Infection

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ABSTRACT

Purulent pericarditis (PP) is a potentially fatal localized infection of the pericardial space accompanied by suppuration. Antimicrobial therapy and pericardial drainage are the two main treatments for PP. One potential linked terrible consequence of PP is constrictive pericarditis (CP), a type of diastolic cardiac failure caused by an inelastic thickened pericardium. Gram-positive, beta-hemolytic, catalase-negative, and coagulase-negative streptococcus pyogenes cocci can be seen in pairs or chains. *Streptococcus pyogenes* is a significant bacterial pathogen that is unique to humans and can appear in a variety of ways. Invasive *Streptococcus pyogenes*-induced pericarditis has seldom ever been documented. Echocardiography revealed a significant pericardial effusion with tamponade physiology in a 61-year-old man who had been experiencing growing left-sided chest discomfort and an erythematous rash. The diagnosis of purulent bacterial pericarditis with streptococcal toxic shock syndrome was established after an emergency pericardial window produced purulent fluid and cultures verified *Streptococcus pyogenes*. Despite the fact that the bacteria were susceptible to antibiotic therapy, the patient died after receiving the proper antibiotics and intravenous glycemic injection. Rarely observed, *S. pyogenes*-induced PP primarily affects newborns and children and progresses quickly in immunocompromised people. The prognosis could be improved with an earlier diagnosis, appropriate drainage surgery, and efficient antibiotic treatment.

Keywords: purulent pericarditis, streptococcal, toxic shock syndrome, streptococcus pyogenes infection

INTRODUCTION

Inflammation of the pericardium, or pericarditis, can have both infectious and non-infectious causes¹. Purulent pericarditis is uncommon and linked to substantial morbidity and mortality, whereas idiopathic and viral pericarditis are the most prevalent types observed today and are typically benign^{1,2}.

Based on incidences recorded worldwide, the combination of purulent pericarditis and toxic shock syndrome (TSS) is very uncommon³. Despite its potentially fatal consequences, myocardial infections are also regarded as rare⁴. Patients with weakened immune systems are more susceptible than the general population because *S. pyogenes* causes the body to produce more super-antigens and trigger an immunological response⁵. The death rate from heart infections is high, particularly in patients who already have cardiomyopathies. Furthermore, in patients with impaired immune systems, toxic shock in any form is extremely deadly⁶. In order to address the resulting health concerns, treatment duration and care precision are essential⁷. Given the rarity of toxic shock syndrome and cardiac infections, prompt therapeutic action and vigilant diagnostics are warranted.

Here, we are presenting a case of 61-year-old man who had purulent pericarditis and streptococcal toxic shock syndrome as rare presentation of streptococcus pyogenes infection.

CASE PRESENTATION

A 61-year-old man with a significant past medical history of type 2 diabetes mellitus, hypertension, hyperlipidemia, coronary artery disease, and rheumatoid arthritis—managed with ibuprofen, methotrexate, and etanercept—presented to the emergency department with left-sided chest pain, dry cough, and sore throat. He denied

experiencing fever, chills, myalgias, or dyspnea, and his symptoms had persisted for approximately one day prior to admission.

At initial evaluation, his vital signs were as follows: blood pressure 152/79 mmHg, heart rate 54 beats/min, temperature 37.2 °C, respiratory rate 18 breaths/min, and oxygen saturation 99% on room air. The physical examination was unremarkable. Laboratory investigations revealed a leukocyte count of $13 \times 10^9/L$, hemoglobin of 10 g/dL, and thrombocytopenia with a platelet count of $61 \times 10^9/L$; renal function was within normal limits with a creatinine of 0.9 mg/dL. A respiratory viral panel, including tests for COVID-19, influenza, and RSV, returned negative. An electrocardiogram demonstrated sinus bradycardia (Figure 1), while chest radiography showed no evidence of infiltrates or pneumothorax (Figure 2). A computed tomography pulmonary angiogram further excluded pulmonary embolism (Figure 3).

The patient was discharged with instructions to continue ibuprofen and to follow up with his primary care provider. However, over the ensuing 7 days, his chest pain progressively worsened despite ongoing therapy, and he subsequently developed an erythematous rash involving his trunk, hands, and ankles. At re-presentation, he remained afebrile; his vital signs with a heart rate of 100 beats/min, blood pressure of 111/56 mmHg, respiratory rate of 20 breaths/min, and oxygen saturation decreased to 95% on room air. However, later became tachycardic and hypotensive, required vasopressor and stress dose steroids, and transferred to ICU.

Repeat laboratory studies revealed a white blood cell count of $13.4 \times 10^9/L$, hemoglobin of 13 g/dL, and thrombocytopenia with platelets at $76 \times 10^9/L$. His renal indices were notably deranged, with a blood urea nitrogen of 70 mg/dL and creatinine of 3.1 mg/dL, while serum lactate was elevated at 2.8 mmol/L. Cardiac biomarkers remained

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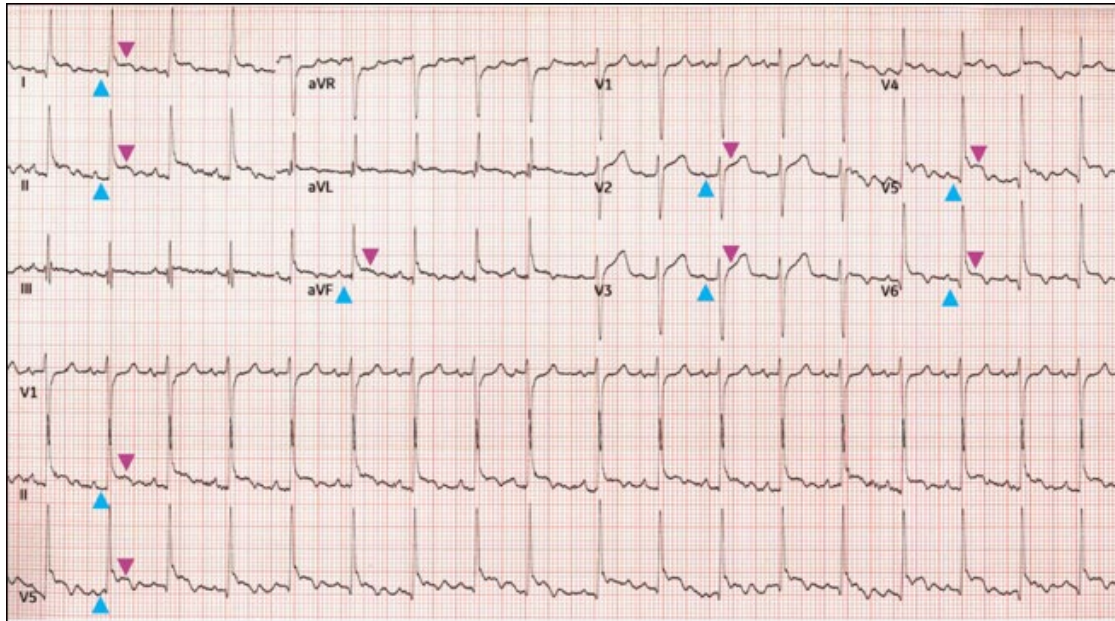


Figure 1. Electrocardiogram demonstrating sinus bradycardia



Figure 2. Radiography showing no evidence of infiltrates or pneumothorax

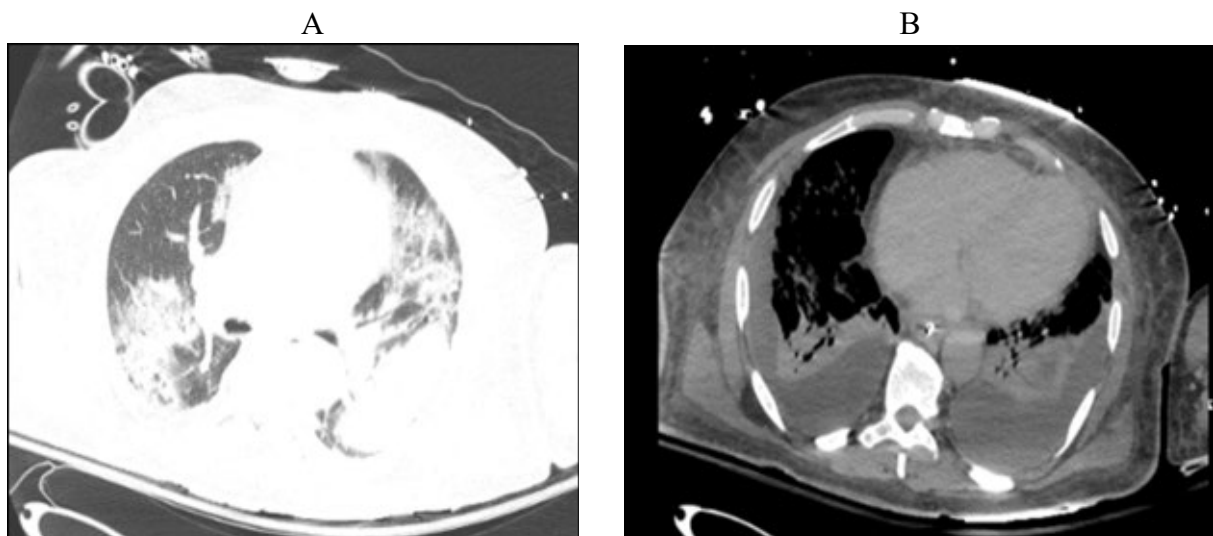


Figure 3. Computed tomography showing pulmonary angiogram excluding pulmonary embolism

unremarkable with troponin levels <0.012 ng/mL, yet brain natriuretic peptide was significantly elevated at 5600 pg/mL. Transthoracic echocardiography demonstrated a moderate pericardial effusion identified circumferential to the heart. There is echocardiographic evidence of elevated pericardial pressures concerning for tamponade physiology.

In response to these findings, an emergent pericardial window was performed, yielding purulent fluid. Both pericardial fluid cultures and concurrent blood cultures subsequently grew *Streptococcus pyogenes*, establishing the diagnosis of purulent bacterial pericarditis with streptococcal toxic shock syndrome (TSS).

DISCUSSION

The patient was presented with atypical symptomatology since he had chest pain but no fever or dyspnea. TSS is usually characterized by fever, hypotension, and a rash⁷. One of the main reasons the physicians took longer to diagnose the heart infection was the unique symptoms. Also, the diagnostic latency reported in the case can be attributed to the normal CXR and negative viral panel.

The failure draws attention to the dangers of detecting some types of bacterial pericarditis. The patient's condition deteriorated after discharge, and new symptoms appeared, such as erythematous rashes on the hands, ankles, and limbs and renal failure. The patient had a mild effusion and was afebrile upon re-presentation. He had a high pericardial pressure and was hypotensive. These results were consistent with the tamponade echocardiographic criteria. Consequently, blood and fluid cultures ought to be regarded as the foundational method for identifying *S. pyogenes* infections.

The example studies how *S. pyogenes* affects the immune system. Because of his pre-existing arthritis, the patient's immune was compromised, which allowed pathogens to enter. The *S. pyogenes* bacteria most likely originated from occult pharyngitis or a skin cut⁸. T-cell hyperactivation and a cytokine storm were brought on by the bacteria's release of exotoxin and M-protein into the patient's bloodstream⁹. Their progressive failure prior to ICU admission indicated that they produced a polyorgan dysfunction syndrome⁹. The patients' ongoing hypotension is also explained by it. Purulent effusion and the resulting diastolic filling can be used to explain tamponade¹⁰. Systemic vasodilation and capillary leak are responsible for the development of TSS and the ensuing organ failure.

The patient's therapeutic therapy was successful in spite of the case's challenges. When testing for tamponade, diagnostic cultures should be collected within the acute pericardial intervention window, per IDSA 2024 guidelines¹¹. This procedure allows for pericardial fluid examination, restores cardiac hemodynamics, and relieves pleuropericardial congestion. The doctors can also determine the etiologic agent thanks to the diagnostic cultures. Notably, *S. pyogenes* bacterial growth was inhibited by the use of beta-lactam antibiotics and clindamycin¹².

Immune homeostasis was restored by adjunctive intravenous immunoglobulin (IVIG), which stopped bacterial-mediated tissue damage to the heart tissue¹³. Norepinephrine vasopressors were administered to the patient as supportive therapy, resulting in blood pressure stabilization. Therefore, treating dual pathology requires a multimodal treatment plan¹².

There are significant knowledge gaps in the present literature on *S. pyogenes* pericarditis cases. Cardiac infections are very uncommon,

according to researchers¹⁴. Further research into this complication is warranted due to the rarity of *S. pyogenes* pericarditis with TSS¹⁵. Toxic shock is rare in the majority of people with pericarditis. However, because it indicates heart compression from pleural effusion, cardiac tamponade is rather prevalent. Researchers also stress how useful echocardiography is for evaluating heart issues¹⁶.

Patients with pericarditis and TSS have comparatively poor survival chances, particularly if they do not receive immediate surgical treatment. Researchers point out that the situation is somewhat exceptional because TSS-pericarditis cohorts show high mortality¹⁷. Timing is crucial whether treating toxic shock, heart infections, or both, as demonstrated by the successful course of treatment and recovery. Immunosuppression has been connected to some drugs, which could lead to fatal infections. Etanercept and other RA medications raise the risk of infection from bacteria like *S. pyogenes*¹⁸. Overall, considering the fatality of the situation, immediate action is required to promote the connection between TSS and pericarditis.

CONCLUSION

In summary, a 61-year-old male patient was presented with TSS and pericarditis. Chest pain, pharyngitis, coughing without result, erythematous eruptions, hypotension, tachycardia, and hypoxemia were among the main symptoms. Multimodal therapy helped to resolve the issue. This case illustrates an uncommon circumstance in which a heart infection and toxic shock combine to cause multiple organ failure. The necessity of early ultrasonography and prompt surgical procedures are among the key clinical outcomes. Since this is the 20th recorded instance of *S. pyogenes* pericarditis with TSS, ID physicians need to learn more about the bacterial etiology of patients on immunosuppressive drugs.

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