Indolent Purulent Pericarditis Due to *Viridans Streptococcus* Infection Successfully Treated by Pericardiocentesis and Penicillin G

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Although purulent pericarditis generally presents with acute cardiovascular decompensation and a sepsis-like appearance, it can be indolent in rare settings, leading to a catastrophic outcome. We report a 77-year-old female who presented with progressive dyspnea without fever. Massive pericardial effusion with cardiac tamponade was detected by the transthoracic echocardiogram. The fluid culture yielded *Viridans Streptococcus* which was susceptible to penicillin G. The patient dramatically improved after 14 days’ penicillin G treatment and did well during the 10 months’ follow-up. Purulent pericardial effusion should be kept in mind as a lethal cause of cardiac tamponade, even in afebrile patients. Emergent pericardiocentesis followed by appropriate antibiotics treatment is essential to avoid delayed diagnosis and improper management.

**Key Words:** Cardiac tamponade • *Viridans Streptococcus* • Purulent pericarditis • Gram stain

**INTRODUCTION**

Purulent pericarditis, characterized by frank pus or microscopically purulent effusion in the pericardial sac, is usually a severely acute illness with still high mortality, especially if both diagnosis and treatment are delayed. It is usually caused by *Staphylococcus aureus*, *Streptococcus pneumoniae*, or *Streptococcus pyogenes*. Predisposing factors include pneumonia, previous cardiac or thoracic surgery, immunosuppression, and pre-existing aseptic pericarditis and infective organisms. Clinical findings include fever, chills, dyspnea, tachypnea, cough, weakness, and tachycardia out of proportion to fever. Classic symptoms of pericarditis, including chest pain and pericardial friction rub, occur in only about 50% of patients.

The clinical course of purulent pericarditis is usually fulminant, manifesting with shock syndrome due to cardiovascular collapse and/or septic phenomena. However, it can also be insidious. Here, we describe an afebrile woman presenting with indolent purulent pericarditis due to *Viridans Streptococcus* infection, successfully treated by both prompt pericardiocentesis and adequate penicillin G treatment.

**CASE REPORT**

A 77-year-old female presented with a one-week history of progressive dyspnea. She was relatively well in the past except for a 10-year history of hypertension under anti-hypertensive medications (losartan and nifedipine). She didn’t take illicit drugs. Before hospitalization, no significant inflammatory symptoms such as fever and chills could be traced. Vital signs included a
body temperature of 36.1 °C, heart rate of 118 beats/minute, respiration of 20 per minute and blood pressure of 160/89 mmHg in supine position, and the oxygen saturation was 92% when she was breathing ambient air. Jugular venous pressure was about 12 cm of water. Lung auscultation showed crackles over bilateral basal lung fields. The patient’s heart sounds were distant but regular, with no significant murmur. Dentition was poor, but there was no abscesses nor gingival tenderness found by a dentist. The remainder of the physical examination was unremarkable.

Pertinent laboratory data showed a white cell count of $7.6 \times 10^3$/mm$^3$ with 60% segmented cells, 22% bands, and 10% lymphocytes, hemoglobin of 12.4 g/dL, and a platelet count of $250 \times 10^3$/mm$^3$. The C-reactive protein level was 12.2 mg/L. Arterial blood gas exam revealed pH of 7.430, oxygen partial pressure of 68 mmHg, and carbon dioxide partial pressure of 34 mmHg. Twelve-lead electrocardiogram revealed sinus tachycardia with rate of 118 beats/min, diffuse low voltage, and left axis deviation (Figure 1). A chest radiograph showed cardiomegaly but no pulmonary infiltrates. Transthoracic echocardiography revealed massive pericardial effusion (2.0 cm) with early right ventricle diastolic collapse but without evidence of oscillating vegetation or valve regurgitation, consistent with cardiac tamponade (Figure 2). Emergent pericardiocentesis was performed, releasing 675 ml of bloody fluid from the pericardial cavity. Laboratory analysis of the pericardial fluid showed 280,000 nucleated cells/mm$^3$ with 70% segmented cells, 10% bands, 15% lymphocytes, and 5% monocytes, 600,000 red blood cells/mm$^3$, lactate dehydrogenase of

![Figure 1. A 12-lead ECG showed diffuse low voltage.](image1)

![Figure 2. Transthoracic echocardiography (A) Diastolic right ventricle free wall collapse (arrow) and large pericardial effusion in M-mode. (B) Apical four-chamber view showed large pericardial effusion (*).](image2)
3200 U/L, triglycerides of 20 mg/dL, glucose of 10 mg/dL, protein of 5.0 g/dL, and negative activity of adenosine deaminase. The acid-fast stain was negative. A rapid Gram stain of pericardial fluid revealed Gram-positive cocci in chain. Antibiotic treatment with intravenous 3 million U of penicillin G every 4 hours was initiated.

Further laboratory studies including thyroid function test, a polymerase chain reaction for tuberculous bacilli in pericardial effusion, autoimmune disease test, and tumor markers were all within normal limits. Three days later, culture from the pericardial fluid yielded *Viridans streptococcus* which was susceptible to penicillin G. We also followed transthoracic echocardiography every day, which revealed only minimal non-lobulated pericardial effusion. All culture reports, including blood culture, sputum culture, and urine culture, showed no bacteria growth. The patient recovered well after 14 days' intravenous penicillin G treatment. On hospital day 14, follow-up transthoracic echocardiography revealed minimal non-lobulated pericardial effusion. Oral amoxicillin treatment was continued for another 4 weeks. The patient did well during the next 10 months’ follow-up.

**DISCUSSION**

Cardiac tamponade is a medical emergency, which should be diagnosed carefully and treated thoroughly. Common etiologies of cardiac tamponade include inflammation, infection, immunologic disorder, neoplasm, myxedema, renal insufficiency, pregnancy, aortic or cardiac rupture, trauma, nephrotic syndrome, hepatic cirrhosis, and chronic heart failure. Despite the indolent disease course in our patient, the positive culture result from the pericardial effusion and dramatic improvement after both antibiotics treatment and adequate drainage confirmed the diagnosis of purulent pericarditis with cardiac tamponade.

Purulent pericarditis is typically an acute and often catastrophic illness. Both early detection and effective management of purulent pericarditis require much effort and skill to achieve correct diagnosis. Therefore, physicians should be very alert to the type of disease setting. Fast diagnosis is often by the aid of echocardiography, computed tomography (CT), and/or physical findings such as pulsus paradoxus or diminished heart sounds, and made when pus is drained from the pericardial space or when bacteria is cultured from the pericardial fluid.

But purulent pericarditis always coexists with another disorder. Viridans streptococcal infection is rarely found in purulent pericarditis. Also, it is usually combined with other organisms. The clinical course of viridans streptococcal infection is usually sub-acute or chronic, with low or absence of toxic sign. The common sources of viridans streptococcal purulent pericarditis include mediastinitis from esophageal rupture, dental caries, and retropharyngeal abscesses, infective endocarditis, chest surgery, trauma, and pneumonia. The possible source in our case might be related to dental caries according to the history, chest radiograph, transthoracic echocardiographic findings, and intact skin.

Pericardiocentesis and drainage of the pus, as well as antibiotic treatment, are mandatory for patients with purulent pericarditis. In patients treated only with antibiotics without pericardial drainage, the rapid unsuspected development of a large pericardial effusion may result in sudden cardiovascular collapse and death due to cardiac tamponade. However, if the clinical course is indolent, bedside Gram stain and acid-fast stain can provide information for clinical decision-making, including antimicrobial agent selection, pericardial surgery, and prognostic prediction. If the causative organism is *Haemophilus influenza*, early pericardiectomy is recommended, since the exudates in the pericardial space are described as having the density of "scrambled eggs" and can hardly be drained with a catheter. Also, the risk and difficulty of closed pericardiocentesis are higher in lobulated effusion. The open approach should be mandatory for safety and adequate management. In our case, no significant lobulated pericardial effusion and no significant strand bands were noted. Then drainage of pericardial fluid could be adequately performed by closed pericardiocentesis to relieve the cardiac tamponade at once and minimize the suffering of the patient.

In conclusion, purulent pericarditis is a potential lethal disease and should be considered as an indolent underlying disease of cardiac tamponade. A stat Gram stain as well as other pericardial effusion analysis should be performed in every patient with cardiac tamponade. Prompt percutaneous catheter drainage of pericardial fluid along with appropriate antibiotic therapy according to bedside Gram stain can rapidly terminate the life-
threatening condition associated with purulent pericarditis and cardiac tamponade.

REFERENCES