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Rare Purulent Cardiac Tamponade Caused by *Streptococcus Constellatus* in a Young Immunocompetent Patient: Case Report and Review of the Literature

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Male, 19
Final Diagnosis: Cardiac tamponade
Symptoms: Chest pain • shortness of breath
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual clinical course

Background: Purulent pericardial tamponade is a very rare occurrence in the current era of widespread antibiotic use. It is even rarer when caused by *Streptococcus constellatus*: a microorganism usually classified among the normal flora of the human body. It is occasionally diagnosed with certain predisposing factors.

Case Report: We present the third case of *Streptococcus constellatus* cardiac tamponade reported in the current medical literature, occurring in a previously healthy young man who was initially admitted and treated for possible community-acquired pneumonia. The patient required immediate subxyphoid pericardiocentesis. He was also treated successfully with a lengthy course of both intravenous and oral antibiotics. Two months post-hospitalization, he was confirmed clinically stable with complete resolution of his purulent effusion. We also conducted a review of the literature for all *Streptococcus milleri* group purulent pericardial infections between 1984 and 2015.

Conclusions: Purulent cardiac tamponade caused by *Streptococcus constellatus* is extremely rare. It can be life threatening, however. Early appropriate diagnosis and therapeutic intervention are critical for a good outcome.

MeSH Keywords: Cardiac Tamponade • Pericardiocentesis • Pericarditis • *Streptococcus Constellatus*

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/900904

Background

The genus *Streptococcus* consists of gram-positive cocci typically arranged in chains. Many of its groups can cause severe infections in both humans and animals [1].

Streptococci, in general, are classified into 3 groups depending on the degree of hemolysis observed on blood agar: β-hemolytic (complete lysis of red cells), α-hemolytic (partial hemolysis with green coloration), and γ-hemolytic (no hemolysis). Among the vast variety of streptococcus groups, the *Streptococcus milleri* group (SMG) can uniquely cause all 3 types of hemolysis [2]. Furthermore, SMG, a subgroup of *Streptococcus viridans*, is considered to be part of the normal flora of the oral cavity, nasopharynx, vagina, and gastrointestinal tract [3]. *Streptococcus constellatus* belongs to this group and occasionally can be behind a multitude of human pathologies [4]. Clinically, SMG has a tendency for abscess formation, especially in patients with chest infections (including pneumonia, pulmonary abscesses, and mediastinitis) [5,6].

Although rare, SMG, including *Streptococcus constellatus*, has been reported to cause purulent pericarditis, mainly among immunocompromised individuals.

Case Report

A 19-year-old male with no known medical illnesses presented to the emergency department with progressive shortness of breath, pleuritic chest pain, and productive cough of 10 days' duration. The patient stated that there was no fever, no unintentional weight loss, no night sweats, and no exposure to sick contacts. Furthermore, he denied history of dental caries, any recent travel, sexual activity, or a prior similar episode.

On admission, the patient was in mild respiratory distress: tachypneic with a respiratory rate of 35/mn and oxygen saturation of 87% on room air. His temperature was 38.5°C, and his blood pressure (BP) was 85/60 mm Hg. On physical examination, there was decreased air entry, dull percussion, and increased tactile fremitus on the lower right zone of the chest. There was no jugular vein distension and no murmurs or rub. Exam of the abdomen and extremities was unremarkable. Complete blood count showed white blood count (WBC) of 17.6×10^3/μL with left shift. His basal metabolic panel was within normal limits. Lactate dehydrogenase (LDH), ALT, and AST were 2500 IU/L, 1893 IU/L, and 3650 IU/L, respectively. Three different sets of cardiac markers were negative. C-reactive protein (CRP) was 232 mg/L, while lactic acid and procalcitonin levels were within normal range.

Initial electrocardiogram (ECG) showed sinus tachycardia with 1 mm ST segment elevation in lateral leads. Chest X-ray (CXR) showed right-lower-zone nonhomogenous opacity with mild pleural effusions bilaterally (Figure 1).

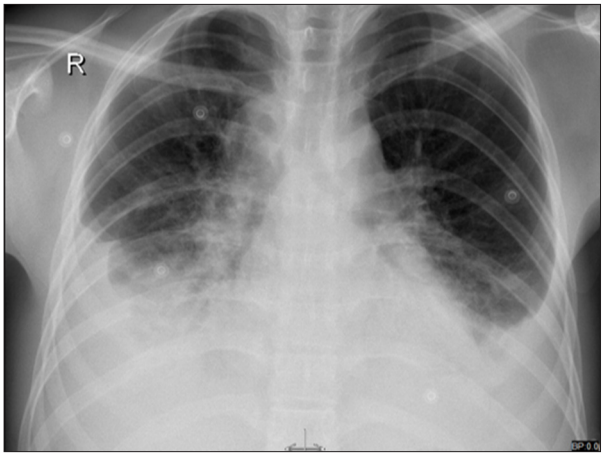

Figure 1. Chest X-ray showing bilateral pleural effusions with nonhomogenous opacity in the right lower zone.

The patient was initially admitted as a case of community-acquired pneumonia. He was started on intravenous ceftriaxone and azithromycin, fluid replacement, and paracetamol. His BP improved to 110/78 mm Hg. Thoracocentesis was performed, and turbid yellowish fluid was drawn. Laboratory analysis showed WBC of 2565/μL with neutrophilic shift. Pleural fluid cultures were negative. Blood and sputum cultures (two sets each) showed no growth. Sputum acid-fast bacilli (AFB; two sets) were negative as well.

The next day and despite treatment, the patient began to worsen clinically with persistent chest pain and reoccurrence of hypotension (BP: 88/57 mm Hg). Repeat ECG showed pronounced diffuse ST segment elevation (Figure 2). A high-resolution computed tomography scan confirmed the presence of pleural and pericardial effusions (Figure 3). He further developed jugular venous distension of 10 cm H_2O and muffled heart sounds. Pulsus paradoxus was also observed, with systolic BP dropping 13 mm Hg on inspiration.

Cardiac tamponade was strongly suspected, and the patient was immediately transferred to the cardiac intensive care unit where an urgent transthoracic echocardiogram (TTE) was done. It confirmed the presence of a large pericardial effusion with right ventricular diastolic collapse (Figure 4) and a dilated inferior vena cava measuring up to 2.5 cm without collapse during inspiration. Up to 700 mL of purulent fluid was removed via pericardiocentesis (subxyphoid approach) and sent for cytopathological examination. Pericardial fluid WBC was 19,375/μL with neutrophilic shift.

Subsequent pericardial fluid cultures grew *Streptococcus constellatus,* which was sensitive to penicillin and ceftriaxone. His

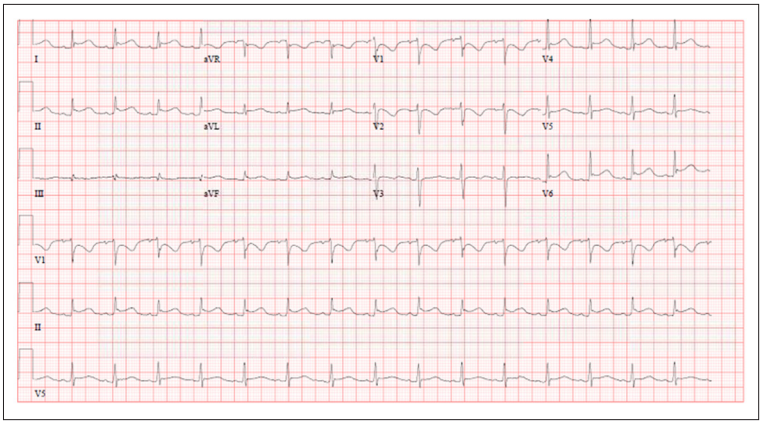

Figure 2. ECG showing sinus rhythm with PR segment depression (lead II) and diffuse ST segment elevation.

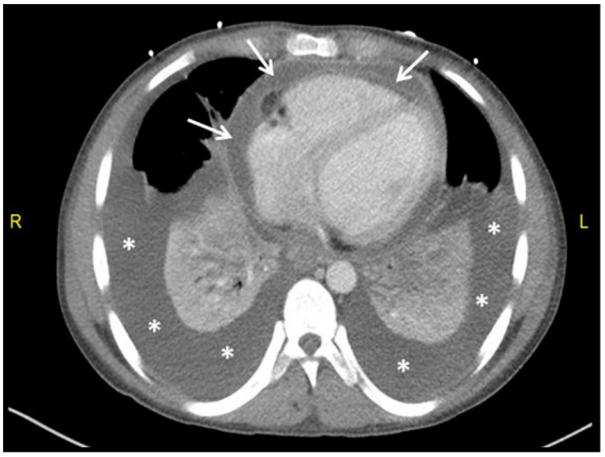

Figure 3. High-resolution computerized tomography (HRCT) showing bilateral pleural effusions (asterisks) and pericardial effusion (arrows).

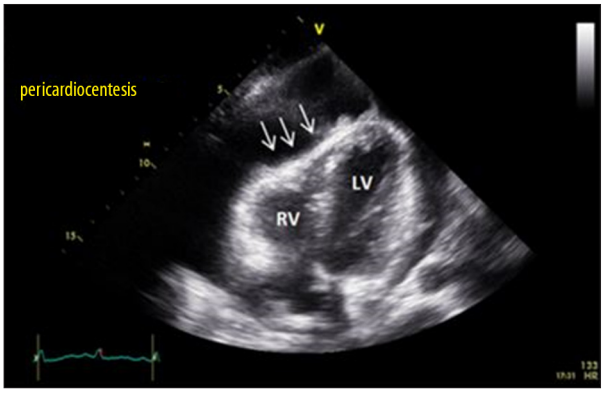

Figure 4. Four-chamber apical view of transthoracic echocardiogram showing early diastolic collapse (arrows) of the right ventricular (RV) free wall surrounded by a large pericardial effusion.

antibiotic regimen was changed accordingly. Two pericardial fluid AFB cultures and human immunodeficiency virus (HIV) testing were negative. The patient had major clinical improvement after nine days of IV ceftriaxone. Repeat CXR showed resolving minimal pleural effusions. He was discharged home with a two-week oral amoxicillin-clavulanate course.

Two months post-discharge the patient was clinically stable. A repeat TTE showed complete resolution of the pericardial effusion.

Discussion

Purulent pericarditis is defined as a confined infection within the pericardial space. It has become generally uncommon since the introduction of antibiotic therapy decades ago [7,8].

Many predisposing factors can contribute to the development of pericardial infection. Historically, chest infections such as pneumonia were the main risk factor for such severe infection. More recently, however, we are witnessing a growing list of conditions identified as potential predisposing factors. These include chest trauma, chest surgery, preexisting pericardial disease, uremia, collagen vascular disease, alcohol abuse, malignancy, and immunosuppression [9].

Clinical symptomatology for purulent pericarditis can vary from subtle findings to the more serious triad of hypotension, dilated jugular veins, and distant heart sounds. This triad, described by Claude Beck in 1935, is suggestive for cardiac tamponade and can be present in up to 40% of cases [10].

Gram-positive organisms (mostly *Staphylococcus aureus*) dominate the spectrum of bacterial pericarditis etiology. Gram-negative bacteria and anaerobes have been documented as causative agents [11–13]. The bizarre occurrence of *Neisseria meningitidis* cardiac tamponade without meningitis was reported [14]. Culture-negative purulent pericardial tamponade has also been described [15].

Searching the medical literature, we identified a total of seventeen (17) pericardial infection cases caused by *Streptococcus*

milleri group (SMG) from 1984 to 2015. Various presentations were reported such as straightforward purulent effusion, pleuro-pericarditis, pericardial abscess, esophago-mediastinal fistula complication, and even postpartum purulent cardiac tamponade [16–29]. *Streptococcus constellatus* purulent pericardial tamponade was clearly identified in only two cases: the first by Reder and colleagues in 1984 [30] and the second by Tokuyasu et al. in 2009 [31]. With no prior record of immunosuppression or a chronic condition, our patient had an insidious presentation with subsequent rapid deterioration. His initial symptoms pointed mostly to a possible acquired pneumonia evidenced by clinical and radiologic findings. Within two days of presentation and despite appropriate antibiotic coverage, he developed a full-blown picture of life-threatening cardiac tamponade. Early recognition of both Beck's clinical triad and typical ECG changes were key elements for timely diagnosis and immediate intervention. Even though diagnosing cardiac tamponade is a clinical achievement, echocardiography when available, as in our setting, is extremely valuable. Transthoracic echocardiography is not only easy and effective in diagnosing cardiac tamponade, but it also can assist in prompt pericardial fluid evacuation, hence improving the chances of recovery and survival [32].

Our case highlights the importance of a good clinical examination (in this instance, a cardiovascular exam). Early clues can quickly guide clinicians to order appropriate testing such as echocardiography. Furthermore, caregivers should not shy away from emergently transferring patient with worrisome findings to an intensive care setting where aggressive therapeutic intervention(s) and close monitoring can be achieved. If not treated in time, purulent pericarditis can have a mortality rate approaching 100% [24]. Prompt therapy is based on two main actions: appropriate antibiotics and immediate pericardiocentesis. These modalities are usually sufficient to bring about recovery unless there is presence of loculated thick pericardial effusion or dense adhesions. In such instances, some authors have opted for pericardial window with irrigation, pericardiotomy, and even intra-pericardial urokinase infusion with varying degree of success [33,34].

Conclusions

Historically more prevalent, purulent pericarditis seems to be reported more often lately due to a possibly increasing list of risk factors. The *Streptococcus milleri* group, including *Streptococcus constellatus*, can occasionally cause such infection. While cases of life-threatening purulent cardiac tamponade remain rare, health care professionals should always have a high index of suspicion for diagnosing it. They should not hesitate to order an immediate echocardiogram to confirm such a diagnosis and assist in the drainage of the pericardial effusion. In addition, broad-based antibiotic coverage must be initiated as soon as possible while awaiting the result of fluid cultures.

Conflict of interests

The author has no conflict of interests regarding publishing this paper.

Statement

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