Received: 2016.12.08
Accepted: 2017.02.03
Published: 2017.04.06

ISSN 1941-5923
© Am J Case Rep, 2017; 18: 355-360
DOI: 10.12659/AJCR.902751

Purulent Pericarditis: An Uncommon Presentation of a Common Organism

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Conflict of interest: None declared

Patient: Female, 65
Final Diagnosis: Purulent pericarditis secondary to *Streptococcus agalactiae*
Symptoms: Altered mental state • fever • general weakness
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Unusual clinical course

Background: In the modern antibiotic era, *Streptococcus agalactiae* infection of the endocardium and pericardial space is a rare occurrence. However, once the disease spreads it can lead to life-threatening illness despite advances in diagnostic and treatment modalities, partly because the symptoms and signs associated with pericarditis are frequently missing, and due to the rarity of the disease, diagnosis is often overlooked. We report an extremely rare case of purulent pericarditis caused by *Streptococcus agalactiae*.

Case Report: A 65-year-old diabetic woman presented with generalized weakness, high-grade fever, and altered mental status. There were no signs or symptoms suggestive of cardiac tamponade on presentation. A computerized tomography (CT) scan of the chest showed a small pericardial effusion. She was managed for diabetic ketoacidosis and sepsis. An electrocardiogram was significant for new-onset atrial fibrillation. Her clinical status deteriorated rapidly as she developed acute hypoxic respiratory failure and shock. A bedside echocardiogram showed large pericardial effusion around the right ventricle and right ventricular diastolic collapse. She developed cardiac arrest, and during resuscitation bedside pericardiocentesis was done with drainage of 15 cc of serosanguineous fluid. However, the patient could not be revived. Subsequently, blood cultures grew *Streptococcus agalactiae* a day after she died. On autopsy, she was found to have findings of infective endocarditis and purulent pericarditis.

Conclusions: A high index of clinical suspicion is crucial when acute pericarditis is suspected, for early diagnosis and for timely initiation of appropriate therapy with antibiotics and aggressive pericardial drainage to prevent fatal outcome.

MeSH Keywords: Endocarditis • Pericarditis • *Streptococcus agalactiae*

Abbreviations: GBS – group B beta-hemolytic *Streptococcus*; IE – infective endocarditis; IDU – injection drug use; *S. agalactiae* – *Streptococcus agalactiae*; HIV – human immunodeficiency virus

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/902751>

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Background

Streptococcus agalactiae, a group B beta-hemolytic *Streptococcus* (GBS), is known to cause bacteremia, chorioamnionitis, septic abortions, urosepsis, and cellulitis in pregnant women, as well as neonatal sepsis and meningitis [1]. The incidence of GBS infection has recently increased among non-pregnant adults. GBS infective endocarditis (IE) is exceedingly rare; however, it is a virulent infection that can cause serious complications. Spread of the infection to the pericardium from the infected endocardium is uncommon and classical signs of pericarditis like chest pain, pulsus paradoxus, pericardial friction rub, and electrocardiographic changes may be absent. The diagnosis should be suspected in cases with refractory shock not responding to initial medical management. This case report describes an extremely unusual manifestation of a relatively uncommon organism leading to a fulminant course.

Case Report

A 65-year-old Hispanic woman was brought to the emergency department with chief complaints of generalized weakness, diarrhea, subjective fever, and altered mental status. Six days prior to presentation, she had been admitted to another facility for generalized weakness and was discharged the next day after management of hyperglycemia. After the discharge, her mentation deteriorated and she was brought back to the emergency department. She remained confused in the emergency room and was unable to provide any meaningful history. As per the family, her past medical history was significant for diabetes mellitus, chronic kidney disease stage III, hyperlipidemia, stroke with residual left-sided weakness, and provoked deep venous thrombosis of the right lower extremity after a right knee surgery. She had eye surgery for retinal detachment and cerebral aneurysmal clipping at the age of 16. She lived with her family, consumed alcohol socially, was a lifelong non-smoker, and never used any illicit drugs. She had not traveled anywhere recently and had no sick contacts. Her home medications included metformin, gabapentin, insulin, sitagliptin, amlodipine, lisinopril, metoprolol, and rosuvastatin.

On physical examination in the emergency room, she was an elderly confused woman who was febrile (T max 102°F [38.9°C]) with a pulse rate of 92 per minute, respiratory rate 18 per minute, blood pressure 112/69 mmHg, oxygen saturation 97% on ambient air, and body mass index of 28 kilograms per m². She had a dry oral mucosa with decreased skin turgor and no jugular venous distension. Skin examination was unremarkable for any rash, discoloration, or marks of intravenous drug use, her nails were normal, and capillary filling was normal in the nail beds. On chest auscultation, normal vesicular breath sounds were heard bilaterally and heart sounds were normal

with no rubs, murmurs, or gallops. No hepatomegaly was appreciable on abdominal examination. She was confused, with a Glasgow comma scale of 13/15.

Initial laboratory findings were significant for leukocytosis with neutrophilic predominance, anion gap metabolic acidosis with blood glucose levels of 717 mg/dl, and acute-on-chronic kidney failure. Liver function tests on admission were normal and urine drug screen was negative. An electrocardiogram was significant for new-onset atrial fibrillation. A chest radiograph showed no infiltrates or effusions. A CT scan of the head showed large extra-axial fluid collection representing an arachnoid cyst and a low-density lesion in the left parietal lobe. CT scans of the chest, abdomen, and pelvis showed bilateral pleural effusions and small pericardial effusions. A few hours later, the patient spiked fever and developed hypotension, and her clinical status deteriorated with worsening of her mental status. She was initially treated for diabetic ketoacidosis and subsequently admitted to the intensive care unit for management of diabetic ketoacidosis and sepsis. On arrival to the intensive care unit, she became hypoxic. She was intubated for acute hypoxic respiratory failure and placed on mechanical ventilator support. She was treated for presumptive meningitis, and health care-associated pneumonia for findings on chest CT. Her hypotension remained refractory to fluids and required vasopressor support. She required escalating doses of vasopressors and developed worsening acidosis and multiorgan failure. A limited bedside echocardiogram showed large fibrinous pericardial effusion around the right ventricle, and right ventricular diastolic collapse with no vegetations (Figures 1, 2). Before any attempt was made to drain the pericardial effusion, she became pulseless approximately 14 h after her arrival to the intensive care unit. Advance cardiac life support protocol was initiated and emergent bedside pericardiocentesis was performed, which drained 15 cc of serosanguinous fluid. We were unable to revive her despite all the resuscitative efforts. Her blood cultures on admission were reported positive for beta-hemolytic Group B *Streptococcus* a day after she died. An autopsy was performed, which showed fibrinous pericarditis and infective endocarditis (Figures 3–5)

Discussion

Infective endocarditis is an uncommon clinical entity that, if unrecognized, leads to serious morbidity and mortality. IE incidence has increased in the United States over the past decade. Incidence of IE hospitalizations from 2000 to 2011 are 11–15 cases per 100 000 population [2]. Males are more commonly affected than females [3]. The majority of cases occur in those with predisposing identifiable, cardiac structural abnormality (congenital or acquired), or with the recognized risk factors of the disease, such as injection drug use (IDU), indwelling

Figure 1. Transthoracic echocardiogram parasternal long axis view showing pericardial effusion.

Figure 2. Transthoracic echocardiogram apical 4-chamber view showing pericardial effusion.

Figure 3. (A) On autopsy, the heart showed cardiomegaly (500 mg) and pericarditis with fibrinous exudate and adhesions; (B) Infective endocarditis with vegetations on the posterior leaflet of the mitral valve.

catheters, poor dental hygiene, previous history of IE, or infection with human immunodeficiency virus (HIV). The mainstay of diagnosis of IE is blood culture and echocardiography.

Various microorganisms can be involved in the pathogenesis of infective endocarditis. Gram-positive organisms are predominant (83%), with *Staphylococcus aureus* being the most common organism [4]. Other organisms involved are streptococci, Coagulase-negative *Staphylococcus*, HACEK

(*Haemophilus aphrophilus*, *Aggregatibacter*, *actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species), Non-HACEK gram-negative bacteria, fungi/yeast, and polymicroorganisms. Streptococci are gram-positive bacteria and are classified based on their hemolytic properties. Alpha-hemolytic (viridans) streptococci, *Streptococcus bovis*, and enterococci are common causes of infective endocarditis [5].

Figure 4. Fibrinous pericarditis with fibrin strands and acute inflammatory cell infiltrate (HE staining, high-magnification).

Figure 5. Gram stain showing positive bacterial colonies.

IE is an uncommon manifestation of *Streptococcus agalactiae* (*S. agalactiae*) invasive disease, with overall incidence of 1.7% [5]. The demographic characteristics and outcome of *S. agalactiae* IE have changed over time. In the pre-antibiotic era, most cases were seen in pregnant women, with almost 100% mortality. Currently, IE caused by *S. agalactiae* is more common in elderly patients and in patients with chronic immunosuppressive conditions such as alcoholism, diabetes mellitus, liver cirrhosis, intravenous drug abuse, neoplasms, and HIV infection [6]. Nosocomial disease may arise from a new acquisition of the organism in the hospital or from preexisting skin or mucosal colonization. The source of infection may not be evident in certain cases.

IE due to GBS may affect both aortic valves and mitral valves. Tricuspid and pulmonic valve involvement has also been described in the literature. Patients with prosthetic valve endocarditis have the highest mortality rate.

It has been demonstrated that complement receptors present in neutrophils play a unique role in susceptibility to the disease. The underlying chronic diseases mentioned above compromise the host-defense mechanisms and alter the function of neutrophils by inhibiting complement receptors. In addition to host factors, the intrinsic virulence of *S. agalactiae* is determined by capsular polysaccharides specific to group B streptococci [7].

S. agalactiae endocarditis is characterized by acute onset of symptomatology and may result in rapid valve destruction [8]. Clinical characteristics of GBS IE are similar to those of IE due to *S. aureus*. Patients can present with signs of heart failure. It is an aggressive disease characterized by presence of large vegetations, rapid valvular destruction, and a high rate of local and systemic complications. The incidence of embolic phenomena is very high (50%) in contrast to patients with infective endocarditis due to viridans group streptococci [6]. The high rate of systemic embolization is explained by the inability of this organism to produce fibrinolysis, leading to large friable vegetations [9]. Embolization most commonly occurs within the first 2 to 4 weeks after onset [10]. The presence of left heart failure or central nervous system involvement is associated with a worse prognosis.

Common complications include cerebral embolism, septic arthritis, endophthalmitis, meningitis, cerebral embolisms, pulmonary embolisms, and peripheral vascular phenomena. Purulent pericarditis is a rare entity in the current antibiotic era and is reported to be caused by a wide variety of bacterial organisms, predominantly gram-positive cocci [11]. Infection of the pericardium can occur via direct extension from infectious endocarditis, perforating injury to the chest wall, or hematogenous spread from a more distant source. Clinical presentation is acute and patients usually manifest high-grade fever, chills, and tachycardia, while chest pain and pericardial friction rub are frequently not present [12].

In this case, the patient was immunocompromised due to uncontrolled diabetes mellitus, which made her susceptible to this life-threatening infection. Her atypical presentation made early diagnosis difficult.

Constitutional symptoms along with typical electrocardiographic findings of acute pericarditis and detection of pericardial fluid on transthoracic echocardiography facilitate the diagnosis. However, the definite diagnosis requires the presence of pus on microbiologic analysis of the pericardial fluid. Partial pericardiectomy showing extensive fibrinous exudate covering the visceral pericardium is typically seen on pathology. Purulent pericarditis has been rarely implicated as a complication of *S. agalactiae* bacteremia [13–15].

Group B streptococci are sensitive to penicillin G, ampicillin, and other semisynthetic penicillins. The concentration of penicillin required to inhibit group B streptococci (0.005 to 0.1 µg/mL) is greater than that needed for group A streptococci and *Streptococcus viridans*. Vancomycin, chloramphenicol,

first- and second-generation cephalosporins (excluding cefoxitin), and third-generation cephalosporins are effective alternatives. Aminoglycosides have little to no activity against group B streptococci when used alone, but provide synergistic activity when combined with ampicillin or penicillin G [16]. For uncomplicated infective endocarditis, antibiotic therapy is recommended for 4–6 weeks, including gentamicin for the first 2 weeks [17,18]. Vancomycin may be effective as an alternative choice in patients with immediate-type hypersensitivity to penicillins and cephalosporins [19]. Because of the high rate of local and systemic complications of *S. agalactiae* infection, non-responders to early medical management should undergo prompt surgical management [20, 21]. Treatment of purulent pericarditis requires a timely and aggressive approach with antibiotics and prompt drainage of the infected pericardial fluid [22].

Conclusions

S. agalactiae bacteremia can occur in immunocompromised patients and can lead to infective endocarditis and purulent pericarditis. High clinical suspicion is necessary for early diagnosis and treatment. Diagnosis is often difficult and delayed recognition can lead to life-threatening consequences. Prompt surgical drainage with complete emptying of purulent exudate should be done in cases where antibiotic treatment alone is not effective to improve prognosis and prevent fatal outcomes.

Conflict of Interests

The author(s) of the manuscript declare that there is no conflict of interest regarding the publication of this paper.

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