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Invasive Group A Streptococcus Infection Presenting as Purulent Pericarditis With Multiple Splenic Abscesses: Case Report and Literature Review

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Abstract

Purulent pericarditis is a localized infection of the pericardium producing an effusion that is both microscopically and macroscopically purulent. Purulent pericarditis is most frequently caused by *Staphylococcus*, although rarely *Streptococcus* and other organisms are implicated. This article describes a case of invasive group A streptococcal disease presenting as purulent pericarditis with multiple splenic abscesses in a 4-year-old boy.

Keywords

purulent pericarditis, group A streptococcus, invasive infection, *Streptococcus pyogenes*, splenic abscess

Introduction

Streptococcus pyogenes is a β-hemolytic bacterium that belongs to Lancefield serogroup A, also known as group A streptococcus (GAS). GAS is one of the most common human pathogens and causes a wide range of clinical diseases that include superficial, invasive, and toxin-mediated infections as well as postinfectious sequelae (Table 1).[1,2] Invasive GAS disease is defined as an infection with isolation of GAS from a normally sterile body site.[1] Purulent pericarditis (PP), a localized infection of the pericardial space, typically presents as an acute illness with fever, chills, and tachycardia. It is most commonly caused by *Staphylococcus aureus*. GAS remains a rare cause, having been reported in 7 cases.[3-7] We report the first case of invasive GAS infection manifesting as PP with multiple splenic abscesses.

Case Report

A previously healthy 4½-year-old boy was referred for evaluation of abdominal pain and fever. Two days prior to admission, he complained that his stomach hurt and pointed to the area around his umbilicus. During this time his appetite decreased but he did not have any vomiting, diarrhea, cough, or cold symptoms. One day prior to admission, he developed fever and decreased urine output. His mother confirmed that he never had an illness like this before, had no ill contacts or visitors from abroad, and had not been exposed to animals (specifically to cats or kittens). He had no prior hospitalizations, no known drug allergies, and his immunizations were up-to-date.

Physical examination revealed a well-developed, well-nourished male child in mild cardiorespiratory distress. Vital signs included a temperature of 97.6°F, heart rate of 147/minute, respiratory rate of 38/minute, blood pressure 86/54 mm Hg, and 100% oxygen saturation on 2 liters of nasal cannula oxygen. Auscultation of his chest revealed diminished breath sounds over the lung bases with slightly increased respiratory rate and muffled heart sounds with a pericardial rub near the lower left sternal border. His heart had a regular rate and rhythm with normal S1, S2, without an S3, S4 or a murmur. His abdomen was nontender, nondistended without mass or hepatosplenomegaly. The remainder of his examination was unremarkable.

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Table 1. Spectrum of Clinical Disease Caused by Group A Streptococcus

Asymptomatic colonization	Necrotizing fasciitis
Throat	Neonatal omphalitis
Skin (including the scalp)	Perianal dermatitis (anusitis)
Perineum	Phlebitis
Invasive disease	Pyoderma
Bacteremia/septicemia	Varicella superinfection
Puerperal sepsis	Wound infection
Cardiac	Suppurative respiratory disease
Endocarditis	Cervical lymphadenitis
Purulent pericarditis	Empyema
Central nervous system	Otitis media
Brain abscess	Parapharyngeal abscess
Meningitis	Peritonsillar abscess
Gastrointestinal/reticuloendothelial	Pneumonia
Liver abscess	Retropharyngeal abscess
Lymphadenitis	Sinusitis
Peritonitis	Postinfectious sequelae
Splenic abscess	Acute post-streptococcal glomerulonephritis
Genitourinary	Cutaneous polyarteritis nodosa
Urinary tract infection	Erythema nodosum
Musculoskeletal	Guttate psoriasis
Osteomyelitis	Pustulosis acuta generalisata
Pyomyositis	Reactive arthritis
Septic arthritis	Rheumatic fever
Skin/soft tissue suppurative disease	Superficial infection
Acropustulosis	Pharyngitis
Blistering distal dactylitis	Tonsillitis
Cellulitis	Toxin-mediated disease
Erysipelas	Bullous impetigo
Impetigo	Scarlet fever
Mastitis	Streptococcal toxic shock syndrome

Laboratory data on admission included a hemoglobin concentration of 9.7 g/dL, hematocrit 27%, platelets 341 000/mm^3, and white blood cell count 30 900/mm^3 with 89% neutrophils, 6% lymphocytes, and 4% monocytes. Serum chemistry demonstrated normal serum hepatic transaminases and renal function, albumin 2.3 g/dL, total protein 5.4 g/dL, glucose 133 mg/dL, prothrombin time 14.5 seconds with an international normalized ratio of 1.39, calcium 8.1 mg/dL, C-reactive protein 313 mg/mL, and D-dimer 2300 ng/mL. Serology for antinuclear antibody, rheumatoid factor, Epstein–Barr virus, and human immunodeficiency virus was negative. A chest radiograph revealed an enlarged cardiomediastinal silhouette, patchy bilateral opacities, and a small right pleural effusion. A computed tomography (CT) scan of the chest and abdomen demonstrated bilateral pleural effusions, bilateral patchy infiltrates, pericardial effusion, pericolic and periportal fluid, small ascites, and multiple small lucencies in the spleen (Figures 1 and 2). An electrocardiogram showed ST segment elevation. Echocardiogram demonstrated a moderate-size globally distributed pericardial effusion with right atrial and ventricular compression, moderate dilatation of the inferior vena cava, and trivial left and small right pleural effusion.

Clinical Course

Pericardial drainage was performed on the day of admission using a combination of CT and ultrasound guidance, removing a total of 150 mL of purulent fluid. Laboratory analysis of the pericardial fluid revealed a total cell count of 83 000/mm^3 including a white cell count of 79 000/mm^3 with 95% neutrophils, 3% lymphocytes, and 2% monocytes; red blood cells 4000/mm^3; lactate dehydrogenase 6450 IU/L; and protein 5 g/dL. Culture of the pericardial fluid yielded group A β-hemolytic streptococcus. There was no residual effusion following initial drainage but a pigtail catheter was placed in the pericardial sac for continuous drainage. He

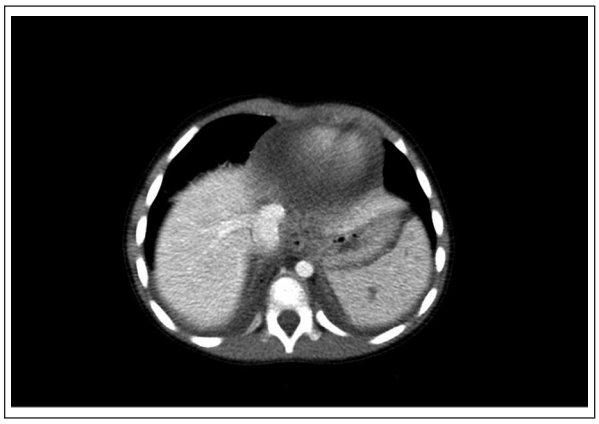

Figure 1. Computed tomographic scan demonstrating lucencies in the spleen and pericardial effusion

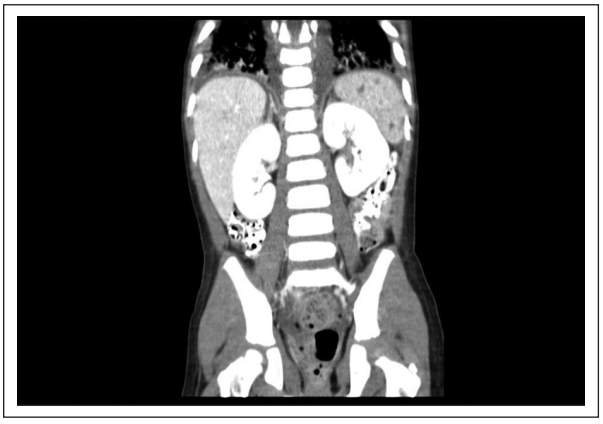

Figure 2. Computed tomographic scan demonstrating lucencies in the spleen, bibasilar atelectasis, and bilateral pleural effusions

had been started on ceftriaxone, clindamycin, vancomycin, and gentamicin. Cultures of blood, urine, and the throat were negative. After the pericardial fluid culture result was available, his antibiotic coverage was changed to penicillin G at a dose of 400 000 units/kg/d. Thereafter, the patient's condition gradually improved and his laboratory abnormalities normalized (Figure 3). One day before discharge, an echocardiogram showed a trivial pericardial effusion; an ultrasound of the abdomen showed several tiny hyperechoic lesions within the splenic parenchyma. At the end of the second week, the patient was discharged on intravenous penicillin G by continuous infusion.

He received 4 weeks of intravenous therapy and 4 weeks of oral amoxicillin therapy, during which time his echocardiogram and ultrasound of the spleen normalized (Figure 3).

Discussion

Group A β-hemolytic streptococcus is a common cause of a wide variety of clinical infections in infants, children, and adults. GAS infections have long been associated with serious morbidity and mortality, but toward the middle of the 20th century, a marked decline in incidence and severity of such infections occurred. However, since the late 1980s to mid-1990s, there has been resurgence in incidence of severe invasive GAS infections.[8,9] This has been attributed to a change in the epidemiology of GAS as well as to the virulence of the organism.[10] Invasive GAS disease is defined as an infection with isolation of GAS from a normally sterile body site and includes 3 overlapping clinical syndromes: GAS toxic shock syndrome, necrotizing fasciitis, and invasive disease not associated with presence of either necrotizing fasciitis or toxic shock syndrome.[11] This third category includes bacteremia with no identified focus and focal infections with or without bacteremia.[12]

Two reports of population-based surveillance of the epidemiology of invasive GAS infection in 5 states in the United States from July 1995 to December 2004 have identified a total of 7402 cases of invasive GAS infection, with a peak incidence in those younger than 2 years and older than 65 years.[13,14] The presence of streptococcal toxic shock syndrome, meningitis, necrotizing fasciitis, pneumonia, bacteremia, and infection with *emm1* or *emm3* serotypes were all noted to be independent predictors of death. Of 7402 cases of invasive GAS infection during a 10-year period, only 104 cases (1 case in <10 years of age and 103 cases in >10 years of age) had either endocarditis and/or pericarditis,[13,14] and GAS was isolated from pericardial fluid in only 1 patient.[13]

PP is defined as a localized infection of the pericardial space producing an effusion that is both microscopically and macroscopically purulent.[15] The advent of antibiotics has changed the epidemiological features of PP. During the pre-antibiotic era, PP accounted for 40% of all cases of acute pericarditis and was related to contiguous spread of lung infections in 72% of cases. Currently, pneumonia remains a primary source of infection in only 22% of cases, while PP is mainly associated with health care and bloodstream infections.[16-18] Patients with PP typically have an acute illness characterized by fever, chills, and tachycardia. However, the clinical presentation and complications of PP can vary. In a recent review of 65 cases, common clinical features were fever (85%), chest pain (31%), pericardial friction rub (33%), electrocardiographic abnormalities (45%), cardiac tamponade (15%), and constrictive pericarditis (3.5%).[18] PP is most frequently caused by *Staphylococcus*, but *Streptococcus*, *Proteus*,

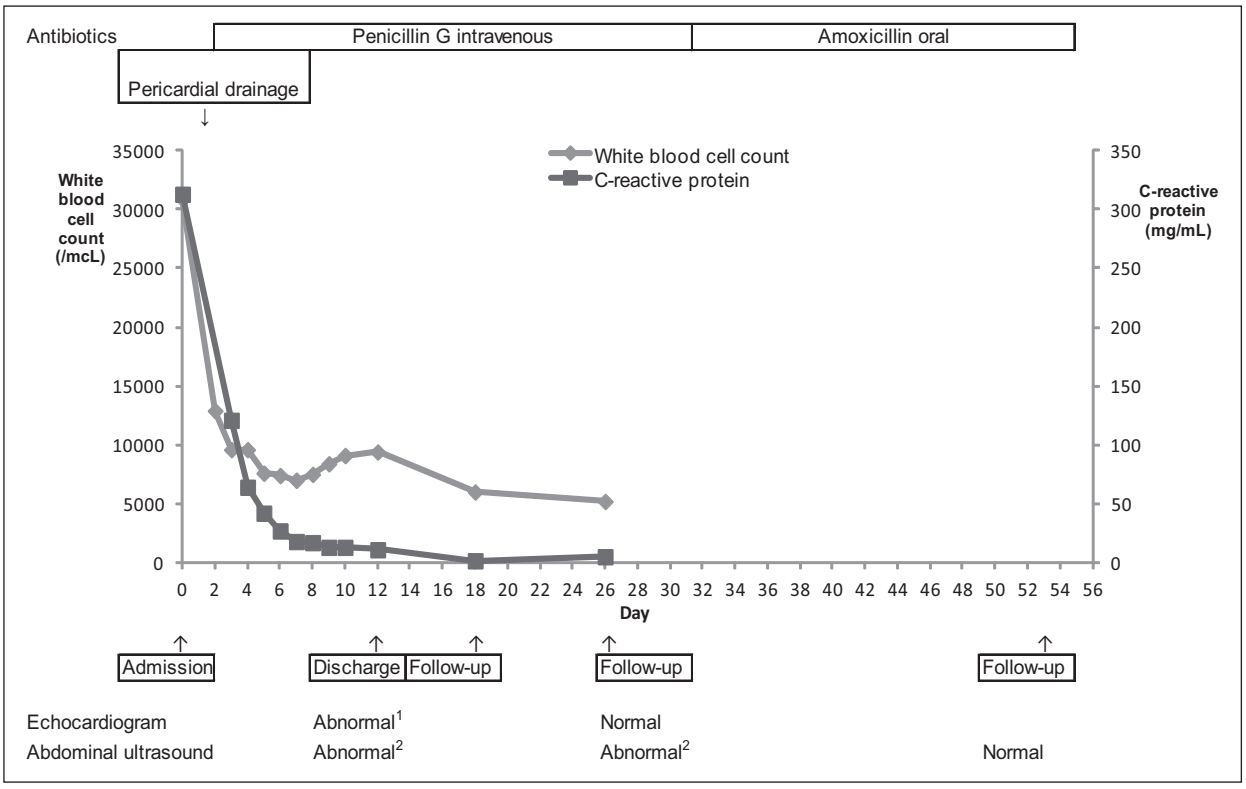

Figure 3. Clinical, laboratory, and imaging features of the case
[1]Presence of trivial effusion.
[2]Several tiny hyperechoic lesions within the splenic parenchyma.

Escherichia, *Pseudomonas*, and *Klebsiella* PP may occur.[19,20]

Among the reported cases of GAS induced PP (Table 2), the ages were 13 months, 14 months, 2½ years, 3 years, 3 years, 4½ years, 6 years, and 14 years (mean age ~4 years). The most common clinical presentation in these patients was fever and tachycardia; other manifestations included respiratory distress, abdominal pain, and altered mental status. The time from initial presentation to diagnosis was approximately 2 to 3 days. Pericardial fluid cultures were positive for GAS in all the patients; blood culture was positive in only 1 patient. Of 7 patients who survived, all were diagnosed with cardiac tamponade based on symptoms and echocardiographic findings, required pericardial drainage, and were treated with antibiotics for 4 to 8 weeks. Serotyping of GAS was performed in only 1 patient.[7] None of the patients developed constrictive pericarditis.[3-7]

A novel finding in the patient described here was the presence of multiple tiny abscesses in the spleen. Splenic abscess in invasive GAS infection has been reported only once, in a 7-year-old boy.[21] Initially, splenectomy combined with antibiotic therapy was the treatment of choice, but medical therapy alone has recently been successful, with serial ultrasound and CT examinations demonstrating resolution of the abscesses.[22,23]

PP is a severe infection that should be diagnosed and treated aggressively before life-threatening complications, such as cardiac tamponade and constrictive pericarditis, develop. Although a few cases of PP caused by GAS have been published in the literature, it generally has not been listed as one of the manifestations of invasive GAS disease.[7] Among 7 reported cases of PP caused by GAS, 6 developed cardiac tamponade and 1 resulted in death, indicating the severity of this disease. Therefore, PP should be considered among the manifestations of severe invasive GAS disease.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Table 2. Purulent Pericarditis Caused by Group A Streptococcus

Patient	Reference	Age	Presenting Symptoms and Signs	Therapy	Complications
1	3	14 years	Sore throat, headache, vomiting, rash; pulsus paradoxus	Benzylpenicillin (1 week) then oral penicillin (4 weeks); pericardiocentesis and pericardial window	Cardiac tamponade
2	4	2½ years	Fever, fussiness, cough, and not sleeping well	Pericardiocentesis	Death
3	5	13 months	Not reported	Ampicillin and gentamicin; pericardiocentesis	Cardiac tamponade
4	5	14 months	Not reported	Oxacillin and amikacin; pericardiocentesis	Cardiac tamponade
5	5	3 years	Not reported	Amoxicillin and netilmycin; pericardiocentesis	Cardiac tamponade
6	6	6 years	Fever and altered mental status	Penicillin G (4 weeks); pericardiocentesis and pericardiectomy	Cardiac tamponade
7	7	3 years	Respiratory distress, abdominal pain, and fever; muffled heart sounds, pulsus paradoxus, and cardiogenic shock	Penicillin and clindamycin (8 weeks); pericardial drainage	Cardiac tamponade
8	This report	4½ years	Abdominal pain and fever, muffled heart sounds, and pericardial rub	Penicillin G (4 weeks) then amoxicillin (4 weeks); pericardial drainage	Cardiac tamponade

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