Introduction  
Since penicillin became available, the incidence of pneumococcal pericarditis has decreased significantly. Although the pathogenesis remains a matter of debate, direct extension from a pleural empyema may explain some cases of pneumococcal pericarditis. However, it is not known which factors determine who will get an infectious cardiac complication following pneumococcal bacteremia. A high index of suspicion is needed to detect patients who present with purulent pericarditis, along with good cooperation and communication between the clinic, and laboratory and radiology services.

Case Report  
A 51-year-old patient was admitted to the emergency department with septic shock. He had a history of epileptic seizures, chronic obstructive pulmonary disease, and a psychiatric disorder. He underwent several surgical procedures involving the femur, shoulder, and appendix. His medication was sodium valproate, acetylsalicylic acid, dipotassium chlorazepate, ranitidine, escitalopram oxalate, and zolpidem hemitartrate. Two weeks before admission, he suffered from a thoracic trauma with contusion of the ribs. No treatment was initiated. Since then, his condition had deteriorated with defined epigastric pain and diarrhea.

On physical examination, the patient appeared ill, neglected, and sweaty and clammy. His temperature was 37.8°C; blood pressure, 55/38 mm Hg; heart rate, 114 beats per minute; and oxygen saturation level, 89%. Physical examination revealed distension of the jugular veins, normal heart sounds without heart murmurs or rubs, and bilateral lower vesicular respiratory sounds.

Blood analysis revealed a moderate anemia with a hemoglobin level of 11.9 g/dl (normal range, 13.1 to 17.2 g/dl), a white blood cell count of 25,900/µl (normal range, 4,000 to 10,000/µl) with 73% polymorphonuclear cells, an elevated erythrocyte sedimentation rate, and an increased C-reactive protein of 29.8 mg/dl (normal range, 0 to 9 mg/dl). His troponin I level was slightly elevated at 0.230 ng/ml (normal cutoff point, <0.05 ng/ml). The aspartate (AST) and alanine (ALT) serum aminotransferase and creatinine levels were elevated. The AST was 88 U/L (normal range, 10 to 41 U/L), and the ALT was 60 U/L (normal range, 10 to 44 U/L). The creatinine level of 3.6 mg/dl (normal range, 0.8 to 1.3 mg/dl) was compatible with the patient’s hemodynamic instability.

Conventional X rays showed a distinct cardiomegaly. An ECG showed a diffuse ST elevation, indicating a possible pericardial tamponade. Two-dimensional echocardiography showed the presence of a large amount of pericardial fluid. CT scan confirmed the presence of a massive amount of circumferential pericardial effusion, together with hepatomegaly and pleural effusion (Fig. 1). Subxiphoid pericardiocentesis yielded over 1,000 ml of purulent fluid and was followed by prompt return of the patient’s blood pressure to normal. Blood cultures were drawn.

Direct Gram stain of the pericardial fluid showed many polymorphonuclear cells with typical gram-positive, lancet-shaped diplococci arranged in longitudinal pairs. The pericardial fluid and blood cultures also yielded gram-positive diplococci suggestive of Streptococcus pneumoniae. The bacteria had large capsules, which is associated with strain virulence. Cultures on horse blood agar grew mucoid, alpha-hemolytic colonies with typical sunken centers due to spontaneous autolysis of older organisms. The isolates were optochin susceptible, and agglutination was positive for S. pneumoniae (Slide pneumo-kit; bioMérieux, Marcy l’Etoile, France). Microbiologic analysis of the isolate at the Laboratory of Microbiology, Catholic University of Leuven, by Professor J. Verhaegen confirmed the identity as S. pneumoniae serotype 12.

The isolates were penicillin and ceftriaxone susceptible (using the CLSI breakpoints for S. pneumoniae), and the MIC for both antimicrobials was...
0.016 mg/L. The patient was treated with penicillin G for 28 days and aminoglycosides for 3 days. After successful treatment, the patient was vaccinated with Pneumovax and discharged after 28 days of hospitalization.

**Discussion**

Hematogenous spread of *S. pneumoniae* may cause infection in different locations, including the pericardium. Since penicillin became available, pneumococcal pericarditis has been rare. Fewer than 20 cases have been reported since 1980 (1). The pathogenesis of pneumococcal complications, such as pericarditis and endocarditis, remains a matter of debate. Although some early studies showed that pneumococcal serotypes 12, 1, and 8 are the most common causes of pneumococcal endocarditis, others have shown a variety of capsular types causing infectious cardiac complications (1,2). To the best of our knowledge, no studies link particular pneumococcal serotypes to pericarditis. Typical clinical signs of pericarditis, such as pulsus paradoxus, friction rub, distended neck veins, and distant heart sounds, are not always present. A direct extension from a pleural empyema may explain some cases of pneumococcal pericarditis, but it is not known which factors determine whether a patient will develop an infectious cardiac complication following pneumococcal bacteremia.

A high index of suspicion and awareness is needed when patients present with purulent pericarditis (3-5). Good cooperation and communication between the clinic and laboratory and radiology services can lead to a rapid and accurate diagnosis that affords the patient the best chances for survival.

**References**