CASE REPORT

A 53-year-old man with long-standing, steroid-dependent rheumatoid arthritis complained of a painful, swollen, left elbow with purulent drainage emanating from what appeared to be a small ulceration. Two weeks before the visit, minor trauma to that area had occurred. Prosthetic articular surfaces had been implanted in the elbow several years earlier. There was no prior history of diabetes mellitus or chronic infections. The wound was cultured and oral cephalaxin was started. He returned to the clinic after several days without any clinical improvement. The patient was admitted to the hospital, and blood and wound cultures were obtained; empiric cefepime and vancomycin were started. Because of persistent fever and continuing joint drainage, he was transferred to a tertiary facility for surgical treatment of presumed septic arthritis. Minimal articular fluid was present at surgery. The early postoperative course, however, was remarkable for persistent fever, hypotension, and tachycardia. On the first postoperative day, he was transferred to the medical service for the management of presumed septic shock.

On physical examination, he was ill-appearing in moderate respiratory distress. The temperature was 39°C, blood pressure was 100/70 mmHg, heart rate was 130 beats per minute, and oxygen saturation was 96% on 3 L oxygen per nasal cannula. Physical examination was remarkable for: marked jugular venous distention; clear lungs; normal heart sounds without heart murmurs or rubs; and a benign abdomen. Severe rheumatoid joint deformities were present. The left elbow revealed periarticular erythema with serous drainage. The white blood cell (WBC) count was 19,000/μL with left shift. Serum chemistries and urinalysis were normal. A chest x-ray (Fig. 1) revealed an enlarged cardiac silhouette and widened mediastinum. An EKG (Fig. 2) showed diffuse ST elevation, PR depression, and lateral T-waves inversion. A clinical diagnosis of pericardial tamponade was made. Two-dimensional echocardiography (Fig. 3) confirmed the presence of a circumferential pericardial effusion. There was evidence of right ventricular diastolic compression, consistent with pericardial tamponade. A subxiphoid pericardiocentesis yielded 500 mL of purulent fluid with prompt normalization of the blood pressure. Pericardial fluid pH was 8.0; WBC, 126,000/μL (100% neutrophils); RBC, 180,000/μL; lactate dehydrogenase (LDH), 43,300 U/L; protein, 4.8 g/dL; triglycerides, 18 mg/dL. A Gram stain was negative for organisms. A pericardial window was later placed surgically with drainage of an additional 375 mL of purulent fluid. Antibiotic treatment was changed to vancomycin, gentamicin, and nafcillin.

KEY WORDS: septic arthritis; purulent pericarditis; pericardial tamponade.
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INTRODUCTION

Septic arthritis is a well recognized occurrence in patients with steroid dependent rheumatoid arthritis. Treatment includes broad-spectrum antibiotics usually accompanied by surgical or needle drainage of the joint. While pericardial effusions are common in patients with rheumatologic disorders, the development of purulent pericarditis with pericardial tamponade is rare. We report a case of purulent pericarditis with pericardial tamponade masquerading as septic shock related to Proteus mirabilis septic arthritis.
While multiple blood cultures were negative, articular and pericardial fluid cultures grew *Staphylococcus epidermidis* and *Proteus mirabilis*. Antibiotic sensitivities were identical in both fluids. Cultures for *Mycobacterium* spp., *Mycoplasma* spp., *Nocardia* spp., and fungi were negative. While *Candida albicans* was present in the urine, there was no evidence of a *Proteus* urinary tract infection. Head, chest, abdomen, and pelvis computerized tomography scans were performed to exclude possible sites of metastatic infection. These were all unremarkable. Coronary arteriography was performed to
exclude a coronary mycotic aneurysm. A radioabeled leukocyte scan was positive in the left elbow and other areas of joint replacement. The patient responded well to intravenous cefotaxime and surgical drainage. The fever and hemodynamic instability resolved. He completed a 6-week course of intravenous antibiotics. There was no recurrence of the pericardial effusion by echocardiography 8 weeks post-discharge. The infection was presumably eradicated.

**DISCUSSION**

In the setting of an infected joint prosthesis, fever, and immunosuppression, this patient’s hemodynamic instability was initially ascribed to septic shock and not to pericardial tamponade. The usual signs and symptoms of pericardial inflammation were absent. The expected chest pain and a pericardial friction rub were missing and were, no doubt, attenuated by the chronic steroid use. Despite a large and grossly purulent pericardial effusion, the electrocardiographic findings were quite subtle. Only a high index of suspicion prompted the consideration of pericardial tamponade. A careful physical examination and critical interpretation of the chest radiograph were essential steps in establishing the correct diagnosis. In this case, prompt consideration of pericardial tamponade led to a life-saving pericardiocentesis. The abnormally high pH of the pericardial fluid presaged the possibility of a Proteus infection. This initial observation facilitated proper antibiotic selection and early surgical intervention.

Purulent pericarditis with tamponade is an uncommon condition with a high mortality. Survival is dependent upon prompt recognition and aggressive treatment. Pericardial involvement is usually a result of contiguous spread of an adjacent intrathoracic infection. Blood borne infections account for less than 30% of the cases of purulent pericarditis. Gram-positive organisms continue to be the most commonly observed pathogens, although, there is evidence that the increased utilization of antibiotics may favor the emergence of Gram-negative organisms. Proteus mirabilis, a common urologic pathogen, is a distinctly uncommon cause of purulent pericarditis. Purulent pericarditis due to Proteus spp. is rare and has been previously reported in association with pneumonia and coronary bypass surgery. Septic arthritis leading to pericardial infection is equally uncommon. An association between septic arthritis and purulent pericarditis has been observed with Hemophilus influenzae, Neisseria meningitides, and Neisseria gonorrhoaeae. The simultaneous occurrence of Proteus arthritis and pericarditis in this patient is unique and has not been previously reported.

It is highly likely that a single pathogen was responsible for both the articular and pericardial infections. Based on antibiotic sensitivities, the Proteus isolated from the joint space and pericardial fluid appears to be identical. An extensive evaluation confirmed that the source of pericardial infection was the infected prosthetic elbow. There was no other evidence of any other extracardiac infection. The traumatized elbow was the most likely portal of entry. The resulting articular infection then spread hematogenously to the pericardial space without any evidence of additional intrapulmonary or intracardiac infection. The emergence of this atypical organism may have been facilitated by several factors. The initial treatment with oral cephalixin may have been inadequate for septic arthritis in an immunocompromised patients. Likewise, early failure to drain and debride the infected joint may have facilitated the systemic spread of the infection.

While pericardial effusions are commonly present in patients with rheumatologic disorders, pericardial infections and pericardial tamponade are not. This case illustrates how, in the setting of a chronic pericardial effusion and chronic immunosuppressive therapy, a blood borne infection may seed the pericardial space. It further demonstrates how rapidly, a closed space bacterial infection may then rapidly progress to pericardial tamponade, masquerading as septic shock.

The present case is noteworthy for several reasons. It emphasizes the importance of a careful physical examination and the thoughtful synthesis of ancillary studies. In immunocompromised patients, the typical signs and symptoms of pericarditis may be absent, and the clinical presentation of pericardial tamponade may be misinterpreted as one of septic shock. It further illustrates the potential for atypical transmission, to the pericardium, of Gram-negative pathogens in patients with a high likelihood of preexisting pericardial disease. Knowledge of this possibility should facilitate prompt recognition and management of this life-threatening condition. This case also underscores the importance of appropriate antibiotic selection in the initial treatment of immunocompromised patients with infected prosthetic joints. This would include the need for the administration of intravenous broad-spectrum antibiotics with surgical lavage and debridement of the infected space.

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