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Staphylococcus aureus Pericarditis in HIV-infected Patients*

Catherine F. Decker, M.D.; and Carmelita U. Tuazon, M.D.

Serious infections caused by Staphylococcus aureus in HIV-infected patients have been reported. Contributing factors in the development of invasive S aureus infections include a high rate of skin and nasal colonization, frequent dermatologic disease, and the use of intravenous catheters. The authors report three cases of S aureus pericarditis in HIV-infected patients. While cases of viral, mycobacterial, and malignant pericardial effusions in HIV-infected patients have been reported, a review of the literature disclosed only three cases of bacterial pericarditis. Despite appropriate antibiotic therapy and drainage, a patient’s condition may abruptly deteriorate and progress to tamponade. Early recognition of bacteremia and pericarditis and monitoring for cardiac tamponade, along with aggressive treatment, can result in a favorable outcome, but mortality remains high, particularly when S aureus is the causative agent.

Serious infections, such as bacteremias and soft-tissue infections, caused by Staphylococcus aureus in HIV-infected patients have been reported recently.14 Risk factors include a high rate of skin and nasal colonization,24 frequent dermatologic disease,3 and the use of intravenous catheters.1

While cases of viral, mycobacterial, and malignant pericardial effusions in HIV-infected patients have been reported, bacterial pericarditis is rare.67 Three cases of S aureus pericarditis in HIV-infected patients and a review of the literature are presented.

CASE REPORTS

Case 1

A 35-year-old HIV-seropositive black man, an intravenous heroin and cocaine user, was admitted for dyspnea, malaise, and chest pain of 3 weeks’ duration. There was no history of opportunistic infection, except for oral candidiasis. Significant findings included oral candidiasis, purpular lesions on the extremities, and a pericardial friction rub. The chest radiograph revealed an enlarged cardiac silhouette, a small pleural effusion, and a retromediastinal avelocrine infiltrate. The Feo was 70 mm Hg on room air. The ECG showed sinus tachycardia with global 1- to 2-mm ST-segment elevation. A two-dimensional echocardiogram revealed moderate pericardial effusion without hemodynamic compromise.

Intravenous trimethoprim-sulfamethoxazole was begun for presumptive Pneumocystis carinii pneumonia. Multiple sets of blood, urine, and sputum cultures were positive for S aureus. Trimethoprim-sulfamethoxazole was discontinued, and intravenous vancomycin was instituted. Pericardiocentesis yielded 350 ml of clear yellow exudative fluid, which grew S aureus. When the antibiotic susceptibility results were obtained, vancomycin was replaced by nafcillin. A pericardial window was performed when a repeat echocardiogram revealed a reaccumulation of pericardial fluid. Repeat blood and pericardial fluid cultures were sterile. The patient did well and was discharged in good condition.

Case 2

A 39-year-old black man with AIDS and cutaneous Kaposi’s sarcoma was admitted because of dyspnea, fever, and cough. Significant findings were fever of 38.4°C, pulse paradoxicus, diffuse ronchi, and an S3-S4 gallop with pericardial rub. A chest radiograph revealed an enlarged cardiac silhouette and bilateral lower lobe and lingual infiltrates. An ECG showed sinus tachycardia, diffuse ST-segment elevation, and diffuse PR depression.

An echocardiogram revealed a large pericardial effusion. Emergent pericardiocentesis yielded 300 ml of serosanguinous fluid with marked relief of respiratory symptoms. Sputum and pericardial fluid cultures grew S aureus, and intravenous nafcillin was administered. Nafcillin was replaced with vancomycin after the patient developed a diffuse rash.

The patient’s clinical course deteriorated and was complicated by development of Candida albicans empyema. An open-lung biopsy also showed cytomegalovirus pneumonitis. Intravenous ganciclovir therapy was begun. His respiratory status continued to decline, and he died 7 weeks after admission.

Case 3

A 35-year-old HIV-positive female drug abuser presented with fever, headache, and cough. She had a history of S aureus endocarditis.
Significant findings included fever, diffuse ronchi and wheezes, a systolic ejection murmur, and multiple skin abscesses on the extremities. A chest radiograph revealed bilateral interstitial markings with a normal cardiac silhouette. She had a PaO₂ of 62 mm Hg on room air. Bronchoscopy with bronchoalveolar lavage was negative for P carinii. Blood cultures grew Gram-positive cocci, and intravenous vancomycin was administered. Twenty-four hours later, the patient became hypotensive and developed ventricular tachycardia. An emergent echocardiogram revealed a large pericardial effusion. Pericardiocentesis yielded 75 ml of turbid brown fluid. Further attempts at resuscitation were unsuccessful, and the patient died. Blood and pericardial fluid grew S aureus.

DISCUSSION
Pericardial effusion, including tamponade, is a common cardiac manifestation of AIDS. Up to 23 percent of HIV-infected patients with cardiac disease will have pericardial effusion. Pericardial disease in AIDS has protein manifestations, including silent pericardial effusion diagnosed incidentally by echocardiography and classic fibrinous pericarditis with pain, friction rub, and ST-segment elevation. Hemodynamically significant pericardial effusion with tamponade has been reported. Patients with pericardial disease usually have concomitant involvement of the myocardium. Pericardial disease may provide a potential site for local or metastatic infection, particularly in the setting of bacteremia or fungemia.

Symptomatic pericardial effusions are usually caused by opportunistic organisms, such as Mycobacterium tuberculosis, Mycobacterium avium complex, cytomegalovirus, Nocardia asteroides, and Cryptococcus neoformans, but malignant pericardial effusion secondary to Kaposi's sarcoma and B cell lymphoma involving the heart and pericardium can also be found.

Although bacterial infections occur with increased frequency in HIV-infected patients, bacterial pericarditis has been notably rare. To our knowledge, only three documented cases of bacterial pericarditis in AIDS patients have been reported: one case of S aureus pericarditis with cardiac tamponade and two cases of pneumococcal pericarditis. The latter two cases progressed to purulent pericarditis and cardiac tamponade, despite prompt and appropriate therapy, which responded to pericardiectomy.

Patients infected with HIV, similar to patients with chronic renal disease, diabetics, and intravenous drug users, are at risk for serious infections caused by S aureus. Patients with AIDS are at higher risk for S aureus infections, which may be related to higher nasal carriage and skin colonization. Colonization has been associated with increased incidence of infections in certain risk groups. Although such association has not been fully determined in the HIV-infected patient, one may postulate that colonization with S aureus may lead to serious infection.

Similarly, a wide spectrum of dermatologic disease affects HIV-infected patients. In addition, their neutrophils have decreased ability to bind and ingest S aureus organisms. Tran-

S aureus Pericarditis in HIV-infected Patients (Decker; Tuazon)

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