Pulmonary Botryomycosis in a Patient With AIDS*

Kanchan Katapadi, MD; Fernando Pujol, MD; Juan C. Valetin, MD; Mannohanan Katapadi, MD; and Bruce R. Pachter, PhD

We describe the clinical and pathologic findings of the first reported case of pulmonary botryomycosis in a patient with AIDS. Botryomycosis is an uncommon, chronic, suppurative disease that is often mistaken clinically and histologically for a fungal infection. The patient responded to systemic antibiotic therapy.

(CHEST 1996; 109:276-78)

Key words: AIDS; pathology; pulmonary botryomycosis

Botryomycosis, which closely mimics fungal infections both clinically and histologically, is an uncommon chronic suppurative and sometimes granulomatous infection generally involving the skin, tela subcutanea (subcutis), and occasionally the viscera and is caused by bacteria. The hallmark of the disease is the presence of eosinophilic fungus-like granules in a suppurative lesion; the granules consist of the causative bacteria.

Recently, three cases of cutaneous botryomycosis1-3 have been reported in AIDS patients. In the present article, we report what is to our knowledge the first case of pulmonary botryomycosis in a patient with AIDS.

CASE REPORT

Clinical Data

A 36-year-old heterosexual male, chronic smoker presented with a history of blood-streaked sputum and cough for 3 days. He had no history of fever, chills, shortness of breath, persistent cough, weight loss, nor any exposure to tuberculosis.

The physical and clinical examination disclosed no abnormalities except for severe human papilloma virus infection involving the penis. The CBC count and differential cell counts were within normal limits. A chest x-ray film showed a single mass, 5 cm in diameter, located in the upper lobe of the right lung with a central area of low attenuation (Fig 1). A CT scan showed the mass to be

![Figure 1. Chest x-ray film showing a single mass (5 cm) in upper area of right lung (arrow).](image1)

![Figure 2. CT scan of the chest showing a pleural-based mass in the upper area of right lung with central low attenuation area (arrow).](image2)

*From the Department of Medicine and Pathology, New York Methodist Hospital, Brooklyn. Reprint requests: Dr. Pachter, Department of Medicine, New York Methodist Hospital, 506 Sixth Street, Brooklyn, NY 11215
Figure 3. Top: botryomycotic granule surrounded by purulent exudate. Under this magnification, the granule cannot be distinguished from an actinomycotic one (hematoxylin-eosin stain, original magnification x125). Bottom: similar granule at higher magnification showing numerous Gram-positive cocci (Brown and Brenn stain, original magnification x500).

adjacent to the pleura (Fig 2). A scan of the liver and spleen showed splenomegaly and an accessory spleen adjacent to the posteromedial portion of the spleen. A CT-guided fine needle aspiration percutaneous lung biopsy was performed.

Pathology

The aspirated specimen consisted of multiple fragments of pink-tan to yellow soft friable material measuring 1.0 x 1.0 x 0.2 cm as an aggregate. Smears and sections from a cell block were prepared and stained with the Papanicolaou and hematoxylin-eosin stains, respectively. Sections were also stained with Brown and Brenn, Gomori’s methenamine silver, and acid-fast stains.

The routine stains showed a purulent infiltrate composed mainly of polymorphonuclear leukocytes, with an admixture of erythrocytes, occasional macrophages, lymphocytes, and plasma cells. Sprinkled in this infiltrate were numerous basophilic coccal colonies which were generally outlined by a layer of hyaline material of variable thickness (Fig 3, top). The bacterial colonies were Gram-positive on the Brown and Brenn stain (Fig 3, bottom). They also stained weakly with the Gomori’s methenamine silver stain. No fungi or acid-fast bacilli were detected. A diagnosis of pulmonary botryomycosis probably due to Staphylococcus aureus was made on the basis of the aforementioned microscopic findings. Bacterial cultures or sensitivity studies were not undertaken. The patient was then discharged and treated successfully with orally administered amoxicillin-clavulanate (Augmentin) at home and was followed up in the Infectious Diseases Clinic. The patient refused to be tested for HIV at this time.

Additional Clinical Data

The patient was readmitted 6 months later with a history of pleuritic chest pain, fever, and productive cough. A diagnosis of pneumonia was made. Both a chest x-ray film and the CT scan of the chest showed that the previously seen pleural mass was no longer visualized and there was a dense area of fibrosis occupying that area (Fig 4). In addition, a new consolidation was seen in the middle lobe of the right lung. The patient was found to be hepatitis B and hepatitis C antibody-positive. He eventually agreed to be tested for HIV and was found to be HIV-positive. The HIV helper cell (CD4+) count was 8/mm³, and the HIV suppressor cell (CD8+) count was 248/mm³. The CD4+/CD8+ ratio was 0.03. The patient was successfully treated with intravenously administered erythromycin for his pneumonia and was discharged to his home. The patient was being followed up in the Infectious Diseases Clinic for several months until he stopped coming to the clinic.

Discussion

Botryomycosis is a chronic supplicative lesion with characteristic grain formation in the pus caused by bacteria and frequently is mistaken for a fungal infection. It was first described in horses in 1870 by Bollinger and termed botryomycosis (Greek botrys for bunch of grapes and mycosis for fungal origin) by Rivolta, who thought it was a fungal infection. It was Magrou, who established the bacterial origin of botryomycosis in 1919, by isolating Staphylococcus aureus from an equine lesion and by reproducing the disease in guinea pigs. Two types of botryomycosis have been described, intemimentary (cutaneous) and visceral forms. More than 80 cases (60 intemimentary and 20 visceral) have been reported in the literature. Those cases involving the visceral organs included the liver, the kidney, the brain, the prostate gland, and the lungs. Recently, three cases have been reported in AIDS patients, all of which were intemimentary.

The exact pathogenesis of the disease is not known. However, several bacterial agents are known to cause it. Most common is S aureus, as presumed in our patient. Less frequently, various other bacteria such as Pseudomonas aeruginosa, Escherichia coli, α-hemolytic Streptococcus, Actinobacillus lignieresii, and more recently Moraxella nonliquefaciens, Serratia marcescens, and Peptostreptococcus.
have been reported.\textsuperscript{10} It has been postulated that various combinations of the following factors are responsible in the pathogenesis of this disease: (1) trauma, (2) presence of foreign body, (3) size of the inoculum, (4) organisms of low virulence and those whose virulence has been altered by antibiotic therapy, and (5) host factors.\textsuperscript{7} Botryomycosis has been reported in patients with alcoholism, diabetes mellitus, cystic fibrosis, malnutrition, steroid treatment, immune disturbances, and more recently, AIDS.\textsuperscript{1-3,7-8}

With most bacterial invasions, cellular defense includes instant killing of bacteria by neutrophils and mononuclear macrophage cells. Their bactericidal functions are enhanced by lymphokines released by T4 cells. In botryomycosis, the bacteria are not destroyed in tissues by host defenses, and they therefore exist in a delicate balance within the host. In a patient with AIDS, the defective CD4\textsuperscript{+} function results in a decreased lymphokine release, which results in a failure of activation of the monocyctic bacterial enzymes leading to defective intracellular killing of IgG-coated organisms.\textsuperscript{10}

Pulmonary botryomycosis previously has been reported in ten patients; eight cases were in children with cystic fibrosis and two were in patients who had no underlying disease. The present patient represents the 11th case of pulmonary botryomycosis to be reported in the English-language medical literature and apparently, to our knowledge, is the first case in an AIDS patient.

This article emphasizes the need for increased physician awareness of this unusual disease entity as a differential diagnosis in pulmonary infections in AIDS patients in contrast to the more common fungal infections, tuberculosis, and actinomycosis.

**REFERENCES**


**Pulmonary Edema Following Electrical Cardioversion of Atrial Fibrillation**

Bongani M. Mayosi, BMEdSc, MBChB; and Patrick J. Commerford, MBChB

A 61-year-old man with hypertrophic cardiomyopathy developed acute pulmonary edema 29 h following cardioversion of chronic atrial fibrillation to sinus rhythm. Doppler echocardiographic evaluation of atrial function showed return of right atrial contraction but absent left atrial systole. This has not been reported previously in a case of postcardioversion pulmonary edema. (CHEST 1996; 109:278-80)

**Key words:** cardioversion; left atrial function; postcardioversion pulmonary edema

Although pulmonary edema has been reported to occur after cardioversion,\textsuperscript{1} the mechanism is poorly understood. Ineffective left atrial function which may occur immediately following cardioversion\textsuperscript{2} has been suggested as a contributing factor, although it has not been demonstrated in a case of postcardioversion pulmonary edema to date. This report describes a patient in whom chronic atrial fibrillation was treated with electrical cardioversion resulting in the development of pulmonary edema 29 h later. Pulsed Doppler echocardiography of tricuspid and mitral inflow patterns showed absent left atrial systole but restoration of right atrial mechanical activity which lends support to the left atrial failure theory of causation of postcardioversion pulmonary edema.

**Case Report**

A 61-year-old normotensive man who had hypertrophic cardiomyopathy for 26 years was admitted to the hospital for elective cardioversion of atrial fibrillation. He presented 9 months previously with acute pulmonary edema for the first time; this episode of edema was precipitated by non-Q wave myocardial infarction. Cardiac catheterization confirmed the features of hypertrophic cardiomyopathy associated with moderately impaired left ventricular function and minimal coronary artery disease. Apart from palpitations caused by atrial fibrillation, he had a normal functional capacity while receiving therapy with the following drugs: digoxin, captopril, furosemide, carvedilol, and warfarin.

On examination, there were no signs of cardiac failure. The blood pressure was 140/70 mm Hg, and the pulse rate 88 beats per minute and irregular. The apex beat was displaced laterally outside the midclavicular line in the fifth intercostal space. The first heart sound was normal, and the second heart sound was physiologically split. There was no third or fourth heart sound, and no murmurs were present. The lungs were clear. His ECG demonstrated atrial fibrillation with a ventricular response of 87 beats per minute. The chest x-ray film done 1 month before entry showed left ventricular cardiomegaly and clear lung fields. The digoxin level was therapeutic.

*From the Cardiac Clinic, Department of Medicine, University of Cape Town, and Groote Schuur Hospital Observatory, Cape Town, South Africa.*