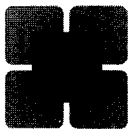

WILLIAM S. WILKE, MD, EDITOR

TREATING RESPIRATORY INFECTIONS IN HIV-POSITIVE PATIENTS

Patients positive for human immunodeficiency virus (HIV) are frequently assumed to have an increased susceptibility to every conceivable pathogen. Actually, though, due to the particular way HIV attacks the immune system, some secondary infections are much more common than others. The most profound immunodeficiency is the loss of T-helper lymphocytes (CD4 cells), which renders the body less resistant to certain fungi, viruses, and parasites. Bacterial infections can also occur because polyclonal B-cells are stimulated when CD4 cells fail to regulate their function, thus creating a functional immunoglobulin deficiency.

The most common infectious manifestations are respiratory diseases, and their management can be challenging. The diagnosis can be complicated by the presence of noninfectious disorders that produce respiratory symptoms, such as visceral Kaposi's sarcoma and lymphocytic pneumonitis. In addition, although the symptoms can usually be treated or at least suppressed, the clinical response may be obscured by side effects of the therapy or complications of the HIV infection.

PNEUMONIA

Pneumonia is the most common secondary infection in HIV-infected patients infected, and *Pneumocystis carinii* is the most common etiologic agent, tending to proliferate when the CD4 count falls below 200/mm^3. Symptoms of *P carinii* pneumonia (PCP) have a gradual onset and include fever, nonproductive cough, weight loss, and dyspnea on exertion. Although measuring the diffusing capacity of the lung for carbon monoxide (DLCO), chest films, and gallium scans can be helpful, the diagnosis is established by the presence of cysts on a silver stain of induced sputum or bronchoalveolar lavage.

The recommended treatment for PCP is either trimethoprim/sulfamethoxazole or pentamidine (which appear to be equally effective) for 14 to 21 days. Steroid therapy has been recommended for selected patients, but the side effects are not yet well understood, and steroids may actually increase the rate of complicating infections. Prophylaxis is recommended for any HIV-positive patient with a CD4 count under 200 or a history of PCP infection. Aerosol pentamidine is frequently used, but its asymmetric distribution in the lungs and a lack of systemic absorption prevent it from fighting PCP infection in the upper lobe of the lung or elsewhere. The recommended prophylactic agent is trimethoprim/sulfamethoxazole, which has been found to be effective at very low doses. One double-strength tablet daily, or even as infrequently as three times weekly, is generally sufficient.

Bacterial pneumonia, which is being recognized more often in this patient population, may be due to *Hemophilus influenzae*, *Streptococcus pneumoniae*, *Nocardia* species, *Staphylococcus aureus*, *Branhamella catarrhalis*, or other pathogens. In contrast to PCP, the symptoms of bacterial pneumonia have a sudden onset, and the cough is often productive. A well-defined infiltrate may be apparent on chest films. Diagnosis is by Gram's stain of sputum and culture of sputum and blood.

TUBERCULOSIS

The importance of the purified protein derivative (PPD) test in HIV-infected patients with upper respiratory tract symptoms cannot be overstated, given the resurgence of tuberculosis that has accompanied the AIDS epidemic. A baseline PPD test should be administered for any HIV-infected patient: even though these patients are typically believed to be anergic, there have been cases of extremely immunosuppressed patients with positive PPDs. If the results indicate that the patient is PPD-positive, isoniazid should be administered prophylactically. It is important to remember that, even in the setting of AIDS, tuberculosis is a very treatable disease.

SINUSITIS

From 10% to 50% of AIDS patients have serious bacterial sinusitis, which is very significant because it can lead to sinobronchial syndrome. The presence of

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unusual fungal pathogens or superinfection with resistant bacteria can cause additional complications. Computed tomography of the sinuses provides a quick and cost-efficient means of diagnosis. The recommended management is antibiotic therapy; patients who fail to respond may require endoscopic drainage of the sinuses.

SUSAN REHM, MD
Department of Infectious Disease
The Cleveland Clinic Foundation

SUGGESTED READING

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A MIMIC OF VASCULITIS

Among the many disorders that mimic vasculitis, atheromatous embolization (AE) syndrome is especially challenging to the diagnostician and produces significant patient morbidity and mortality. AE may appear in a variety of ways, and physicians in many different specialties see patients with it. Despite this, AE has received little attention in major medical textbooks and is frequently misdiagnosed or unrecognized by physicians.

PATHOPHYSIOLOGY

AE syndrome involves the noncardiac embolization of atheromatous plaques that contain red blood cells, fibrin, platelet aggregates, and cholesterol crystals, and is sometimes referred to as "cholesterol embolization." It is seen most often in patients with advanced atherosclerosis.

AE can occur spontaneously; however, three events are known to precipitate AE: abdominal surgery, anticoagulation, and arteriography. Abdominal surgery or any surgery involving manipulation of the aorta carries an obvious risk for AE in the presence of diffuse, severe atherosclerosis. Anticoagulation with heparin or coumadin apparently leads to AE by causing the release of atheromatous material from the aorta, although the precise mechanism is not known. Arteriography is the most common precipitator of AE; it is not uncommon for patients to undergo arteriography, then come back 3 to 4 weeks later with an elevated serum creatinine related to atheromatous emboli.

Interestingly, the incidence of AE is very low (approximately 0.7%) when coronary arteriography is done via the arm, whereas the incidence is much greater when done via the leg, no matter how flexible the guidewire.

DIAGNOSTIC CLUES

AE is a multisystem disorder involving the skin, kidneys, central nervous system, and gastrointestinal tract; it causes a host of constitutional symptoms such as fever, malaise, weight loss, and anorexia, simulating vasculitis or underlying malignancy.

Skin changes include livedo reticularis and purple or blue toes and are the most common manifestations of AE. Livedo reticularis is due to embolization to the dermal blood vessels. If these lesions are biopsied, typical cholesterol clefts are seen microscopically. Livedo reticularis may occur associated with various forms of vasculitis and may also be a normal finding in young, healthy women who have increased vascular reactivity.

Blue, cyanotic, or purple toes may be seen in AE. More often than not, physicians misdiagnose this as being due to emboli coming from the heart, whereas the emboli are often from a severely atherosclerotic aorta.

Livedo reticularis on the lateral aspect of the foot or on the heel, along with bluish or purplish toes, is highly suggestive of AE. The ability to demonstrate lesions on both feet isolates the atherosclerotic plaque to the area above the aortic bifurcation. If only one foot is affected, then the lesion can be in the iliac artery, femoral artery, superficial femoral artery, or other distal artery.

Ischemic-appearing ulcerations in unusual locations (ie, neither distally nor over bony prominences) are another feature of AE, but also may suggest arteriosclerosis obliterans with underlying trauma, leukocytoclastic vasculitis, or other vasculitides. Hollenhorst plaques or cholesterol deposits seen on fundus examination reinforce the diagnosis of AE in the presence of skin lesions.

AE frequently involves the kidneys and, when it does, can cause accelerated hypertension, ischemic atrophy of large segments of the kidney, and progressive renal failure, often leading to end-stage renal disease. Pathological evidence of AE in the kidneys has been found in as many as 77% of patients who underwent surgery for abdominal aortic aneurysm and subsequently died.