TABLE 101-6 PULMONARY COMPLICATIONS OF HIV INFECTION: DIFFERENTIAL DIAGNOSIS AND TREATMENT

CONDITION	CHARACTERISTICS	CHEST RADIOGRAPH	DIAGNOSIS	TREATMENT
Pneumocystis jirovecii pneumonia	Subacute onset, dry cough, dyspnea	Interstitial infiltrate most common	BAL or induced sputum for organism by stain	TMP-SMX, pentamidine or atovaquone or primaquine + clindamycin
Bacterial (*Pneumococcus*, *Haemophilus* most common)	Acute onset, productive cough, fever, chest pain	Lobar or localized infiltrate	Sputum Gram stain and culture, blood culture	Cefuroxime or alternative antibiotics
Tuberculosis	Chronic cough, weight loss, fever	Localized infiltrate, lymphadenopathy	Sputum acid-fast stain and mycobacterial culture	Isoniazid, rifampin, pyrazinamide, ethambutol
Kaposi's sarcoma	Asymptomatic or mild cough	Pulmonary nodules, pleural effusion	Open lung biopsy	Chemotherapy

BAL, Bronchoalveolar lavage; TMP-SMX, trimethoprim-sulfamethoxole.

therapy should follow existing guidelines for empirical treatment of pneumonia (see Chapter 92). Pursuit of diagnostic studies that can support a definitive diagnosis is important, particularly if the patient's CD4 count is near 200 cells/mm^3 or the features or clinical course of pneumonia are atypical.

As with acute bacterial pneumonia, active pulmonary tuberculosis may develop at a time when the CD4 count remains well above 200 cells/mm^3 (see Table 101-6). Chest radiographs in HIV-infected patients may show features of primary tuberculosis, including hilar adenopathy, lower or middle lobe infiltrates, miliary pattern, or pleural effusions, as well as classic patterns of reactivation. Extrapulmonary *Mycobacterium tuberculosis* infection also occurs with increased frequency in patients with advanced immunodeficiency. Both pulmonary and extrapulmonary tuberculosis respond promptly to the use of four antituberculosis drugs.

Pneumonia caused by PCP remains a common life-threatening infection in persons with AIDS. Patients frequently report a gradual onset of nonproductive cough, fever, and shortness of breath with exertion; a productive cough suggests another process. A substernal "catch" with inspiration is common and is suggestive of PCP. In contrast to the acute onset of PCP in other immunocompromised patients, AIDS patients with PCP may have pulmonary symptoms for weeks before consulting a physician. Arterial hypoxemia is typical and rapidly worsens with slight exertion. Oxygen desaturation with exercise, as measured by pulse oximetry, can suggest the diagnosis. The chest radiograph often shows a subtle interstitial pattern but may be entirely normal. The presence of pleural effusions suggests a cause other than PCP.

If PCP is suspected clinically, therapy should be started immediately; treatment for several days does not interfere with the ability to make a specific diagnosis. Confirmation of PCP is essential; delay in establishing a correct diagnosis of another treatable condition may be lethal. An induced sputum sample can sometimes confirm the diagnosis, but most patients require bronchoalveolar lavage, which is adequate to diagnose PCP in more than 95% of patients. Treatment with high-dose trimethoprim-sulfamethoxazole (TMP-SMX) for 3 weeks is effective therapy (see Table 101-6). Patients with PCP and arterial hypoxemia (oxygen tension 75 mm H_2O on breathing of room air) benefit from the administration of corticosteroids (40 mg of prednisone twice daily), with tapering of the drug over a 3-week period.

For a deeper discussion of these topics, please see Chapter 391, "Pulmonary Manifestations of Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome," in Goldman-Cecil Medicine, *25th Edition.*

Disseminated histoplasmosis and coccidioidomycosis occur with much greater frequency in persons with HIV infection. Either fungal infection can cause nodular infiltrates or a miliary pattern on chest radiography. Histoplasmosis usually involves bone marrow as well as skin, and examination of the bone marrow often shows the organism. Standard treatment of disseminated mycoses in AIDS patients is high-dose liposomal amphotericin. Because relapse is common, oral azole therapy (fluconazole for coccidioidomycosis, itraconazole for histoplasmosis) must be continued even after resolution of signs and symptoms. For patients treated for histoplasmosis, secondary prophylaxis may be discontinued after 1 year of therapy, provided that the CD4 count is at least 150 cells/mm^3, blood cultures are negative, and the HIV serum *Histoplasma* antigen titers are low. Patients treated for systemic, meningeal, or diffuse pulmonary coccidioidomycosis have a greater risk of relapse and probably should continue suppressive therapy indefinitely.

For a deeper discussion of these topics, please see Chapter 332, "Histoplasmosis," and Chapter 333, "Coccidioidomycosis," in Goldman-Cecil Medicine, *25th Edition.*

Cardiovascular Disease

As the population with HIV ages, cardiovascular disease is becoming an increasingly important cause of morbidity and mortality. Persons with HIV experience higher rates of cardiovascular disease. Even with suppressive ART, some increased risk remains, likely attributable in part to risk factors such as persistent residual immunologic activation, high rates of tobacco use, hyperlipidemia, metabolic syndrome, diabetes, or chronic kidney disease.

Pericarditis and Pericardial Effusions

Pericardial effusions are a well recognized complication of HIV disease and may be result from infections or malignancy (see Chapter 10). In many cases, no specific cause is identified and effusions resolve without specific treatment. In places where tuberculosis is endemic, constrictive pericarditis secondary to tuberculosis is an important consideration. In addition, pericarditis and effusions secondary to acute infections and both non-Hodgkin's lymphoma and Kaposi's sarcoma may occur.