

especially in view of the variety of HIV-related pulmonary conditions mimicking TB. Extrapulmonary TB is common among HIV-infected patients. In various series, extrapulmonary TB—alone or in association with pulmonary disease—has been documented in 40–60% of all cases in individuals co-infected with HIV. The most common forms are lymphatic, disseminated, pleural, and pericardial. Mycobacteremia and meningitis are also common, particularly in advanced HIV disease. The diagnosis of TB in HIV-infected patients may be complicated not only by the increased frequency of sputum-smear negativity (up to 40% in culture-proven pulmonary cases) but also by atypical radiographic findings, a lack of classic granuloma formation in the late stages, and a negative TST. The Xpert MTB/RIF assay is the preferred initial diagnostic option for pulmonary TB ensuring a sensitivity of 81% and a specificity of 98%, and therapy should be started on the basis of a positive result because treatment delays may be fatal. A negative Xpert MTB/RIF result, however, does not exclude a diagnosis of TB. Culture remains the gold standard. Recent assessment of a test based on the detection of mycobacterial lipoarabinomannan antigen in urine has shown favorable results in assisting with the detection of TB in HIV-positive people (see “Additional Diagnostic Procedures,” below).

The *immune reconstitution inflammatory syndrome (IRIS)* or *TB immune reconstitution disease* consists of exacerbations in systemic manifestations (lymphadenopathy, fever) or respiratory signs (worsening of pulmonary infiltrations, pleural effusion) as well as laboratory or radiographic manifestations of TB. This syndrome has been associated with the administration of ART and occurs in ~10% of HIV-infected TB patients. Usually developing 1–3 months after initiation of ART, IRIS is more common among patients with advanced immunosuppression and extrapulmonary TB. “Unmasking IRIS” may develop after the initiation of ART in patients with undiagnosed subclinical TB. The earlier ART is started and the lower the baseline CD4+ T-cell count, the greater the risk of IRIS. Death due to IRIS is relatively infrequent and occurs mainly among patients who have a high preexisting mortality risk. The presumed pathogenesis of IRIS consists of an immune response that is elicited by antigens released as bacilli are killed during effective