

Pulmonary Nontuberculous Mycobacterial Infection: Radiologic Manifestations¹

Jeremy J. Erasmus, MD

H. Page McAdams, MD

Michael A. Farrell, MB, BCh

Edward F. Patz, Jr, MD

LEARNING OBJECTIVES

After reading this article and taking the test, the reader will:

- Know the most important causes of nontuberculous mycobacterial (NTMB) infection in the lungs.
- Understand the epidemiology, diagnosis, and treatment of these infections.
- Be able to describe the radiologic manifestations of the classic form of pulmonary NTMB infection.
- Be able to describe the radiologic manifestations of the common nonclassic form of pulmonary NTMB infection.
- Be able to describe the radiologic manifestations of NTMB infection in patients with acquired immunodeficiency syndrome.

The nontuberculous mycobacteria (NTMB) are a group of bacteria that can infect the cervical lymph nodes, skin, soft tissues, and lung. Pulmonary NTMB disease is increasing in prevalence and is most commonly caused by *Mycobacterium avium-intracellulare* or *M kansasii*. Occasionally, *M xenopi*, *M fortuitum*, or *M chelonae* also causes pulmonary disease. Diagnosis of pulmonary NTMB infection is often difficult because isolation of the organism from sputum or bronchoalveolar lavage fluid can represent airway colonization. The radiologic manifestations of pulmonary NTMB infection are protean and include consolidation, cavitation, fibrosis, nodules, bronchiectasis, and adenopathy. Pulmonary NTMB infection has five distinct clinicoradiologic manifestations: (*a*) classic infection, (*b*) nonclassic infection, (*c*) nodules in asymptomatic patients, (*d*) infection in patients with achalasia, and (*e*) infection in immunocompromised patients. Although classic NTMB infection may be indistinguishable from active tuberculosis, it is usually more indolent. The radiologic features of nonclassic NTMB infection are characteristic: bronchiectasis and centrilobular nodules isolated to or most severe in the lingula and middle lobe. In patients with acquired immunodeficiency syndrome, mediastinal or hilar adenopathy is the most common radiographic finding. Knowledge of the full spectrum of clinical and radiologic features of pulmonary NTMB infection is important to facilitate diagnosis and treatment.

Abbreviations: AIDS = acquired immunodeficiency syndrome, HIV = human immunodeficiency virus, NTMB = nontuberculous mycobacteria

Index terms: Lung, diseases, 60.2031 • Lung, infection, 60.2031 • Mycobacteria, 60.2031

RadioGraphics 1999; 19:1487–1503

¹From the Department of Radiology, Duke University Medical Center, Erwin Rd, Durham, NC 27710. Presented as a scientific exhibit at the 1997 RSNA scientific assembly. Received February 11, 1999; revision requested March 16 and received June 7; accepted June 10. **Address reprint requests to** J.J.E.

©RSNA, 1999

See the commentary by Choplin.

Table 1
Runyon Classification of NTMB Species

Group	Name	Characteristics
I	Photochromogens	Colonies require 2–4 weeks to grow in culture and change from colorless to yellow on exposure to light; <i>M kansasii</i> is the most common pulmonary pathogen in this group
II	Scotochromogens	Colonies require 2–4 weeks to grow in culture and change from yellow to orange on exposure to light; these organisms (<i>M gordonaie</i> , <i>M szulgai</i>) are rare pulmonary pathogens
III	Nonphotochromogens	Colonies require 2–4 weeks to grow in culture, are beige or white, and do not change color on exposure to light; this group includes <i>M avium-intracellulare</i> (the most common pulmonary pathogen) and <i>M xenopi</i>
IV	The rapid growers	Colonies are visible after 3–5 days of culture and do not change color on exposure to light; these organisms (<i>M fortuitum</i> , <i>M cheloneae</i>) are uncommon pulmonary pathogens

■ INTRODUCTION

The nontuberculous mycobacteria (NTMB) are a group of ubiquitous, low-grade pathogens that typically infect the cervical lymph nodes, skin, soft tissues, and lung (1). Pulmonary NTMB infection is increasing in prevalence and is most commonly caused by *Mycobacterium avium-intracellulare* or *M kansasii* (2,3). *M xenopi*, *M fortuitum*, and *M cheloneae* are uncommon pulmonary pathogens (2,3).

The NTMB usually cause chronic, indolent pulmonary infection. The symptoms and the severity of infection depend on several factors, including the presence of underlying lung disease and the patient's immune status (3). Diagnosis of pulmonary NTMB infection is often difficult because isolation of the organism from sputum or bronchoalveolar lavage fluid may represent airway colonization, not infection (3,4).

The radiologic manifestations of pulmonary NTMB infection are protean and often subtle and can be indistinguishable from those of tuberculosis. Knowledge of the full spectrum of findings is important to facilitate diagnosis and treatment (3). In this article, the classification of NTMB species and the epidemiology, diagnosis, treatment, and clinical and radiologic manifestations of pulmonary NTMB infection are reviewed.

■ CLASSIFICATION OF NTMB SPECIES

Runyon (5) classified the NTMB into four groups according to rate of growth, pigment production, and morphologic features (Table 1) (1,3).

■ EPIDEMIOLOGY

Although there is variability in their geographic distribution, NTMB are found throughout the environment and have been isolated from water, soil, milk, fish, birds, and animals (1,3,6). Infection can be acquired by inhalation, ingestion, or direct inoculation. Human-to-human transmission is rare, and isolation of infected individuals is not required (7). Despite high rates of exposure to NTMB, there is a low rate of clinical infection (1,6). Most pulmonary infections occur in patients over 50 years of age who have underlying lung disease or an immunologic disorder (1,3,8). An increased risk of NTMB infection is also present in patients with rheumatoid arthritis, diabetes mellitus, alcoholism, or nonpulmonary malignancies (6). However, infection is not uncommon in patients without an obvious underlying disorder (9,10). In the past decade, *M avium-intracellulare* has become an important pathogen in patients with acquired immunodeficiency syndrome (AIDS) (11).

■ DIAGNOSIS

Diagnosis of pulmonary NTMB infection is often difficult. Routine screening for mycobacterial infection is usually performed only for *M tuberculosis*, and the results will be negative in

Table 2
Diagnostic Criteria for Pulmonary NTMB Infection

Diagnosis	Criteria
Definite infection	Isolation of NTMB from specimens obtained with autopsy, lung biopsy, or transbronchial biopsy Transbronchial biopsy specimen with granulomas or acid-fast bacilli (or both) in association with a culture of NTMB from respiratory secretions Four or more sputum cultures with heavy growth of NTMB Culture of <i>M kansasii</i> from bronchoscopy washings (this organism, unlike <i>M avium-intracellulare</i> , is not a common contaminant) NTMB organisms in bronchoscopy washings in association with a positive blood or marrow culture (AIDS patients only)
Probable infection	Bronchoscopy brushings with acid-fast bacilli at histochemical staining in association with a culture of NTMB from bronchoscopy lavage fluid plus radiographic findings suggestive of pulmonary NTMB infection Two or three sputum cultures of NTMB plus radiographic findings suggestive of pulmonary NTMB infection
Possible infection	One or more cultures of a respiratory secretion with growth of NTMB in association with other pulmonary disease
Colonization or contamination	One or more cultures of a respiratory secretion with growth of NTMB without findings of pulmonary disease at chest radiography

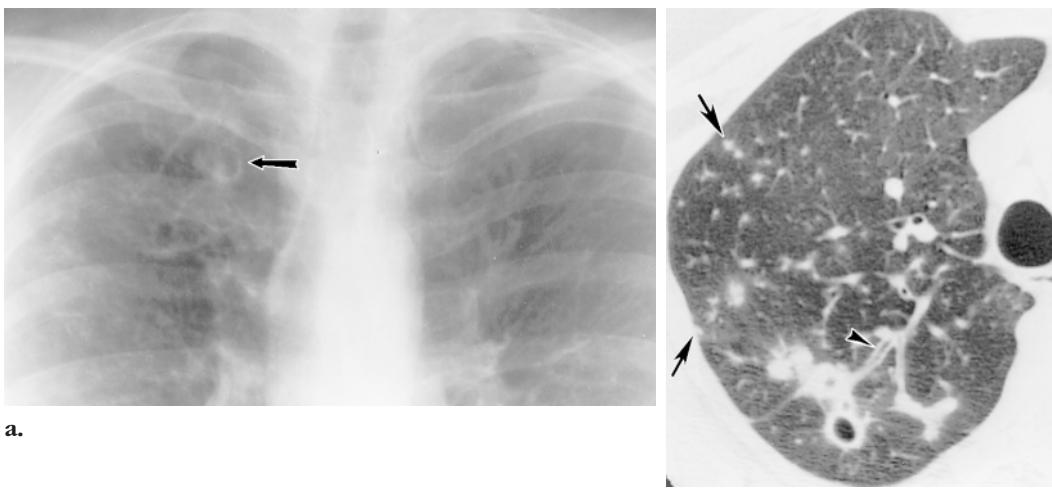
85% of patients with NTMB infection (3). Furthermore, a positive tuberculin skin test does not allow distinction between pulmonary NTMB and *M tuberculosis* infection (3). Specific intradermal skin testing for *M kansasii*, *M scrofulaceum*, *M avium-intracellulare*, and *M fortuitum* may be useful in distinguishing true infection from contamination or colonization, but clinical use is limited by cross-reaction between mycobacterial antigens (3).

Cultures of sputum or bronchoalveolar lavage fluid can be falsely positive in patients with chronic lung disease and airway colonization and falsely negative in patients with noncavitory pulmonary infection (3,4). Therefore, the diagnosis of pulmonary NTMB infection is usually made on the basis of (a) positive cultures of sputum or bronchoalveolar lavage fluid, (b) appropriate clinical and radiologic findings, and (c) a therapeutic response (Table 2) (3,4). Infection can be confirmed by isolation of NTMB from transbronchial or open lung biopsy specimens (4). Patients with AIDS are considered to have pulmonary NTMB infection if cultures of sputum or bronchoalveolar lavage fluid are positive, even if the chest radiograph is normal (3,12).

■ TREATMENT

Pulmonary NTMB infections are generally indolent with a natural history of slow progression (10). The clinical course and specific infecting organism determine the treatment. *M kansasii* infection, which usually responds well to antimycobacterial therapy, is typically treated with combination therapy of isoniazid, rifampin, and ethambutol (3). However, treatment of *M avium-intracellulare* infection is often difficult, and there are differing opinions regarding the treatment of infected patients (3,9). Because some patients may have a stable clinical course, some authors suggest that patients be observed for clinical and radiologic signs of progression before treatment is initiated (10). However, because untreated pulmonary infection can have significant morbidity and mortality, other authors advocate immediate treatment after the diagnosis of invasive infection is established (9). The best therapeutic results require treatment with a combination of five or six antimycobacterial drugs for 12–36 months.

Figure 1. Pulmonary *M avium-intracellulare* infection in a 42-year-old woman with a chronic cough. Sputum cultures were negative; the diagnosis was made with bronchoscopy and transbronchial biopsy. (a) Posteroanterior chest radiograph shows scattered, poorly defined linear and nodular areas of increased opacity with cavitation (arrow) in the right upper lobe. (b) Close-up computed tomographic (CT) scan of the right upper lobe shows peripheral centrilobular nodules (arrows), a thin-walled cavity, and bronchial wall thickening (arrowhead).



(3). Therapy with clarithromycin combined with isoniazid, rifampin, ethambutol, ethionamide, pyrazinamide, or streptomycin is the preferred long-term treatment regimen but is curative in only 60%-80% of patients (3,13,14). Surgical therapy is sometimes recommended for persistent, localized disease. Lobectomy can be effective for treating infections caused by *M avium-intracellulare* or *M xenopi* (15,16). Antimycobacterial therapy is required for 6-12 months after resection.

■ CLINICAL AND RADIOLOGIC MANIFESTATIONS

The clinical and radiologic manifestations of pulmonary NTMB infection can be divided into five groups: (a) classic infection, (b) nonclassic infection, (c) nodules in asymptomatic patients, (d) infection in patients with achalasia, and (e) infection in immunocompromised patients (1,2).

● Classic Infection

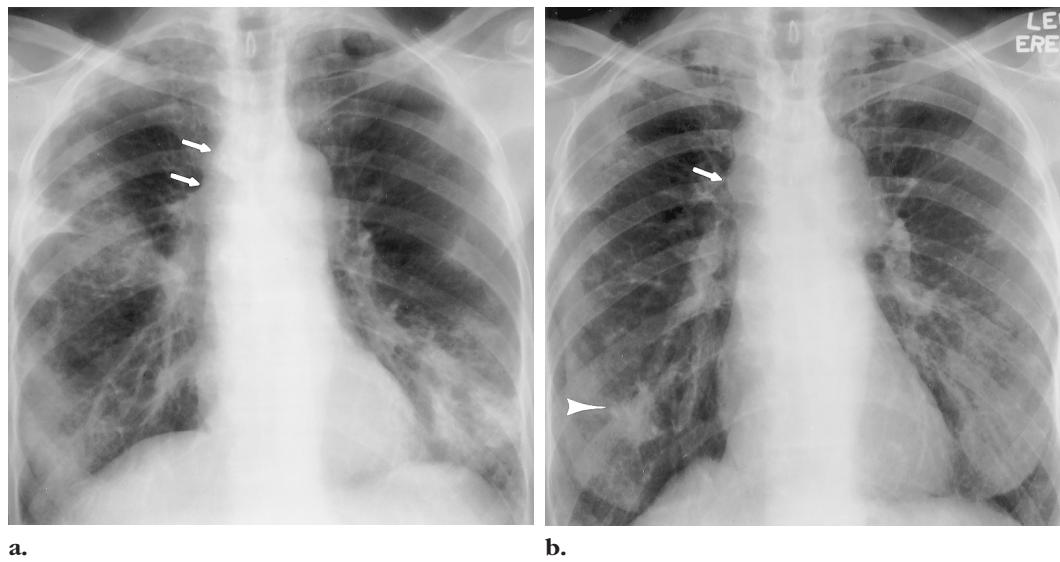
Classic infection is the most common form of pulmonary NTMB infection. Affected patients are typically elderly white men with underlying lung disease such as chronic obstructive pulmonary disease or pulmonary fibrosis (1,2,4). Classic NTMB infection may be indistinguishable from active tuberculosis; however, NTMB infection typically progresses more slowly than active tuberculosis (2).

Classic NTMB infection commonly manifests on chest radiographs with features similar to those of postprimary tuberculosis. Features that resemble those of healed primary tuberculosis, such as calcified pulmonary nodules and hilar nodes (Ranke complex), are less common (3,17). The most common findings are heterogeneous linear and nodular areas of increased opacity in the apical and posterior segments of the upper lobes with or without calcification (Fig 1) (2,3). Lower lobe disease is uncommon. The areas of increased opacity vary from subtle abnormalities that involve one segment to bilateral multi-segment disease (Figs 2, 3) (3). Although the

Figure 2. Pulmonary *M avium-intracellulare* infection in a 50-year-old woman with a chronic cough. **(a)** Posteroanterior chest radiograph shows heterogeneous areas of increased opacity in the right upper lobe with volume loss. The patient responded poorly to antimycobacterial therapy and underwent right upper lobe resection. **(b)** Posteroanterior chest radiograph obtained 3 years after resection shows consolidation in the upper aspect of the right lung and new areas of increased opacity in the left lung. The diagnosis of recurrent *M avium-intracellulare* infection was confirmed with transbronchial lung biopsy. The infection responded poorly to antimycobacterial therapy, and right pneumonectomy was performed. Persistent infection resulted in chronic empyema in the right pleural space. **(c)** Posteroanterior chest radiograph obtained 1 year later shows air in the right pleural space, a finding consistent with a bronchopleural fistula from chronic *M avium-intracellulare* infection. Note the scattered heterogeneous areas of increased opacity in the left lung.



Figure 3. Pulmonary *M avium-intracellulare* infection in a 72-year-old woman with a chronic cough. *M avium-intracellulare* was cultured from the sputum. (a) Posteroanterior chest radiograph shows scattered, bilateral, pulmonary areas of increased opacity with focal consolidation in the lingula. There is right paratracheal adenopathy (arrows). (b) Posteroanterior chest radiograph obtained 5 years later after long-term antituberculous drug therapy shows progressive volume loss in the upper lobes, increased paratracheal adenopathy (arrow), and improvement in the areas of increased opacity in the right upper lobe and lingula. New areas of increased opacity have developed in the middle lobe (arrowhead).



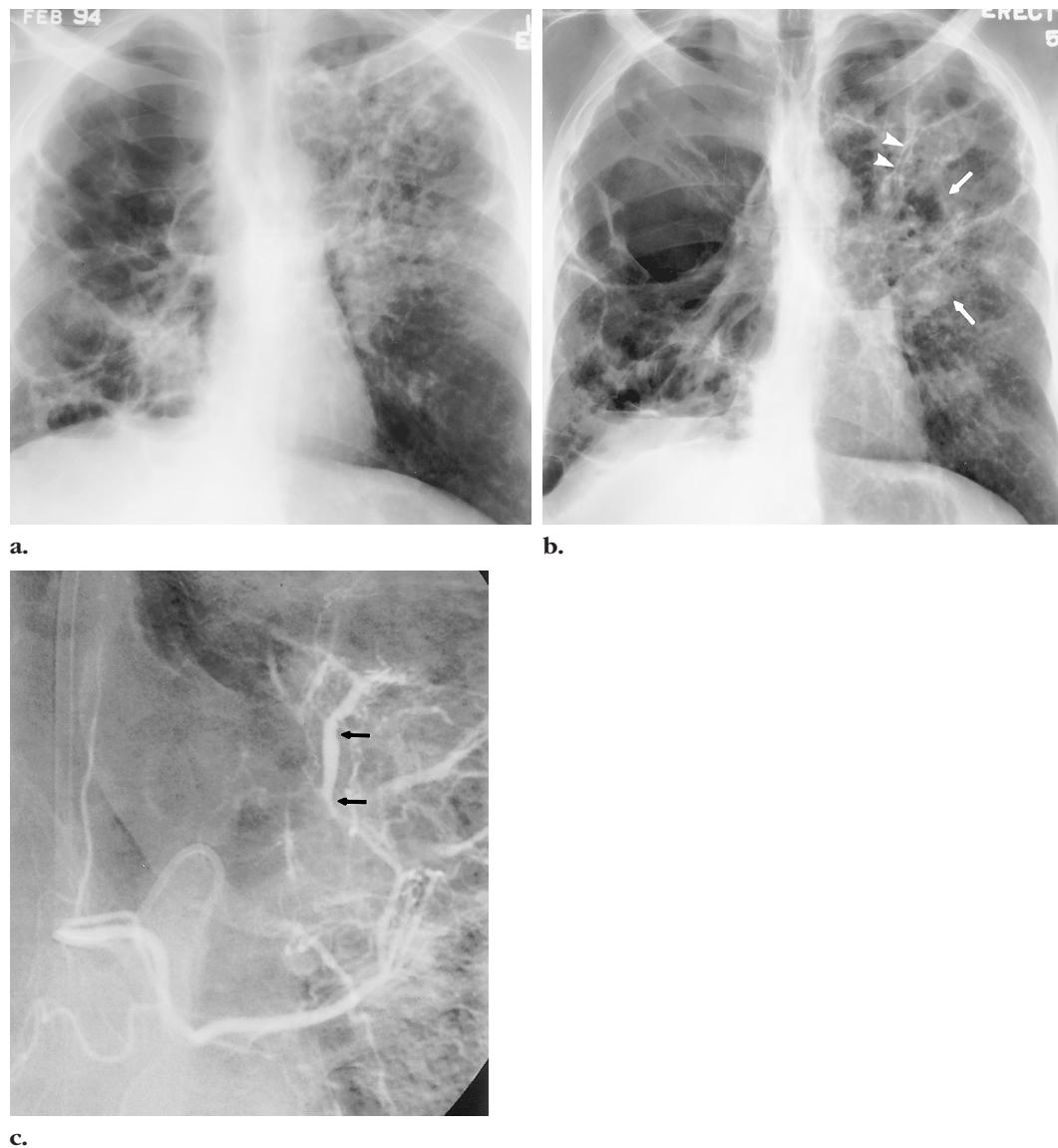
areas of increased opacity can remain unchanged for many years, more commonly they progress slowly (Fig 4) (2,17). Focal areas of homogeneous parenchymal opacification or masslike areas of increased opacity resembling primary lung carcinoma are occasionally seen in association with linear and nodular areas of increased opacity or as an isolated finding (Figs 5, 6) (3,17).

Cavitation is common and usually occurs in the upper lobes (Fig 7) (2,4). The cavities are usually small (mean diameter, 2.5 cm) and thin walled (Fig 8) (2,4). Cavitation facilitates endobronchial spread of disease, which manifests as unilateral or bilateral scattered nodular areas of increased opacity (Fig 9) (2). These nodular areas of increased opacity range from 5 to 15 mm in diameter and have a centrilobular distribution at CT (17,18).



Figure 5. Pulmonary *M avium-intracellulare* infection in a 58-year-old woman with a history of chronic cough and recent onset of shortness of breath and fatigue. Posteroanterior chest radiograph shows thin-walled cavities in the right upper lobe and a well-defined nodule in the left upper lobe (arrow). There are scattered heterogeneous and small nodular areas of increased opacity bilaterally.

Figure 4. Pulmonary *M avium-intracellulare* infection in a 43-year-old man with chronic obstructive lung disease, digital clubbing, and a chronic productive cough. Bronchial washings were positive for *M avium-intracellulare*. (a) Posteroanterior chest radiograph shows heterogeneous linear and nodular areas of increased opacity in the left lung. There is marked destruction of the right lung with architectural distortion and an air-fluid level in the superior segment of the right lower lobe. The patient was poorly compliant with antituberculous therapy and presented 20 months later with progressive weight loss and hemoptysis. (b) Posteroanterior chest radiograph shows progressive destruction of the upper lobes with a large bulla in the right upper lobe. Heterogeneous areas of increased opacity are present in the left upper lobe (arrows), and there is associated architectural distortion and traction bronchiectasis (arrowheads). (c) Left bronchial arteriogram shows a bronchial artery-pulmonary artery fistula (arrows). The bronchial arteries were embolized with polyvinyl alcohol foam powder (Ivalon; M-Pact, Eudora, Kan). The patient died after massive hemoptysis.



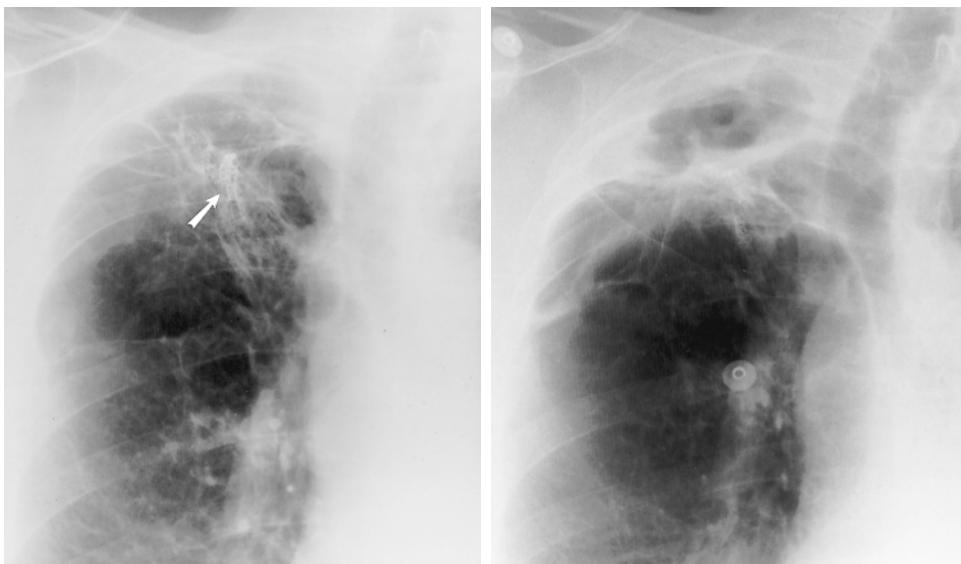


Figure 6. Pulmonary *M. avium-intracellulare* infection in a 50-year-old man with a history of resected non-small cell lung cancer and recent onset of weight loss and hemoptysis. (a) Posteroanterior chest radiograph obtained 4 years before admission shows sutures (arrow) and scarring in the right upper lobe from partial pulmonary resection. (b) Posteroanterior chest radiograph obtained at admission shows progressive volume loss, more areas of increased opacity around the sutures, and adjacent pleural thickening. *M. avium-intracellulare* was cultured from bronchial washings. No malignant cells were found, and the patient's condition improved with appropriate antimycobacterial therapy.

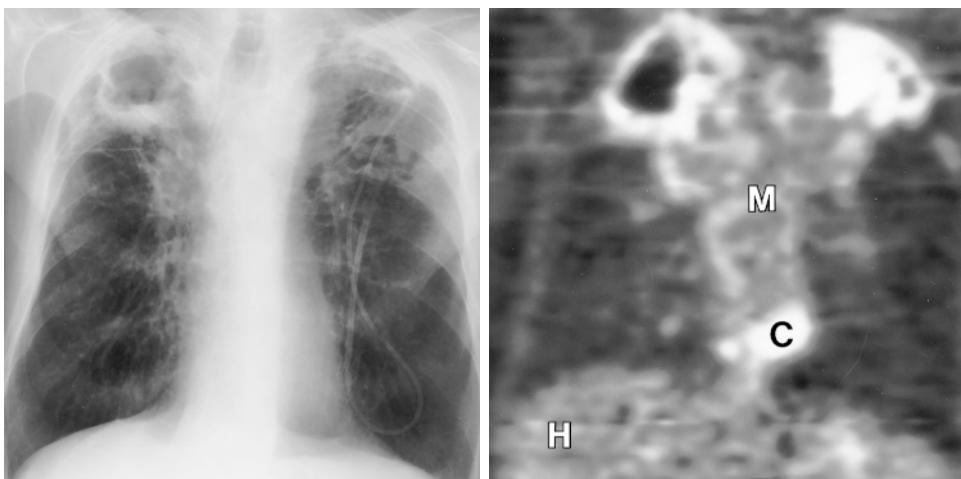
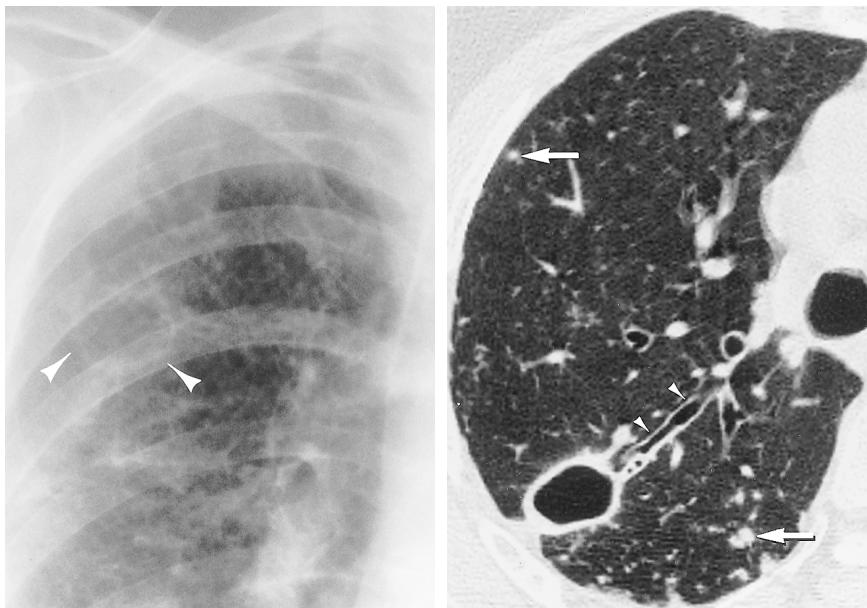


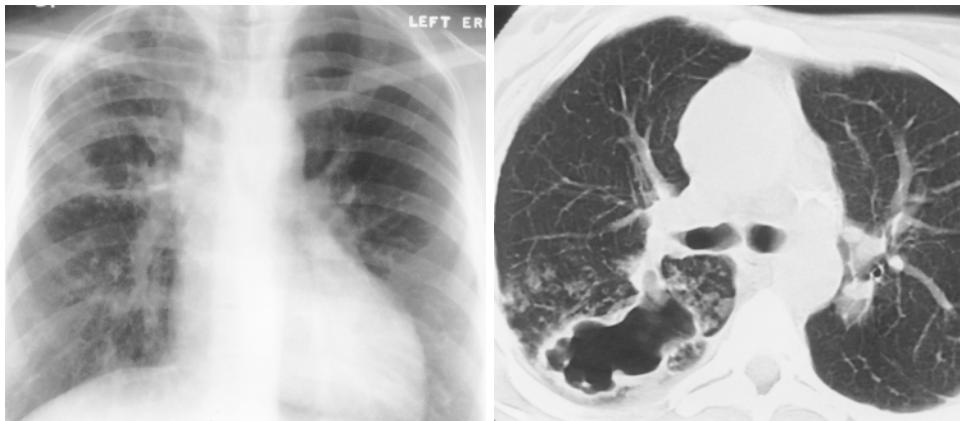
Figure 7. Pulmonary *M. avium-intracellulare* infection in a 64-year-old man with a history of chronic weight loss, cough, and occasional hemoptysis. (a) Posteroanterior chest radiograph shows scattered nodular areas of increased opacity and volume loss in both upper lobes. Note the cavity in the right upper lobe with an air-fluid level and biapical pleural thickening. (b) Coronal 2-[fluorine-18]fluoro-2-deoxy-D-glucose (FDG) positron emission tomographic scan shows marked increased FDG uptake in the upper lobes and in the wall of the right upper lobe cavity. Although increased FDG uptake is usually indicative of malignancy, false-positive studies can occur with NTMB infection. C = normal cardiac activity, H = hepatic activity, M = mediastinal activity.



a.

b.

Figure 8. Pulmonary *M avium-intracellulare* infection in a 60-year-old asymptomatic woman. (a) Close-up posteroanterior chest radiograph of the right lung shows scattered, small, heterogeneous areas of increased opacity and a thin-walled cavity in the right upper lobe (arrowheads). (b) Close-up thin-section CT scan of the right lung shows the thin-walled cavity in the right upper lobe, as well as a communicating bronchus (arrowheads) and small centrilobular nodules (arrows).



a.

b.

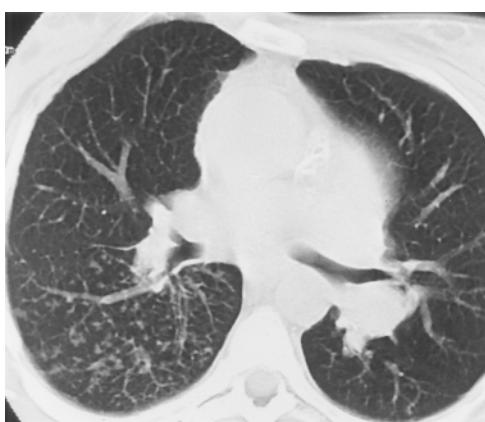
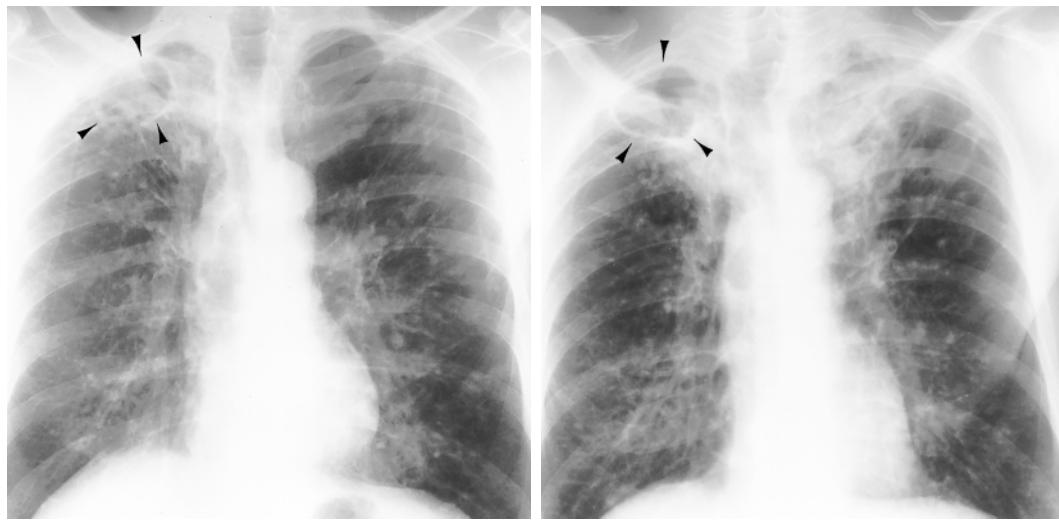


Figure 9. Pulmonary *M kansasii* infection in a 28-year-old woman with a history of surgically treated tricuspid atresia who presented with weight loss, fever, and a cough. (a) Posteroanterior chest radiograph shows heterogeneous areas of increased opacity in the right upper lobe. (b, c) CT scans show a large upper lobe cavity (b) and small, nodular, tree-in-bud areas of increased opacity (c) in the dependent portion of the right lung, which are due to endobronchial spread of infection.



a.

b.

c.

Figure 10. Chronic pulmonary *M avium-intracellulare* infection in a 51-year-old man treated with multiple antimycobacterial drugs, including ethambutol, pyrazinamide, isoniazid, rifampin, and clofazimine. (a) Posteroanterior chest radiograph shows heterogeneous areas of increased opacity and cavitation (arrowheads) in the right upper lobe. Small, poorly defined nodules in both lungs are suggestive of endobronchial spread of infection. (b) Posteroanterior chest radiograph obtained 4 years later shows volume loss and persistent cavitation (arrowheads) in the right upper lobe. Consolidation is now present in the left upper lobe. (c) Posteroanterior chest radiograph obtained 2 years later shows cavitation in both upper lobes (arrowheads); progressive volume loss in the right upper lobe with adjacent apical pleural thickening; and scattered, small, well-defined, nodular areas of increased opacity in regions of prior endobronchial infection.

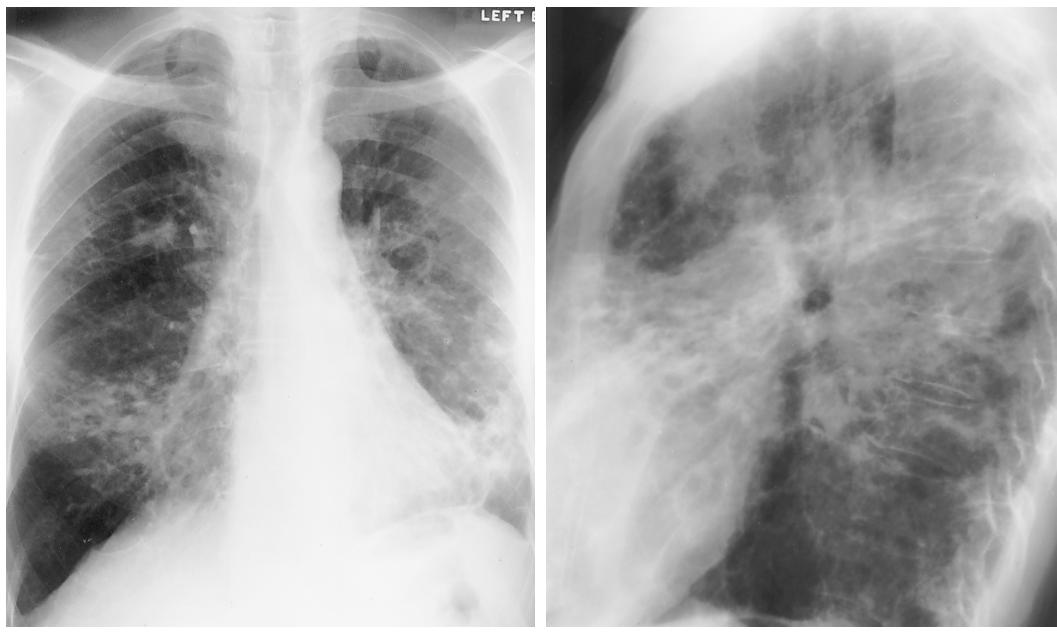
Miliary disease is rare in immunocompetent hosts. Adenopathy and pleural effusion are uncommon and almost never occur in isolation (1,3,4,17).

Progressive fibrosis with volume loss and traction bronchiectasis in the upper lobes occurs in one-third of patients (Fig 10) (1,3,17). Apical pleural thickening is also common (2).

● Nonclassic Infection

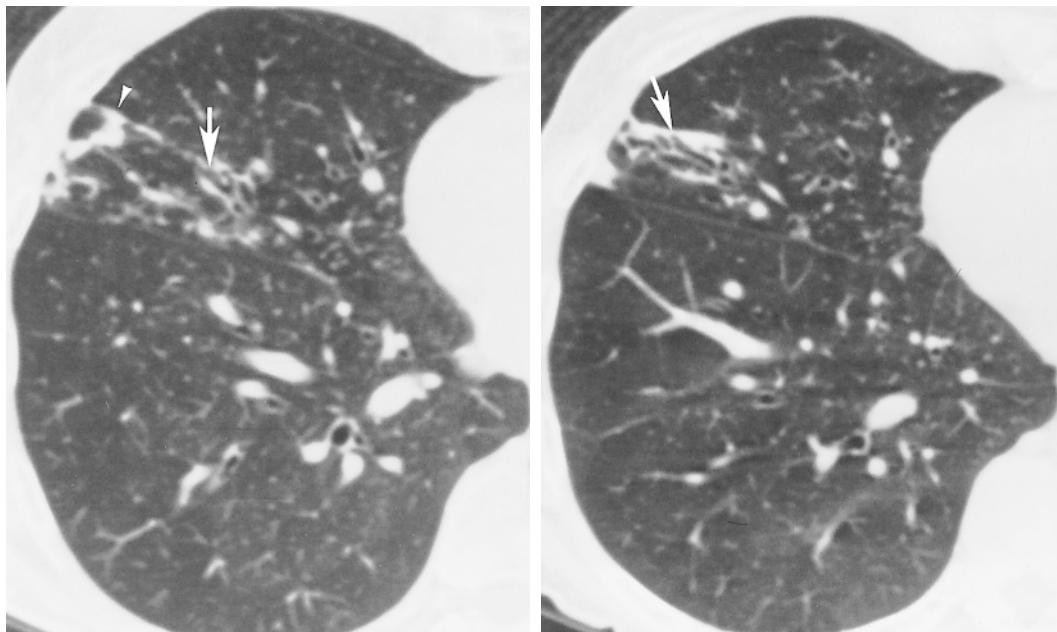
Nonclassic infection is the second most common form of pulmonary NTMB infection. Affected patients are typically elderly white women without underlying lung disease (2,18). These patients typically have a chronic cough; systemic symptoms are uncommon.

The radiologic findings of nonclassic infection are characteristic: mild to moderate cylindrical bronchiectasis and multiple 1–3-mm-diameter centrilobular nodules. Disease is usually isolated to or most severe in the lingula and middle lobe (Figs 11–13) (2,18). Although



a. b.

Figure 11. Pulmonary *M. avium-intracellulare* infection in a 42-year-old woman with a chronic cough. Posteroanterior (a) and lateral (b) chest radiographs show poorly defined, heterogeneous areas of increased opacity with associated tubular lucencies representing bronchiectasis in the lingula and middle lobe and scattered nodular areas of increased opacity in the right upper lobe.



a. b.

Figure 12. Pulmonary *M. avium-intracellulare* infection in a 67-year-old woman. The infection was proved with resection of the lingula. Close-up CT scans of the right lung show mild cylindrical bronchiectasis (arrow) and small centrilobular nodules in the middle lobe (arrowhead in a).

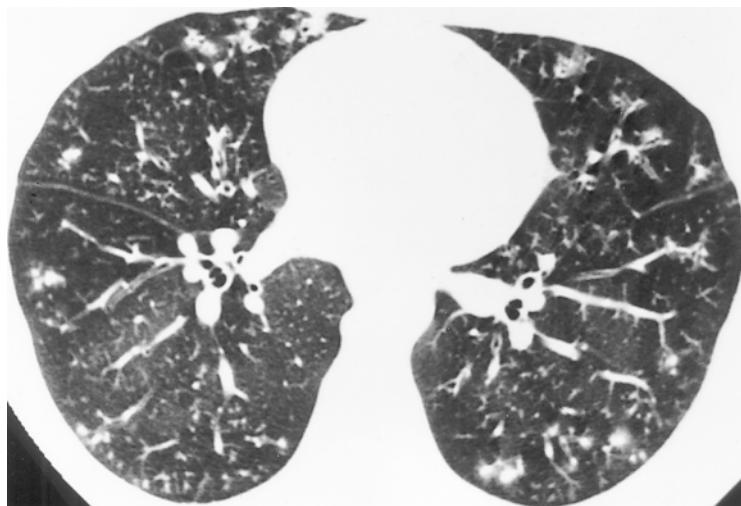


a.

b.

Figure 13. Pulmonary *M avium-intracellulare* infection in a 70-year-old white woman with a chronic cough, malaise, and weight loss. *M avium-intracellulare* was cultured from bronchial washings. Thin-section CT scans (1-mm collimation) show atelectasis and bronchiectasis bilaterally, more severe in the middle lobe and lingula. Note the small, peripheral, tree-in-bud areas of increased opacity (arrow in a) and the 1.5-cm-diameter nodule in the left lower lobe (arrow in b).

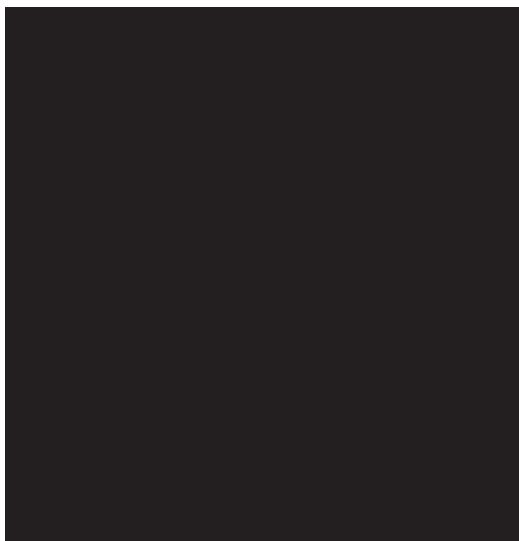
Figure 14. Pulmonary *M avium-intracellulare* infection in a 42-year-old woman with a chronic cough. Sputum cultures were negative. *M avium-intracellulare* infection was diagnosed with transbronchial lung biopsy. Thin-section CT scan (1-mm collimation) shows cylindrical bronchiectasis, bronchial wall thickening, and tree-in-bud areas of increased opacity.



these features may be suspected on chest radiographs, they are best seen with thin-collimation CT (Fig 14) (18–20). Cavitation, ground-glass areas of increased attenuation, volume loss, and adenopathy are uncommon findings of non-classic infection (2).

● Nodules in Asymptomatic Patients

Occasionally, NTMB infection results in solitary or multiple nodules, which are usually incidentally detected in asymptomatic patients (Fig 15) (1,2). The nodules are macroscopic granulomas and may represent the initial manifestation of pulmonary infection. Unlike in malignancy, multiple nodules are usually of similar size and clustered together (2).



a.



b.

Figure 16. Permission to reprint this figure electronically was denied by the publisher. See print version.

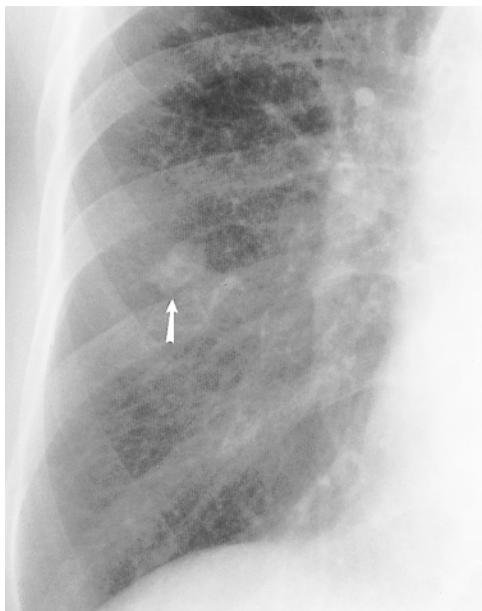


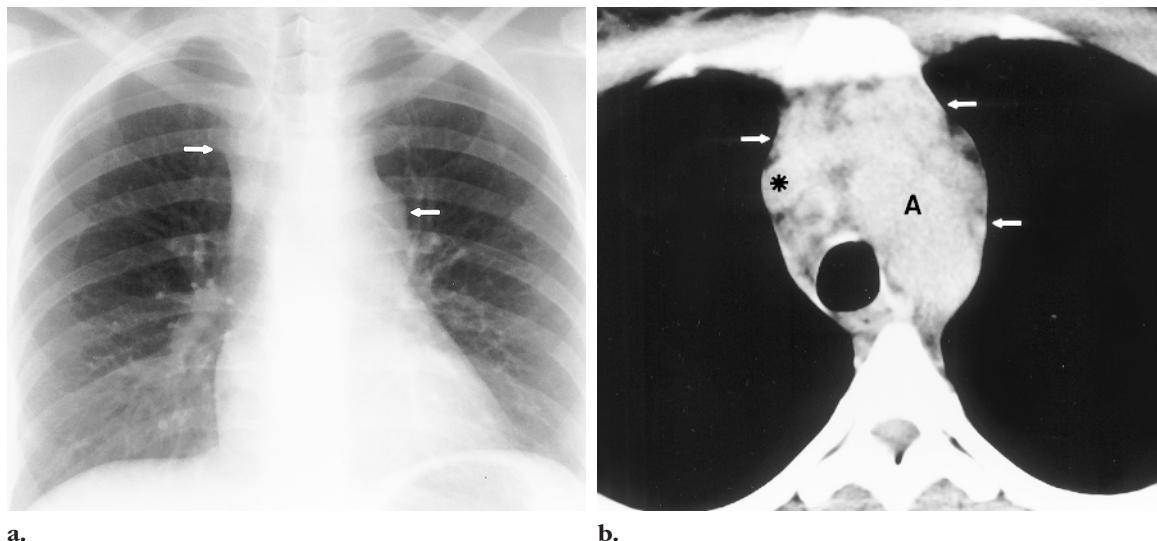
Figure 15. Pulmonary *M chelonae* infection in a 45-year-old asymptomatic woman. Close-up posteroanterior chest radiograph of the right lower lobe shows a well-defined, noncalcified, 1-cm-diameter nodule (arrow). *M chelonae* infection was diagnosed at resection.

● Infection in Patients with Achalasia

Patients with achalasia are predisposed to NTMB infection, usually with *M fortuitum-cheloneae*. Typically, infection results in large, bilateral, confluent areas of increased opacity that resemble aspiration pneumonia at radiography (Fig 16) (1).

● Infection in Immunocompromised Patients

Patients with AIDS.—NTMB infection is generally associated with marked immunosuppression and usually occurs late in the clinical course in patients with AIDS (CD4⁺ cell count less than 70/mm³ [$70 \times 10^6/L$]) (11). Unlike other human immunodeficiency virus (HIV)-associated infections, NTMB infection usually results from primary exposure, not reactivation of latent organisms (11). Fifteen percent to 24% of AIDS patients develop disseminated extrathoracic disease, although pulmonary infection is not common (2,22).



a.

b.

Figure 17. Disseminated *M avium-intracellulare* infection in a 33-year-old woman with AIDS and a CD4⁺ cell count of 55/mm³ ($55 \times 10^6/L$) who presented with weight loss and diarrhea. (a) Posteroanterior chest radiograph shows paratracheal and aortopulmonary window adenopathy (arrows). The lungs are normal. (b) Chest CT scan also shows mediastinal adenopathy (arrows). The lung parenchyma is normal. A = transverse aorta, * = superior vena cava.

The chest radiograph is often normal in patients with *M avium-intracellulare*-positive blood and sputum cultures (2). Mediastinal or hilar adenopathy is the most common finding (Figs 17, 18) (2,12). Small scattered alveolar and nodular areas of increased opacity, miliary nodules, and masslike lesions occur occasionally (Figs 18–21) (2,12).

Other Immunocompromised Patients.— Other immunocompromised patients with NTMB infection usually have lymphoproliferative disorders or are being treated with immunosuppressive drugs. Radiologic manifestations of NTMB infection are varied and include extensive mediastinal or hilar adenopathy, scattered heterogeneous and linear pulmonary areas of increased opacity, cavitation, and miliary nodules (2,3,17).

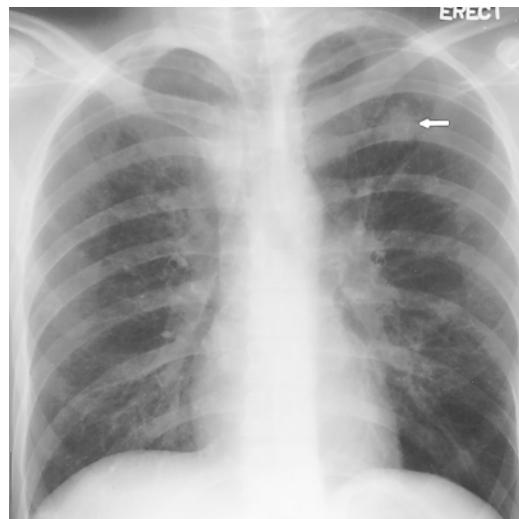
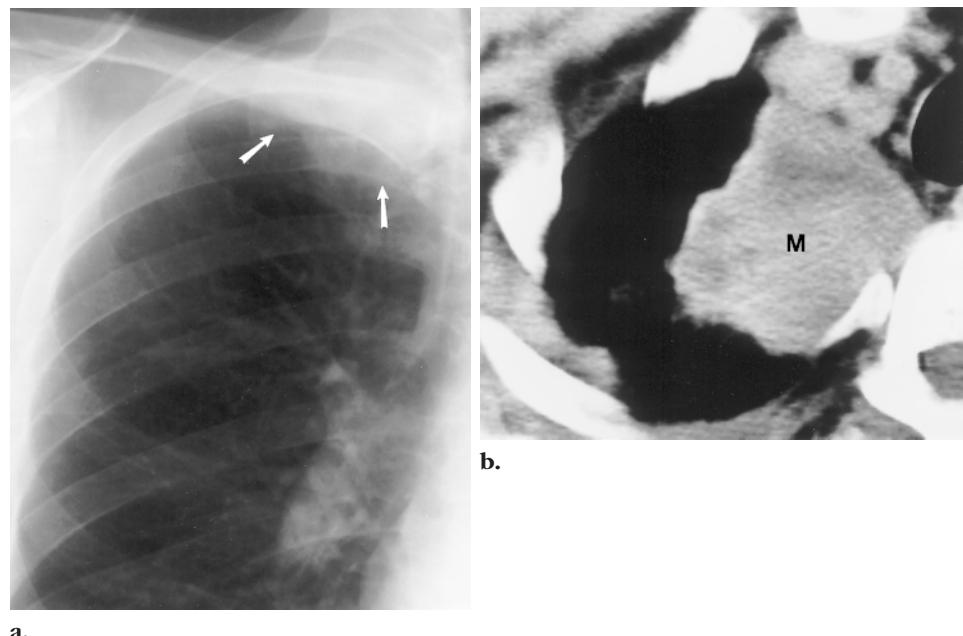


Figure 18. Pulmonary *M kansasii* infection in a 29-year-old cachectic man with AIDS who presented with dyspnea and a nonproductive cough. Posteroanterior chest radiograph shows paratracheal adenopathy and poorly defined scattered areas of increased opacity with a more focal, nodular area of increased opacity in the left upper lobe (arrow).

Figure 20. Pulmonary *M avium-intracellulare* infection in a 29-year-old man with AIDS. (a) Close-up posteroanterior chest radiograph of the upper right lung shows a mass in the apex of the lung (arrows) without hilar or paratracheal adenopathy. (b) CT scan shows a heterogeneous soft-tissue mass (*M*) in the right upper lobe abutting the mediastinum and chest wall. Biopsy revealed granulomatous inflammation, and a culture was positive for *M avium-intracellulare*.

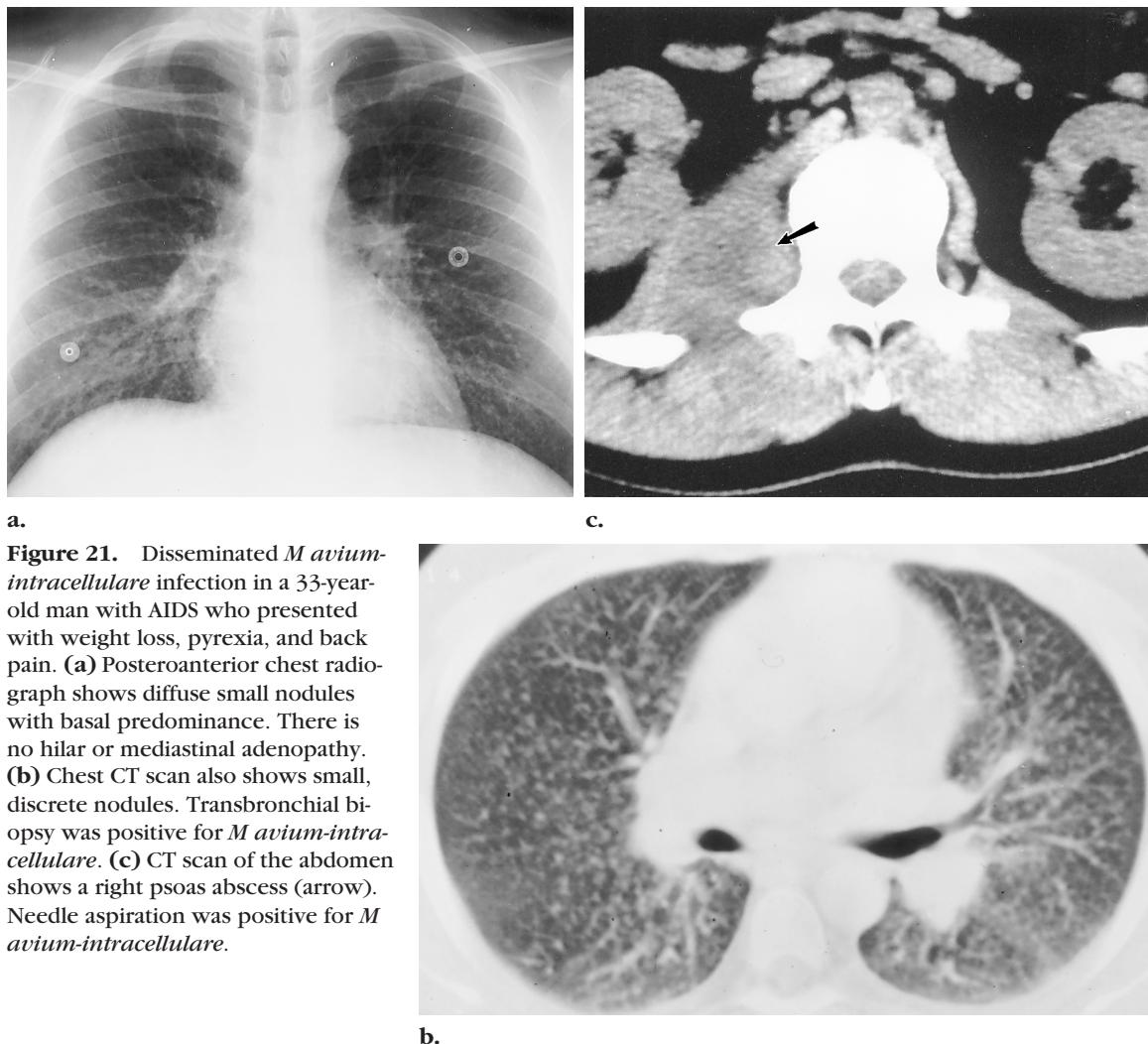


a.

b.



Figure 19. Disseminated *M avium-intracellulare* infection in a 35-year-old man with AIDS who presented with a cough and fever. The CD4⁺ cell count was 10/mm³ ($10 \times 10^6/L$). Sputum cultures were negative. *M avium-intracellulare* infection was diagnosed with bronchoscopy and transbronchial biopsy. Posteroanterior chest radiograph shows masslike areas of increased opacity and smaller, scattered, nodular areas of increased opacity in the upper lobes. There is no hilar or mediastinal adenopathy.



a.

c.

b.

Figure 21. Disseminated *M avium-intracellulare* infection in a 33-year-old man with AIDS who presented with weight loss, pyrexia, and back pain. (a) Posteroanterior chest radiograph shows diffuse small nodules with basal predominance. There is no hilar or mediastinal adenopathy. (b) Chest CT scan also shows small, discrete nodules. Transbronchial biopsy was positive for *M avium-intracellulare*. (c) CT scan of the abdomen shows a right psoas abscess (arrow). Needle aspiration was positive for *M avium-intracellulare*.

■ CONCLUSIONS

1. Pulmonary NTMB infection is increasing in prevalence. In many regions in the United States, it is more common than tuberculosis.
2. Diagnosis of pulmonary NTMB infection is difficult because the organism can colonize the airways. Definitive diagnosis may require biopsy.
3. Classic NTMB infection has clinical and radiologic features similar to those of tuberculosis but is usually more indolent than active tuberculosis.
4. The CT features of nonclassic NTMB infection are characteristic: bronchiectasis and centrilobular nodules isolated to or most severe in the lingula and middle lobe.
5. *M avium-intracellulare* infection is the most common NTMB infection in AIDS pa-

tients. Extrathoracic infection of blood, bone marrow, and lymph nodes is more common than intrathoracic disease. When the chest radiograph is abnormal, the most common finding is mediastinal or hilar adenopathy.

■ REFERENCES

1. Miller WT Jr, Miller WT. Pulmonary infections with atypical mycobacteria in the normal host. *Semin Roentgenol* 1993; 28:139-149.
2. Miller WT Jr. Spectrum of pulmonary nontuberculous mycobacterial infection. *Radiology* 1994; 191:343-350.
3. Woodring JH, Vandiviere HM. Pulmonary disease caused by nontuberculous mycobacteria. *J Thorac Imaging* 1990; 5:64-76.
4. Albelda SM, Kern JA, Marinelli DL, Miller WT. Expanding spectrum of pulmonary disease caused by nontuberculous mycobacteria. *Radiology* 1985; 157:289-296.
5. Runyon EH. Anonymous mycobacteria in pulmonary disease. *Med Clin North Am* 1959; 43: 273-290.

6. Fraser RG, Paré JAP, Paré PD, Fraser RS, Genereux GP. Diagnosis of diseases of the chest. 3rd ed. Philadelphia, Pa: Saunders, 1990; 933-941.
7. Buckner CB, Leithiser RE, Walker CW, Allison JW. The changing epidemiology of tuberculosis and other mycobacterial infections in the United States: implications for the radiologist. *AJR* 1991; 156:255-264.
8. O'Brien RJ. The epidemiology of nontuberculous mycobacterial disease. *Clin Chest Med* 1989; 10:407-418.
9. Iseman MD. *Mycobacterium avium* complex and the normal host. *N Engl J Med* 1989; 321: 896-898.
10. Rosenzweig DY. Pulmonary mycobacterial infections due to *Mycobacterium intracellulare-avium* complex. *Chest* 1979; 75:115-119.
11. Murray JF, Ellis K. Pulmonary complications of human immunodeficiency virus infection in adults. In: Potchen EJ, Granger RG, Greene R, eds. *Pulmonary radiology*. Philadelphia, Pa: Saunders, 1993; 285-307.
12. Marinelli DL, Albelda SM, Williams TM, Kern JA, Iozzo RV, Miller WT. Nontuberculous mycobacterial infection in AIDS: clinical, pathologic, and radiographic features. *Radiology* 1986; 160:77-82.
13. Dutt AK, Stead WW. Long-term results of medical treatment in *Mycobacterium intracellulare* infection. *Am J Med* 1979; 67:449-453.
14. Wallace RJ, Brown BA, Griffith DE, et al. Initial clarithromycin monotherapy for *Mycobacte-*
rium avium-intracellularare complex lung disease. *Am J Respir Crit Care Med* 1994; 149: 1335-1341.
15. Moran JF, Alexander LG, Staub EW, Young WG Jr, Sealy WC. Long-term results of pulmonary resection for atypical mycobacterial disease. *Ann Thorac Surg* 1983; 35:597-602.
16. Corpe RF. Surgical management of pulmonary disease due to *Mycobacterium avium-intracellularare*. *Rev Infect Dis* 1981; 3:1064-1067.
17. Woodring JH, Vandiviere HM, Melvin IG, Dillon ML. Roentgenographic features of pulmonary disease caused by atypical mycobacteria. *South Med J* 1987; 80:1488-1497.
18. Hartman T, Swensen SJ, Williams DE. *Mycobacterium avium-intracellulare* complex: evaluation with CT. *Radiology* 1993; 187:23-26.
19. Swensen SJ, Hartman TE, Williams DE. Computed tomographic diagnosis of *Mycobacterium avium-intracellulare* complex in patients with bronchiectasis. *Chest* 1994; 105: 49-52.
20. Moore EH. Atypical mycobacterial infection in the lung: CT appearance. *Radiology* 1993; 187:777-782.
21. Patz EF Jr, Swensen SJ, Erasmus J. Pulmonary manifestations of nontuberculous *Mycobacterium*. *Radiol Clin North Am* 1996; 33:719-729.
22. MacDonnell KB, Glassroth J. *Mycobacterium avium*-complex and other non-tuberculous mycobacteria in patients with HIV infection. *Semin Respir Infect* 1989; 4:123-132.