Tuberculosis has shown a resurgence in nonendemic populations in recent years, a phenomenon that has been attributed to factors such as increased migration and the human immunodeficiency virus epidemic. Although the thorax is most frequently involved, tuberculosis may involve any of a number of organ systems (eg, the respiratory, cardiac, central nervous, musculoskeletal, gastrointestinal, and genitourinary systems), and timely diagnosis of the disease is paramount, since delayed treatment is associated with severe morbidity. Unfortunately, a history of infection with or exposure to tuberculosis may or may not be present, and evidence of active tuberculosis is present in less than 50% of cases. A negative tuberculin skin test does not in itself exclude infection. Furthermore, the clinical and radiologic features of tuberculosis may mimic those of many other diseases. Therefore, although in many cases biopsy or culture specimens are required to make the definitive diagnosis, it is imperative that radiologists and clinicians understand the typical distribution, patterns, and imaging manifestations of tuberculosis.
Introduction
The prevalence of tuberculosis has continued to decline in the United States over the past few years. However, the smallest annual decrease in the past 10 years occurred in 2003, with the reported prevalence of tuberculosis actually rising in some states and in certain populations (1). There has also been an increase in global prevalence, particularly in immunocompromised patients, with a rate of increase of approximately 1.1% per year (2). This increase has been seen not only in Africa and Asia, but also in Europe. For example, in the United Kingdom, there has been an increase in the prevalence of tuberculosis over the past 20 years, particularly in London and in immigrant populations (3,4). In addition, drug-resistant strains of tuberculosis have emerged. Early diagnosis promotes effective treatment and is, therefore, essential.

Tuberculosis is usually confined clinically to the respiratory system. However, it can affect any organ system, particularly in immunocompromised individuals. In this article, we review multidrug-resistant (MDR) tuberculosis, tuberculosis in immunocompromised patients, appropriate safety precautions for health care workers who are caring for tuberculosis patients, and the use of nuclear imaging in this setting. In addition, we discuss and illustrate the clinical and radiologic features of tuberculosis involving the lungs, heart, central nervous system (CNS), head and neck, musculoskeletal system, and abdomen.

Multidrug-resistant Tuberculosis
The imaging appearances of MDR tuberculosis are the same as those of non-MDR tuberculosis. Moreover, MDR tuberculosis is no more infective than normal tuberculosis (3). However, it is a more serious infection, requiring prolonged administration of more toxic second-line drugs associated with higher morbidity and mortality rates. Patients also remain infectious for a longer period once treatment has been started, with associated higher risk to others (5).

Tuberculosis in Immunocompromised Patients
Immunocompromised patients have a significantly higher prevalence of tuberculosis than does the general population (6,7) and are also more likely to be infected with MDR tuberculosis (8). In addition, the pattern of disease is different in immunocompromised patients, who have a higher prevalence of extrapulmonary involvement. In one study, 38% of immunocompromised patients with tuberculosis had pulmonary involvement only, 30% had extrapulmonary involvement only, and 32% had both pulmonary and extrapulmonary involvement (9). Even when there is pulmonary involvement, a limited immune response can give rise to normal chest radiographic findings (10). Therefore, in immunocompromised patients, it is important to be aware of the extrapulmonary features of tuberculosis (discussed later).

Protection of the Health Care Worker
Tuberculosis is an airborne communicable disease caused by Mycobacterium tuberculosis. Health care workers are at a greater risk for infection than is the general population; therefore, their protection needs to be considered (11).

Patients should be treated as infectious if they have or are suspected of having pulmonary tuberculosis. These patients, particularly those with lung cavitation, aerosolize the organism through coughing, expelling 1–5-μm particles that can remain suspended in the air for several hours (12). Coughing hygiene helps reduce patient infectivity. Ideally, infectious patients should not be admitted to the hospital unless it is clinically required, and patients who are admitted should be kept in isolation in a single room (8), ideally with at least two air exchanges per hour (13). The use of negative-pressure rooms is recommended for patients who either have MDR tuberculosis or must be in the same ward as immunocompromised patients (5,11). Although patients with nonpulmonary tuberculosis are generally treated as noninfectious and nursed in general wards, procedures that expose tuberculous collections to air (eg, care of skin wounds or the draining of abscesses, pleural effusions, or peritoneal effusions) should prompt patient isolation (11,14).

A patient with non-MDR pulmonary tuberculosis is deemed to be noninfectious and can be moved to a general ward after 2 weeks of treatment if he or she has improved clinical symptoms and three negative sputum samples, ideally collected on different days (5,12,15).

Any imaging of an infectious patient should ideally be carried out within the patient’s isolation room, and no specific protective measures are required for a health care worker carrying out a procedure that does not involve aerosolizing tuberculous collections, except when the patient is MDR positive (8). In such a case, or if there is a risk of aerosolizing tuberculous collections, a certified respirator should be worn by the health care worker (5,15). Because tuberculosis is infective only if airborne, the imaging equipment used can be cleaned with a standard germicide.
Departmental radiologic imaging should be scheduled for off-peak times, away from immunocompromised patients; an infectious patient should be fitted with a surgical mask and imaged in the shortest time possible, preferably in his or her own well-ventilated area, and in the best-ventilated room available (15,16). If the patient is MDR positive, it is probably worthwhile to fit him or her with a certified respirator. Negative-pressure waiting rooms and imaging rooms are probably not required unless the department is serving a population with a high prevalence of tuberculosis (15).

No protection is required for general radiology personnel unless they are performing procedures that may aerosolize a tuberculous collection, and no protection is needed for subsequent patients (8). However, all health care workers should have their tuberculosis status assessed prior to commencing employment, be aware of the symptoms of tuberculosis, and be told to seek advice if they have any concerns.

When the diagnosis of tuberculosis cannot be established with noninvasive techniques in patients who are suspected of having the disease, biopsy tissue or aspirate should be obtained for histologic analysis and culture (16). Imaging-guided biopsy techniques may be of assistance in this regard, but the radiologist should be informed of the possible diagnosis prior to undertaking any procedure. Specimens should be placed in an appropriate sterile container, either without preservative or in the correct medium for culture, and taken to the laboratory as quickly as possible. Formalin should not be used for culture specimens (5,11,16).

Nuclear Imaging

Gallium-67 citrate, Indium-111–labeled autologous leukocyte (white blood cell) scintigraphy, and fluorodeoxyglucose (FDG) positron emission tomography (PET) are useful in the setting of pyrexia of unknown origin, in which tuberculosis is implicated and no definitive source has been identified with other imaging techniques. In one study, Ga-67 scintigraphy had a sensitivity of 78% in identifying extrapulmonary tuberculosis but failed to help diagnose any cases of tuberculous meningitis (17). When the differential diagnosis includes skeletal infection, technetium-99m methylene diphosphonate bone scintigraphy can help localize focal sepsis and is about as sensitive as In-111 white blood cell scintigraphy (18). Ga-67 scintigraphy has a similar sensitivity for detecting bone lesions but is also capable of helping identify paraspinal abscesses and other extraspinal foci (19). Nuclear imaging techniques do not help distinguish between the different causes of sepsis, but they do help identify a focus of interest. Further imaging of the area in question, along with additional tissue sampling, can then be performed to aid in diagnosis (20).

FDG PET has several advantages over gallium and indium scanning (21): (a) it can be performed immediately, with no delay required between injection and scanning; (b) it generally results in a lower radiation dose due to the short half-life of FDG; (c) it demonstrates little normal organ uptake, except in the brain and heart; and (d) it provides a quantitative measurement of the absolute fraction of the injected dose reaching a tissue (21). Tuberculomas usually show uptake at FDG PET (22). Increased uptake is also seen with other granulomatous diseases and infections such as sarcoidosis, histoplasmosis, aspergillosis, and coccidioidomycosis. Therefore, in the setting of a known pulmonary lesion, FDG PET cannot be used to differentiate between neoplastic and nonneoplastic causes (23,24). This limitation is particularly pertinent in geographic areas where tuberculosis is endemic because, in approximately 2% of cases, malignancy and pulmonary tuberculomas may coexist (25). However, one study suggests that using carbon-11 choline PET can help differentiate between lung cancer and tuberculosis. The standardized uptake value is high in malignant masses and low in tuberculomas with C-11 choline PET but is high in both lesions with FDG PET (26).

Pulmonary Tuberculosis

Historically, pulmonary tuberculosis has been divided into primary and postprimary tuberculosis, with primary tuberculosis being considered a disease of childhood and postprimary tuberculosis a disease of adulthood. However, a reduction in the prevalence of tuberculosis in most Western countries (1,2) owing to effective treatment and public health measures has resulted in large unexposed adult populations who are at risk for contracting primary tuberculosis. As a result, primary tuberculosis now accounts for 23%–34% of all adult cases of tuberculosis (27). It can sometimes be difficult to differentiate between primary and postprimary tuberculosis both clinically and radiologically, since their features can overlap. However, confirming the diagnosis is more important than identifying the subtype because it allows initiation of correct clinical management.
Primary Tuberculosis

Primary tuberculosis is seen in patients not previously exposed to *M. tuberculosis*. It is most common in infants and children and has the highest prevalence in children under 5 years of age. The prevalence of primary tuberculosis in adults is increasing for the reasons outlined earlier; however, because primary tuberculosis is perceived to be a disease of childhood, it is often not suspected in adults, resulting in misdiagnosis (28). Chest radiography remains the mainstay of diagnosis; however, normal radiographic findings may be seen in up to 15% of patients with proved tuberculosis (29).

At radiology, primary tuberculosis manifests as four main entities: parenchymal disease, lymphadenopathy, miliary disease, and pleural effusion.

**Parenchymal Disease.**—Typically, parenchymal disease manifests as dense, homogeneous parenchymal consolidation in any lobe; however, predominance in the lower and middle lobes is suggestive of the disease, especially in adults. Its appearance is often indistinguishable from that of bacterial pneumonia; however, it can be differentiated from bacterial pneumonia on the basis of radiographic evidence of lymphadenopathy and the lack of response to conventional antibiotics (Fig 1).

In children under 2 years of age, lobar or segmental atelectasis is frequently seen, most often involving the anterior segment of an upper lobe or the medial segment of the middle lobe.

In approximately two-thirds of cases, the parenchymal focus resolves without sequelae at conventional radiography; however, this resolution can take up to 2 years. In the remaining cases, a radiologic scar persists that can calcify in up to 15% of cases, an entity that is known as a Ghon focus. Other calcified foci can also be seen, and persistent masslike opacities called tuberculomas are seen in approximately 9% of cases. Tuberculomas can cavitate and undergo calcification.

**Lymphadenopathy.**—Radiographic evidence of lymphadenopathy is seen in up to 96% of children and 43% of adults. Lymphadenopathy is typically unilateral and right sided, involving the hilum and right paratracheal region (Fig 2), although it is bilateral in about one-third of cases. Any nodes greater than 2 cm in diameter generally have a low-attenuation center secondary to necrosis at CT and are highly suggestive of active disease (30). Although lymphadenopathy is usually associated with other manifestations of tuberculosis, it can be the sole radiographic feature, a finding that is more common in infants and decreases in frequency with age. CT is more sensitive than chest radiography for assessing lymphadenopathy. The combination of calcified hilar nodes and a Ghon focus is called a Ranke complex and is suggestive of previous tuberculosis, although it can also result from histoplasmosis.
With treatment, there is usually slower resolution of the lymphadenopathy than of the parenchymal disease, and nodal calcification may develop. However, this calcification usually occurs 6 months or more after the initial infection.

Miliary Disease.—Clinically significant miliary disease affects between 1% and 7% of patients with all forms of tuberculosis. It is usually seen in the elderly, infants, and immunocompromised persons, manifesting within 6 months of initial exposure. Chest radiography is usually normal at the onset of symptoms, and hyperinflation may be the earliest feature. The classic radiographic findings of evenly distributed diffuse small 2–3-mm nodules, with a slight lower lobe predominance, are seen in 85% of cases (Fig 3). High-resolution CT is more sensitive than conventional radiography, with nodules seen in a random distribution. The nodules usually resolve within 2–6 months with treatment, without scarring or calcification; however, they may coalesce to form focal or diffuse consolidation.

Pleural Effusion.—A pleural effusion is seen in approximately one-fourth of patients with proved primary tuberculosis (29). The effusion is often the sole manifestation of tuberculosis and usually manifests 3–7 months after initial exposure. Pleural effusion is a very uncommon finding in infants. The effusion is usually unilateral, and complications (e.g., empyema formation, fistulization, bone erosion) are rare. Residual pleural thickening and calcification can result. Ultrasonography (US) often demonstrates a complex septated effusion.

Postprimary Tuberculosis

Postprimary tuberculosis remains primarily a disease of adolescence and adulthood. It occurs in patients previously sensitized to *M* tuberculosis. The term *postprimary tuberculosis* is generally used to refer to both reinfection with and reactivation of tuberculosis. Primary tuberculosis is usually self-limiting, whereas postprimary tuberculosis is progressive, with cavitation as its hallmark, resulting in hematogenous dissemination of the disease as well as disease spread throughout the lungs. Healing usually occurs with fibrosis and calcification.

The features of primary and postprimary tuberculosis may overlap; however, the distinguishing features of postprimary tuberculosis include a predilection for the upper lobes, the absence of lymphadenopathy, and cavitation.

At radiology, postprimary tuberculosis may manifest as parenchymal disease, airway involvement, and pleural extension.

Parenchymal Disease.—The earliest finding in parenchymal disease is patchy, poorly defined consolidation, particularly in the apical and posterior segments of the upper lobes (28). In the majority of cases, more than one pulmonary segment is involved, with bilateral disease seen in one-third to two-thirds of cases.

Cavitation, the hallmark of postprimary tuberculosis, affects about 50% of patients. The cavities typically have thick, irregular walls, which become smooth and thin with successful treatment. Cavities are usually multiple and occur
within areas of consolidation (Figs 4, 5). Resolution can result in emphysematous change or scarring. A minority of cavities demonstrate air-fluid levels; however, these findings can indicate the presence of superinfection.

If there is airway disease and, in particular, endobronchial spread of infection, tree-in-bud opacities may develop. These findings, which are usually visible in the lung periphery and resemble a branching tree with buds at the tips of the branches, are indicative of active tuberculosis (Fig 6).

Lymphadenopathy and pneumothoraces are seen in only about 5% of patients (27).

**Airway Involvement.**—Airway involvement is characterized by bronchial stenosis, leading to lobar collapse or hyperinflation, obstructive pneumonia, and mucoid impaction. Bronchial stenosis is seen in 10%–40% of patients with active tuberculosis (27) and is best demonstrated with CT, which usually shows long segment narrowing with irregular wall thickening, luminal obstruction, and extrinsic compression (30). It also results in tree-in-bud opacities and traction bronchiectasis, particularly of the upper lobes.

**Pleural Extension.**—Pleural effusions occur most often in primary tuberculosis but are seen in approximately 18% of patients with postprimary tuberculosis; they are usually small and associated with parenchymal disease. The effusions are typically septated and can remain stable in size for many years (Fig 7). The pleura may become thickened, which can result in a tuberculous empyema and an associated risk of developing a bronchopleural fistula. Residual pleural thickening and calcification may also occur.

**Cardiac Tuberculosis**

Tuberculosis involving the heart is rare, accounting for only 0.5% of cases of extrapulmonary tuberculosis (31). The main presenting finding is pericardial involvement (32), particularly in immunocompromised patients (33), whereas myocardial involvement is seen less often.

The primary sign of tuberculous pericarditis is pericardial thickening of more than 3 mm in
adults; this finding is seen in the majority of cases. CT demonstrates a thickened, irregular pericardium (Fig 8), frequently with associated mediastinal lymphadenopathy. Most patients have distention of the inferior vena cava to a diameter exceeding 3 cm; pleural effusions, typically bilateral; and deformities of the intraventricular septum. Less than 20% of patients have pericardial effusions or develop localized pericardial calcification (32).

Myocardial tuberculosis is usually associated with other foci of tuberculosis. These foci can be miliary lesions or tuberculomas. Myocardial tuberculosis tends to be asymptomatic and discovered incidentally at postmortem examination (31).

**Tuberculosis Involving the CNS**

Involvement of the CNS is seen in approximately 5% of patients with tuberculosis (34). However, its prevalence is greater in immunocompromised patients. CNS involvement is seen in up to 15% of cases of acquired immunodeficiency syndrome–related tuberculosis (34,35).

CNS tuberculosis usually results from hematogenous spread. However, it may result from direct rupture or extension of a subependymal or subpial focus (Rich focus) and may be located in the meninges, brain, or spinal cord. CNS tuberculosis can manifest in a variety of forms, including tuberculous meningitis, tuberculomas, tuberculous abscesses, tuberculous cerebritis, and miliary tuberculosis.

**Tuberculous Meningitis**

Tuberculous meningitis is the most common manifestation of CNS tuberculosis across all age groups (35), and early diagnosis is important to reduce morbidity and mortality. Tuberculous meningitis is usually due to hematogenous spread but can also be secondary to rupture of a Rich focus or direct extension from cerebrospinal fluid (CSF) infection (34–36).

The typical radiographic finding is abnormal meningeal enhancement, usually most pronounced in the basal cisterns (Fig 9), although some degree of involvement of the meninges...
within the sulci over the cerebral convexities and in the sylvian fissures is also seen in most cases (35,37). These findings are better seen at gadolinium-enhanced MR imaging than at CT. Appearances usually resolve relatively quickly with adequate treatment; however, radiographic resolution is delayed if there are thickened exudates (37). This appearance is nonspecific and has a wide differential diagnosis that includes meningitis from other infective agents; inflammatory diseases such as rheumatoid arthritis and sarcoidosis; and neoplastic causes, both primary and secondary.

The most common complication of tuberculous meningitis is communicating hydrocephalus, which can be seen at both MR imaging and CT and is caused by blockage of the basal cisterns by inflammatory exudates (37). Occasionally, noncommunicating hydrocephalus occurs due to the mass effect of a tuberculoma causing the obstruction of CSF flow.

Ischemic infarcts are also a common complication, being seen in 20%–40% of patients at CT (Fig 10), mostly within the basal ganglia or internal capsule regions and resulting from vascular compression and occlusion of small perforating vessels (34,36,37). Cranial nerve involvement occurs in 17%–70% of cases (35,37), most commonly affecting the second, third, fourth, and seventh cranial nerves.

### Parenchymal Tuberculosis

The most common CNS parenchymal lesion of tuberculosis is tuberculoma (tuberculous granuloma). This lesion may be solitary, multiple, or miliary and may be seen anywhere within the brain parenchyma, although it most commonly occurs within the frontal and parietal lobes. Tuberculomas can exist in conjunction with tuberculous meningitis, although this combination is not a consistent finding (35).

At CT, tuberculomas appear as round or lobulated masses with low or high attenuation (Fig 11). They demonstrate homogeneous or ring enhancement and have irregular walls of varying thickness. One-third of patients demonstrate the “target sign” (ie, central calcification or punctate enhancement with surrounding hypoattenuation and ring enhancement) (35). This finding is suggestive of, but not pathognomonic for, tuberculosis.
The MR imaging findings depend on whether the tuberculoma is caseating, and if so, whether the center is liquid or solid. It is thought that there is a progression from noncaseating to caseating and then from a solid to a liquid center (37). A noncaseating tuberculoma is hypointense relative to gray matter on T1-weighted images and hyperintense on T2-weighted images, with homogeneous gadolinium enhancement (Fig 12) (35,38).

Caseating tuberculomas with a solid center are isointense to hypointense on both T1- and T2-weighted MR images. They usually have a variable amount of surrounding edema, which is hyperintense on T2-weighted images (35). Caseating tuberculomas with a liquid center are hypointense on T1-weighted images and centrally hyperintense on T2-weighted images, with a peripheral hypointense rim on T2-weighted images that represents the capsule. Rim enhancement is usually seen at gadolinium-enhanced MR imaging. After treatment, tuberculomas can completely resolve; however, calcification is seen in up to one-fourth of cases and is identified most clearly at CT (35).

Miliary CNS tuberculosis is usually associated with tuberculous meningitis and appears at MR imaging as multiple tiny (<2-mm), hyperintense T2 foci that homogeneously enhance on contrast-enhanced T1-weighted images (Fig 13) (35).

Tuberculous abscesses are rarely seen and can be similar in appearance to liquid-centered caseating tuberculomas, although they tend to be larger and are more often multiloculated (35). At CT, these abscesses appear as hypoattenuating lesions with surrounding edema, mass effect, and ring enhancement. Tuberculous cerebritis occurs very rarely (34).

**Spinal Tuberculous Meningitis**

The MR imaging features of spinal tuberculous meningitis consist of CSF loculation and obliteration of the spinal subarachnoid space, with loss of outline of the spinal cord in the cervicothoracic spine and matting of the nerve roots in the lumbar region (37). Contrast-enhanced imaging reveals nodular, thick, linear intradural enhancement (Fig 14), which can completely fill the subarachnoid space, sometimes giving the appearance of a normal unenhanced MR image (39). Chronic spinal tuberculous meningitis may not enhance.

Syringomyelia can occur as a complication of arachnoiditis and is seen as spinal cord cavitation that typically demonstrates CSF signal intensity...
on both T1- and T2-weighted images and does not enhance after contrast material administration (37).

**Head and Neck Tuberculosis**

Tuberculosis in the head and neck represents about 15% of cases of extrapulmonary tuberculosis, with about 1.5% of all new cases manifesting in this way.

The most common location is within neck nodes, often manifesting as bilateral painless cervical lymphadenitis, also known as scrofula (Fig 15). The involved nodes are initially homogeneous but later undergo central necrosis, manifesting with central low attenuation at CT (Fig 16) and with central hypointensity and hyperintensity on T1- and T2-weighted MR images, respectively. Peripheral rim enhancement is seen at both modalities. These nodes may be difficult to differentiate from the necrotic nodes seen in metastatic head and neck squamous cell carcinomas. Nodal calcification often develops late in tuberculosis, which helps differentiate tuberculous nodes from malignancy; however, nodal calcification may also be seen in other malignancies such as metastatic thyroid cancer.

Extranodal tuberculous disease is rarely seen; the most commonly affected sites include (in descending order of frequency of occurrence) the larynx, temporal bone, and pharynx. The sinonasal cavity, thyroid gland, and skull base are very rarely involved.

At radiology, laryngeal tuberculosis manifests as soft-tissue thickening and infiltration of the preepiglottic and paraglottic spaces, without the presence of a focal mass. The laryngeal framework usually remains intact. The differential diagnosis consists mainly of other inflammatory laryngeal conditions.

The imaging findings at other head and neck sites are also rather nonspecific; inflammatory soft-tissue thickening is usually seen, but in advanced cases, neoplasm-like soft-tissue masses and bone erosions may be encountered.

**Musculoskeletal Tuberculosis**

The musculoskeletal system is involved in only 1%–3% of cases of tuberculosis (40). However, the resultant bone and joint destruction is the cause of severe morbidity and in cases of spinal involvement can cause severe neurologic sequelae. The disease affects patients of all ages—although it is rare in the 1st year of life—and most frequently affects the spinal column, pelvis, hip, and knee (41).

Diagnosis is often difficult, with an average delay of 16–19 months between the onset of symptoms and reported diagnosis (42). A history of infection with or exposure to tuberculosis may be present, and evidence of concurrent active intrathoracic tuberculosis is present in less than 50% of cases (40,43,44). In addition, although a positive tuberculin skin test helps support the diagnosis, a negative result should not be considered as evidence excluding it. Indeed, in one series, a false-negative rate of 14% was reported (45). Only histologic analysis and tissue culture
can help confirm the diagnosis, although this combination is not particularly sensitive. Therefore, although there are no pathognomonic radiologic features of musculoskeletal tuberculosis, knowledge of the features discussed in the following sections may help reduce the time to diagnosis and, hence, the associated morbidity.

**Tuberculous Spondylitis**

Approximately 50% of skeletal tuberculosis involves the spine (40,46). The lower thoracic and upper lumbar levels are most commonly affected (46–48).

The disease process is thought to result from hematogenous spread via the venous plexus of Batson. Infection usually begins in the anterior part of the vertebral body adjacent to the end plate. Subsequent demineralization of the end plate results in loss of definition of its dense margins on conventional radiographs. These end plate changes allow the spread of infection to the adjacent intervertebral disk. The loose internal structure of the disk allows the infection to disseminate more widely into additional spinal segments, resulting in the classic pattern of involvement of more than one vertebral body together with the intervening disks. It also allows spread into the paraspinal tissues, resulting in the formation of a paravertebral abscess known as a Pott abscess (Fig 17). In the lumbar spine, a psoas abscess may extend into the groin and thigh and may simply manifest as lateral bowing of the psoas shadow on conventional radiographs. An abscess that lies more anteriorly may result in anterior scoliosis of the vertebral bodies similar to that seen with lymphoma or abdominal aortic aneurysm. Calcification within the abscess is virtually diagnostic for tuberculosis (41). If left untreated, the infection eventually results in vertebral collapse and anterior wedging, leading to kyphosis and gibbus formation (Fig 18). With healing, ankylosis of the vertebral bodies occurs, with obliteration of the intervening disk space.

However, if there is anterior subligamentous involvement of the spine, infection can extend both superiorly and inferiorly, with sparing of the intervertebral disks. The involvement of a single vertebral body with sparing of the adjacent disks has also been described (42,48–50). Whatever the nature of the spread of infection, tuberculosis is characteristically associated with little or no reactive sclerosis or local periosteal reaction, a feature that helps distinguish it from pyogenic infections of the spine.

**Figures 17, 18.** (17) Pott abscess in a patient with tuberculous spondylitis. Radiograph of the thoracic spine demonstrates vertebra plana of D11 with an associated soft-tissue-density mass, the latter finding being consistent with a tuberculous (Pott) abscess. (18) Gibbus deformity secondary to tuberculous spondylitis. Sagittal T1-weighted (a) and T2-weighted (b) MR images show vertebral collapse with high signal intensity in the adjacent vertebral bodies. The vertebral collapse has resulted in a gibbus deformity and spinal cord compression.
MR imaging is the preferred imaging modality in the diagnosis and assessment of tuberculous spondylitis because of its sensitivity to soft-tissue abnormalities (Fig 19) and multiplanar capability (51,52). Scintigraphy adds very little information, with a 35% false-negative rate reported for isotope bone scans in patients with radiographic and clinical evidence of active disease (48). Reported false-negative rates with gallium are as high as 70% (45).

The differential diagnosis for tuberculous spondylitis includes metastatic disease, low-grade pyogenic infection (eg, brucellosis), fungal infection, and sarcoidosis, all of which have similar imaging characteristics. In the early stage of infection, imaging appearances are entirely nonspecific. However, there are some clinical and radiologic features (discussed later) that may help differentiate among these conditions.

Tuberculosis rarely affects the posterior vertebral elements (including the pedicles), in contrast to metastatic disease (41,48). However, the anterior scalloping seen with subligamentous spread of infection can also be seen with paravertebral lymphadenopathy, secondary to metastases or lymphoma. In differentiating tuberculosis from pyogenic infection, the clinical picture is as important as the radiologic features, with insidious onset of symptoms, a normal erythrocyte sedimentation rate, relevant respiratory symptoms, and slow disease progression favoring the diagnosis of tuberculosis. Radiologic features that favor the diagnosis include involvement of one or more segments; a delay in destruction of the intervertebral disks; a large, calcified paravertebral mass; and the absence of sclerosis. Sarcoidosis can produce multifocal lesions of vertebrae and disks, along with paraspinal masses that appear identical to tuberculosis.

**Tuberculous Osteomyelitis**

Isolated tuberculous osteomyelitis in the absence of associated tuberculous arthritis is relatively rare. When it does occur, however, the femur, tibia, and small bones of the hands and feet are most commonly affected (40,41). Typically, the metaphyses are involved, with radiographic features that include osteopenia and poorly defined lytic lesions with minimal surrounding sclerosis. In the immature skeleton, the spread of infection across the epiphyseal plate is a feature that helps distinguish tuberculosis from pyogenic infection.

Cystic tuberculosis is an unusual pattern of osteomyelitis that occurs more commonly in children than in adults. It is characterized by multiple small, well-defined oval lytic lesions of variable size that usually lack sclerotic margins. In children, the metaphyses of the long bones tend to be affected, whereas in adults, the axial skeleton (skull, shoulder, pelvis) is involved.

Tuberculous dactylitis, the painless involvement of the short tubular bones of the hands and feet, is also more common in children. At radiography, pronounced fusiform soft-tissue swelling with or without periostitis is the most common finding (Figs 20, 21). Periostitis indicates involvement of the underlying bone. Other changes sometimes seen in the underlying bone include coarsening of the trabecular pattern and acro-osteolysis with reactive sclerosis and joint involvement. Chronic untreated infection may lead to the formation of sinus tracts. The differential diagnosis for tuberculous dactylitis includes pyo-
genic or fungal infections, leukemia, sarcoidosis, hemoglobinopathies, hyperparathyroidism, and syphilis. Distinguishing tuberculous dactylitis from pyogenic osteomyelitis can be particularly difficult.

**Tuberculous Arthritis**

Tuberculous arthritis is characteristically a mono-arthritis affecting large weight-bearing joints (53). The imaging findings are similar to those of other infectious and inflammatory arthritides and are, therefore, nonspecific. These findings include osteopenia, synovitis and other soft-tissue swellings, marginal erosions, and varying degrees of cartilage destruction. Joint space narrowing occurs with highly variable rapidity but is usually delayed. As with any chronic infection, synovial involvement in the young results in hyperemia and epiphyseal overgrowth, most commonly in the knee (54). With progression of infection, bone sequestration and sinus formation can develop. The end result is usually fibrous ankylosis of the joint (Fig 22). Bone ankylosis occasionally occurs but is more commonly seen with pyogenic infections.

Again, the differential diagnosis includes pyogenic and fungal infections. Factors favoring a diagnosis of tuberculosis include insidious onset, minimal sclerosis (Figs 23, 24), the relative absence of periosteal reaction and bone proliferation, and relative preservation of joint space in the early stages (41).
Although MR imaging is more sensitive than conventional radiography in assessing the extent of bone and joint involvement, the findings are again nonspecific, particularly in early disease. This fact reinforces the importance of joint aspiration for microscopic analysis and culture.

**Abdominal Tuberculosis**

The abdomen is the most common focus of extrapulmonary tuberculosis, with the solid viscera being affected more often than the gastrointestinal tract (55). CT is the mainstay for investigating possible abdominal tuberculosis; however, knowledge of other imaging modalities, such as barium enema examination, is important to avoid misdiagnosis in cases in which tuberculosis is not initially suspected.

**Lymphadenopathy**

Abdominal lymphadenopathy is the most common manifestation of abdominal tuberculosis, being seen in 55%–66% of patients (56). The characteristic pattern is mesenteric and peripancreatic lymph node group enlargement, with multiple groups affected simultaneously. The majority (40%–60%) of patients with lymphadenitis have enlarged nodes with hypoattenuating centers and hyperattenuating enhancing rims at CT (Fig 25), findings that are characteristic of, but not pathognomonic for, caseous necrosis (56,57). These lymph node masses do not tend to cause biliary, gastrointestinal, or genitourinary obstruction, the presence of which would suggest an alternative diagnosis.

Other nodal patterns include conglomerate mixed-attenuation masses, enlarged homogeneous-attenuation nodes, and an increased number (more than three) of normal or mildly enlarged homogeneous nodes.

**Tuberculous Peritonitis**

Peritonitis is the most common clinical manifestation of abdominal tuberculosis, affecting one-third of all patients (58). Peritonitis is thought to originate primarily from hematogenous spread; however, it may be secondary to a ruptured lymph node or gastrointestinal deposit or to fallopian tube involvement. The condition is subdivided into three main types—wet, fibrotic, and
Dry Type Peritonitis.—Dry type peritonitis is seen in 10% of cases and is characterized by mesenteric thickening, fibrous adhesions, and caseous nodules. Its imaging manifestations are highly suggestive of, but not specific for, tuberculosis. Varying degrees of omental and mesenteric involvement are seen, and the omentum appears smudged, caked, or thickened with equal frequency. Mesenteric involvement ranges from mild to extensive. Peritoneal thickening with associated enhancement occurs; nodular implants with irregular thickening are uncommon and are more suggestive of peritoneal carcinomatosis (59).

The CT findings in tuberculous peritonitis are nonspecific, with disseminated peritoneal malignancy, nontuberculous peritonitis, and mesothelioma being noteworthy alternatives in the differential diagnosis.

Gastrointestinal Tuberculosis
Gastrointestinal tuberculosis is rare; when present, however, it almost always involves the ileocecal region (90% of cases), usually both the terminal ileum and the cecum (56). The most common CT finding is mural thickening, which is typically concentric but if eccentric tends to involve the medial cecal wall (60). Localized lymphadenopathy is usually seen (59).

Skip areas of concentric mural thickening with associated luminal narrowing with or without proximal dilatation can occur elsewhere in the small bowel, findings that strongly suggest tuberculosis in the presence of ileocecal involvement.

On barium studies, the earliest manifestation is spasm and hypermobility with edema of the valve. Thickening of an incompetent ileocecal valve has been described as being characteristic of tuberculosis (Fig 28). Shallow ulceration with elevated margins is seen at double-contrast enema examination. Advanced gastrointestinal tuberculosis characteristically appears as napkin ring stenoses, with a conical, shrunken cecum retracted out of the right iliac fossa by mesocolon retraction (56).

Involvement of the esophagus, stomach, and proximal small bowel is rare. Esophageal tuberculosis is usually due to extrinsic compression at the level of the carina from lymphadenopathy, although it can progress (56). Gastric tuberculosis usually affects the antrum and distal body and can simulate peptic ulcer disease; however, a sinus or fistula suggests tuberculosis. Proximal small bowel disease manifests as nonspecific mucosal fold thickening (Fig 29).

Dry—although there is considerable overlap in their CT appearances (56).

Wet Type Peritonitis.—Wet type peritonitis is the most common type of peritonitis (90% of cases) and features large amounts of free or loculated ascites, which, at CT, is usually slightly hyperattenuating (20–45 HU) relative to water due to its high protein and cellular content (Fig 26).

Fibrotic Type Peritonitis.—Fibrotic type peritonitis accounts for 60% of cases of peritonitis and is characterized by large omental and mesenteric cake-like masses with matting of bowel loops. At CT, it manifests as mottled low-attenuation masses with nodular soft-tissue thickening (Fig 27). Omental thickening and caking can also be seen at US.
Hepatosplenic Tuberculosis

Hepatosplenic involvement is common in patients with disseminated disease and is either micro-nodular-miliary or macronodular (56).

Miliary hepatic involvement is seen in patients with miliary pulmonary tuberculosis and is characterized by innumerable 0.5–2.0-mm nodules, which may not be detected at CT (Fig 30). The liver appears hyperechoic at US.

Macronodular hepatic tuberculosis is uncommon, and lesions are hypovascular at CT with irregular ill-defined margins and minimal central but definite peripheral contrast enhancement. At MR imaging, these lesions are hypointense with T1-weighted sequences and hyperintense with T2-weighted sequences. These imaging appearances are nonspecific and are similar to those of multiple metastases and abscesses. However, hepatic tuberculomas eventually tend to calcify, and the presence of calcified granulomas at CT in patients with known risk factors and in the absence of a known primary tumor should raise suspicion for tuberculosis (Fig 31) (59).

Adrenal Tuberculosis

In one autopsy study, the adrenal glands were the fifth most common site of extrapulmonary tuberculosis after the liver, spleen, kidneys, and bones (55). Adrenal tuberculosis is seen in up to 6% of patients with active tuberculosis. These patients

Figure 30. Miliary hepatic tuberculosis. CT scan shows multiple hypoattenuating lesions within the liver, findings that are consistent with miliary tuberculosis.

Figure 31. Hepatosplenic tuberculosis. CT scan shows multiple calcified granulomas within the liver, spleen, and periportal and peripancreatic lymph nodes. The right kidney is hydronephrotic, and a small calculus is seen within the collecting system.

Figure 32. Adrenal tuberculosis. CT scan demonstrates bilateral adrenal enlargement (arrows).

Figure 33. Renal tuberculosis. Intravenous urogram shows the characteristic appearance of caliceal erosions in the lower pole calices of the left kidney due to tuberculosis.
almost always present with bilateral adrenal involvement and an Addisonian type clinical picture (Fig 32).

The CT signs of active tuberculous adrenalitis are bilateral enlarged glands associated with large, hypoattenuating necrotic areas, with or without dotlike calcification (61).

Genitourinary Tuberculosis

Genitourinary tuberculosis is the most common clinical manifestation of extrapulmonary tuberculosis (62). Infection is spread either hematogenously to organs such as the prostate gland, seminal vesicles, and kidneys, or by direct extension (eg, to the bladder or epididymis).

Renal Tuberculosis.—Approximately 75% of renal tuberculous involvement is unilateral, the most common CT finding being renal calcification (50% of cases) (60). At intravenous urography, the earliest abnormality is a “moth-eaten” calix due to erosions (Fig 33), which progresses to papillary necrosis. Hydronephrosis tends to have irregular margins and filling defects owing to caseous debris. Renal parenchymal cavititation may be detected as irregular pools of contrast material. Dilated calices (hydrocalicosis) with related infundibular stricture at one or more sites within the collecting system may be seen. Characteristic calcifications in a lobar distribution are often seen in end-stage tuberculosis (tuberculous autonephrectomy).

Ureteric Tuberculosis.—Ureteric tuberculosis is characterized by a thickened ureteric wall and strictures, which occur in almost one-half of all cases of renal tuberculosis. Involvement is most common in the distal third of the ureter (60). Strictures have a predilection for points of normal anatomic narrowing: at the pelviureteric junction, across the pelvic brim, and at the vesicoureteric junction. Complications include hydronephrosis and hydroureter of varying degrees, usually due to obstruction at the vesicoureteric junction but possibly due to reflux (56).

Bladder Tuberculosis.—Bladder tuberculosis commonly manifests as reduced bladder volume with wall thickening, ulceration, and filling defects due to granulomatous material. In advanced disease, there is eventual scarring with long-term loss of cystic volume and a small, irregular, calcified bladder. Tuberculosis is a rare cause of a urethral stricture.

Genital Tuberculosis.—Genital tuberculosis almost always involves the fallopian tubes in women (94% of cases), usually causing bilateral salpingitis (63). Findings at hysterosalpingography are always abnormal, with obstruction and multiple constrictions of the fallopian tubes and endometrial adhesions or deformity of the cavity.

Male involvement is confined to the seminal vesicles or prostate gland, with occasional calcification (10% of cases) (Fig 34). Contrast-enhanced CT shows hypoattenuating prostatic lesions, which likely represent foci of caseous necrosis and inflammation. Nontuberculous pyogenic prostatic abscesses have a similar CT appearance. Spread is hematogenous and self-limiting.

The testes and epididymides are rarely involved. US shows focal or diffuse areas of decreased echogenicity; however, these findings are very nonspecific (Fig 35).
Conclusions
The clinical and radiologic features of tuberculosis may mimic those of many other diseases. A high degree of suspicion is required, especially in high-risk populations. Although in many cases biopsy or culture specimens are still needed to yield the definitive diagnosis, it is important for radiologists and clinicians alike to understand the spectrum of imaging features of tuberculosis to aid in making an early diagnosis.

References
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Typically, parenchymal disease manifests as dense, homogeneous parenchymal consolidation in any lobe; however, predominance in the lower and middle lobes is suggestive of the disease, especially in adults.

The earliest finding in parenchymal disease is patchy, poorly defined consolidation, particularly in the apical and posterior segments of the upper lobes (28). In the majority of cases, more than one pulmonary segment is involved, with bilateral disease seen in one-third to two-thirds of cases.

At CT, tuberculomas appear as round or lobulated masses with low or high attenuation (Fig 11). They demonstrate homogeneous or ring enhancement and have irregular walls of varying thickness. One-third of patients demonstrate the "target sign" (ie, central calcification or punctate enhancement with surrounding hypoattenuation and ring enhancement) (35). This finding is suggestive of, but not pathognomonic for, tuberculosis.

Tuberculosis rarely affects the posterior vertebral elements (including the pedicles), in contrast to metastatic disease (41,48). However, the anterior scalloping seen with subligamentous spread of infection can also be seen with paravertebral lymphadenopathy, secondary to metastases or lymphoma. In differentiating tuberculosis from pyogenic infection, the clinical picture is as important as the radiologic features.

Gastrointestinal tuberculosis is rare; when present, however, it almost always involves the ileocecal region (90% of cases), usually both the terminal ileum and the cecum (56). The most common CT finding is mural thickening, which is typically concentric but if eccentric tends to involve the medial cecal wall (60).