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CDR Contemporary Diagnostic Radiology

Volume 44 • Number 21
October 15, 2021

A BIWEEKLY REVIEW OF CLINICAL RADIOLOGIC PRACTICE

Imaging of Tuberculosis in Children

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This module meets the American Board of Radiology's (ABR's) criteria for self-assessment toward the purpose of fulfilling requirements in the ABR Maintenance of Certification (MOC) program.

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After participating in this educational activity, the radiologist should be better be able to identify the organs affected by tuberculosis and describe its radiologic manifestations.

Category: Pediatric Imaging
Modality: Multiple

Key Words: Tuberculosis, Tuberculosis in Children

Tuberculosis (TB) is highly prevalent worldwide, and it affects children and adults equally, causing significant morbidity and mortality. Imaging plays a crucial role in diagnosis and management. Knowledge of imaging modalities available, imaging manifestations of both pulmonary and extrapulmonary tuberculosis, is vital for the radiologist for timely diagnosis among children with TB.

TB remains one of the most common infectious diseases worldwide. The causative organism is an acid-fast bacillus, *Mycobacterium tuberculosis* (*M. tuberculosis*). Children younger than 15 years account for 11% of TB cases and 14% of TB-related deaths.[1] Usually, there is a delay in management in children due to difficulties in making accurate diagnosis. Only 35% of the pediatric cases are diagnosed accurately.[1] The usual mode of infection is airborne. The mode of infection can also be vertical from an infected mother to the newborn through the umbilical cord or inhalation or ingestion of infected amniotic fluid. The risk factors for contracting the disease include nutritional deficiency, immunodeficiency states including HIV infection, chronic renal disease, malignancy, or immunosuppressive medications.[2] Childhood TB is usually disseminated. Clinical manifestations differ significantly from adults. Main factors determining the nature of presentation are the patient's age, immune status and other comorbidities. Anatomic differences in children also modify the presentation of TB compared with adults.

Children younger than 1 year (including neonates) and immunocompromised children have the highest risk of progression to miliary TB and meningeal involvement. The most common sites of disease in children are intrathoracic and superficial lymph nodes. Nonspecific symptoms are fever of 2 to 3 weeks' duration, weight loss, dry cough, and cervical lymphadenopathy. Other clinical features are specific to organ involvement. Many cases of primary TB infection in children remain asymptomatic.[3]

Pathophysiology

Most cases of TB are caused by *M. tuberculosis*. Other less common species, which are considered a part of *M. tuberculosis* complex, include *M. africanum, M. bovis, and M. microti*. Human-to-human transmission is typical with the most common mode of spread being airborne. After inhalation

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of the bacteria, there can be either immediate clearance of the bacilli from the lung by the body defense, or the child can develop latent infection, active (primary) or reactive disease. The developing immune system in children is a contributing factor in disease manifestation and progression. Once the infection is established in the alveolar spaces, the defense system of the host comes to play, and if it fails to eliminate the infection, the bacilli proliferate inside alveolar macrophages. The infected macrophages attract other phagocytic cells by producing cytokines and chemokines, which eventually form the tubercle. Bacilli from the tubercle enter the local draining lymph nodes.

The lesion produced by the expansion of the tubercle into the lung parenchyma (Ghon's focus) and draining lymph node involvement is called Ghon's complex. The subsequent course of the disease depends upon the host cell-mediated immunity (CMI), which usually develops 2 to 6 weeks after infection. In case of good immunity, Ghon's complex calcifies and is known as Ranke's complex. Failure of an effective CMI leads to progressive destruction of the lung. Tumor necrosis factor-α, reactive oxygen, and nitrogen intermediates and the contents of cytotoxic cells may all contribute to the development of caseating necrosis, characteristic of a tuberculous lesion. Uncontrolled bacilli growth may lead to hematogenous spread of bacilli to other organs. The subsequent outcome depends upon the treatment given, immune status, and underlying comorbidities.[4,5]

Diagnosis

There is usually a delay or difficulty in diagnosing childhood TB. Challenges faced in the diagnosis of childhood TB are due to varied and vague clinical presentations because of the immature immune system, difficulty in collecting samples for diagnosis, and paucibacillary type of the disease making culture and isolation difficult. Young children are unable to cough sputum, making sputum-based diagnosis difficult. The gold standard test for the diagnosis remains the demonstration of acid-fast bacilli in the smear and culture; however, it has a lower and varied sensitivity.[6] There has been a growing role of molecular and nonmolecular assays for detection of TB including the γ-interferon release assay and nucleic acid amplification test using the polymerase chain reaction. However, their use remains a challenge in high burden areas and low-resource settings. Imaging plays a crucial role in diagnosis of childhood TB. Chest radiography is the single most important modality for diagnosing chest TB and is performed in all cases of suspected TB, irrespective of the site of the disease. The role of other imaging modalities depends on the site of involvement.[7]

Central Nervous System Tuberculosis

TB of the central nervous system (CNS) is uncommon in children, affecting less than 2% of all cases, with 50% of them being younger than 2 years.[8] It begins with small TB foci within the subpial or subependymal space around the brain and spinal cord, which ruptures into the subarachnoid space ultimately leading to meningitis, tubercular granuloma formation, vasculitis, and uncommonly encephalitis. There are two types of intracranial TB: meningeal disease (70%–80% cases) and parenchymal TB (tuberculoma, abscess).[9]

Contrast-enhanced CT (CECT) is the initial modality of choice; however, these can be normal in initial stages. MRI has been shown to be superior to CT. Apart from the routine MRI brain sequences (T1-weighted, T2-weighted, and FLAIR sequences), post-contrast T1-weighted sequence, diffusion-weighted imaging, magnetic resonance spectroscopy (MRS), and magnetic resonance

The continuing education activity in *Contemporary Diagnostic Radiology* is intended for radiologists.

Contemporary Diagnostic Radiology (ISSN 0149-9009) is published bi-weekly by Wolters Kluwer Health, Inc. at 14700 Citicorp Drive, Bldg 3, Hagerstown, MD 21742. **Customer Service: Phone (800) 638-3030; Fax (301) 223-2400; E-mail: customerservice@lww.com.** Visit our website at LWW.com. Publisher, Stella Bebos.

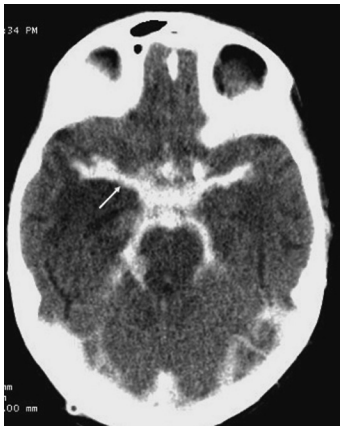

Figure 1. CECT head (axial section) in a 12-year-old girl with tubercular meningitis, showing thick nodular basal meningeal enhancement (*arrow*).

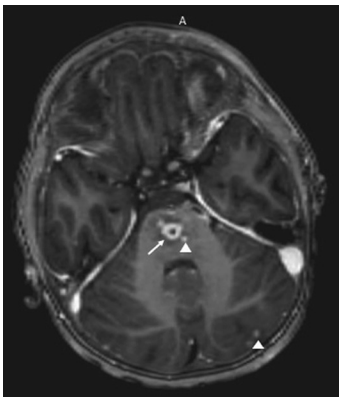

Figure 2. Axial section of post-contrast T1-weighted sequence depicting ring (*arrow*) and disc (*arrowhead*)-enhancing lesions suggestive of tuberculomas. Perilesional edema around the lesion in pons is observed.

angiography (in case of suspected vascular complications) form part of the imaging protocol in suspected TB.

Tubercular meningitis is the most common and severe form of CNS TB. The classical imaging features include hydrocephalus (with or without periventricular ooze), basal exudates, and diffuse basal leptomeningeal enhancement and thickening (Figure 1). Hydrocephalus is the most common abnormality detected, which is caused by obstruction of basal cerebrospinal fluid (CSF) space by the exudates. The exudates are hyperdense on CT and appear as dirty CSF on fluid-attenuated inversion recovery (FLAIR) and T1-weighted images, showing postcontrast enhancement. The leptomeningeal enhancement is linear or nodular with predilection for basal cisterns, Sylvian fissure, and rarely over the cerebral convexity, although if present in isolation is not pathognomonic for TB. The disease can involve adjacent brain parenchyma leading to meningoencephalitis. Vascular complications occur in 20% to 50% cases and include irregularity and absent flow in major vessels, penetrating artery ischemia/infarcts with most common locations being basal ganglia and thalamus. Cranial nerve involvement is seen in 17% to 40% cases, with affected cranial nerves being thickened and showing intense postcontrast enhancement.[9,10]

The most common parenchymal disease is tuberculoma, which can occur in isolation or in combination with meningitis. Tuberculomas vary in size and number and can occur anywhere, with the infratentorial site being more common in children. Imaging features depend on the stage of the lesion, with noncaseating granulomas appearing as isodense to slightly hypodense on unenhanced CT, T2 hyperintense, T1 hypointense on MRI, and showing homogeneous nodular enhancement. The caseating granuloma shows low signal intensity on both T1- and T2-weighted MRI sequences, with ring-like postcontrast enhancement and variable perilesional edema (Figure 2). MRS shows large lipid peak in majority of the cases. Old-healed granulomas appear as a calcified focus. Tubercular abscesses are rare in immunocompetent children and appear as single or multiple, irregular peripherally enhancing lesions with central T2 hyperintense/T1 hypointense component and central diffusion restriction.[9-11]

Head and Neck Tuberculosis

The most common manifestation of head and neck TB in children is cervical lymphadenopathy.[12] Tubercular cervical lymphadenitis is the most common form of extrapulmonary TB in children. Ultrasonography (US) typically is performed as initial investigation and shows bilateral enlarged cervical lymph nodes, with central anechoic areas representing necrosis. The lymph nodes are initially homogeneous, but later undergo conglomeration and necrosis, which appears as the central hypodense area on CT and T1 hypointense/T2 hyperintense foci on MRI images, with peripheral enhancement. Sometimes they can rupture leading to persistently discharging sinuses. Chronic, healed cervical lymph nodes can appear calcified.[13] Other nonosseous rarer sites of involvement include sinonasal cavity, eye, pharynx, and larynx.

Chest Tuberculosis

The most common site of tubercular involvement in children is the chest, with the lung being the most commonly affected region.[14] Chest TB comprises involvement of lung parenchyma, lymph nodes, pleura, and the chest wall. Traditionally, pulmonary TB was divided into primary disease, progressive primary disease, and postprimary disease, the distinction being based on the time-related course of the disease. However, there is a significant overlap in imaging features, and also the recent literature supports the fact that imaging manifestations are dependent on the host immune response. It is more important to distinguish active from inactive disease.[14]

Chest radiography is the primary imaging modality used for screening, initial diagnosis, and monitoring response to treatment. However, it lacks specificity and can be normal in up to 15% of patients with TB. Ultrasound is a radiation-free, cost-effective modality, which can be used to diagnose pleural disease. Also, mediastinal USG to detect mediastinal lymph nodes and even lung parenchyma evaluation in children has been found to be useful. CECT with high-resolution CT reconstruction remains the modality of choice in evaluation of chest TB, despite risk of radiation, which is of a primary concern especially in children. It is performed in cases of the equivocal or normal chest radiograph, severe or complicated infection, and immunocompromised host.[7] MRI has recently emerged as a radiation-free alternative to CT for imaging in the chest with multiple technical advances. It has a high sensitivity to detect lymph nodes and pleural and parenchymal disease.[15]

Lymphadenopathy is seen in up to 96% children and is the radiologic hallmark of primary TB with or without parenchymal abnormality. It can be unilateral or bilateral with the right side more commonly involved, particularly the right paratracheal and hilar region. Enlarged mediastinal and hilar lymph nodes are commonly seen in TB. The lymph nodes in active disease show conglomeration, heterogenous, or rim enhancement depending on the amount of necrosis. With

chronicity of disease or after treatment, there is a decrease in size and homogeneity of enhancement. Conglomeration also reduces, with lymph nodes appearing as a discrete focus. Calcification is more common (Figure 3) in healed than in active disease (41.7% vs. 28.4%).[16] On MRI, enlarged lymph nodes appear hyperintense on T2-weighted sequence/hypointense on T1-weighted sequence with peripheral or heterogenous enhancement and restricted diffusion.[17]

Parenchymal abnormalities include homogeneous lobar consolidation, multifocal consolidation, and diffuse nodules in both lungs. Typically, there is presence of a homogeneous lobular pattern of consolidation in any lobe, with predilection of middle and lower lobes in primary TB and apical and posterior segments of upper lobes in postprimary TB. More than one lobe is frequently involved. There can be internal necrosis. In children younger than 2 years, lobar or segmental atelectasis is frequently seen. The consolidation can resolve without any sequel; however, in some cases it can calcify with associated architectural distortion and fibrosis. Cavities are the hallmark of postprimary TB and are often multifocal, thick walled, frequently within the consolidation focus with or without air fluid levels[11,14] (Figure 4). Multiple clustered centrilobular nodules, especially in the "tree-in-bud" pattern, which signify endobronchial spread of disease, are often seen (Figure 5). There can be secondary air trapping or atelectasis after airway compromise by intrinsic or extrinsic compression.

Pleural effusion occurs mostly in the primary disease, and usually it is unilateral. If there are echoes, septations, loculations, it suggests empyema formation.[18] On CT, there is usually a higher attenuation of pleural fluid with pleural thickening and enhancement seen in cases of empyema. Underlying bone erosions can be seen. There is a risk of spontaneous bronchopleural fistula formation. In later stages, there is volume loss with development of pleural calcification.

In active disease, larger airways can be involved with imaging appearance ranging from smooth or irregular wall thickening, intraluminal mass, or peribronchial soft tissue with extrinsic luminal narrowing. Bronchiectasis is a common sequel of healed TB, with predilection for upper lobes. There is associated destruction of lung parenchyma and fibrosis.[19]

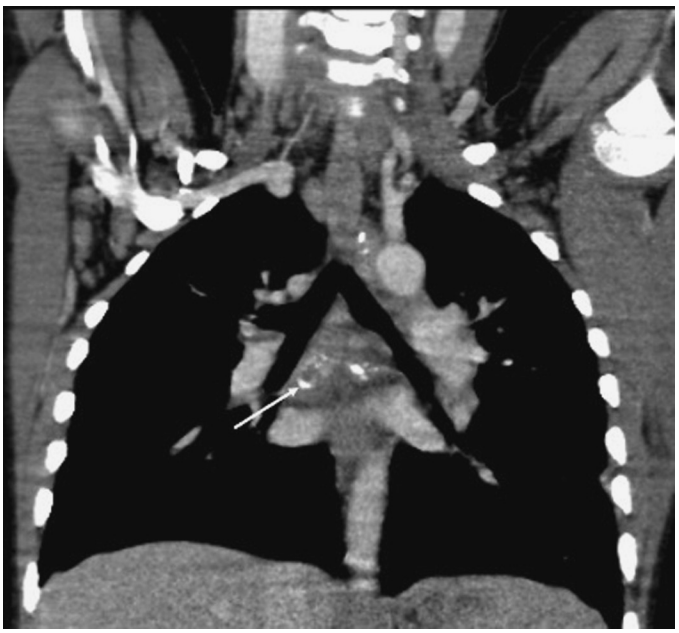

Figure 3. CECT chest mediastinal window coronal section in a 7-year-old child with HIV infection, chronic cough and fever, and positive Mantoux test showing enlarged mediastinal lymph nodes with calcification (*arrow*).

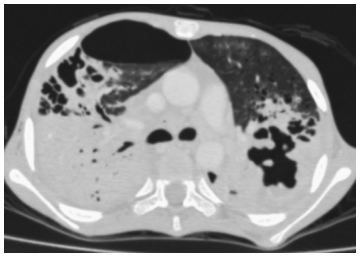

Figure 4. CT chest axial section (lung window) showing large areas of consolidation with breakdown at places in bilateral lungs in an 11-year-old boy with postprimary TB. Presence of pneumothorax on the right side is noted.

If there is hematogenous dissemination of disease to the pulmonary parenchyma, multiple miliary nodules, which are 1 to 3 mm in diameter and show random distribution, are seen (Figure 6). The miliary nodules are typically centered on small blood vessels.

Tubercular pericardial effusions are rare manifestations of extrapulmonary TB, which are often seen in coinfection with HIV. The pericardium is irregularly thickened, frequently in association with mediastinal lymphadenopathy. In chronic stages, pericardium can calcify leading to constrictive pericarditis.[20]

Abdominal Tuberculosis

Abdominal TB is an uncommon presentation especially in children without any other comorbid condition. The various manifestations of abdominal TB include gastrointestinal tract (GIT) involvement, lymph node disease, solid visceral involvement, and peritoneal disease. Of these, the most common form of abdominal TB in children is nodal disease and adhesive peritonitis.[11,21]

Radiographs of the abdomen can show dilated bowel loops with features of obstruction, calcific foci representing calcified lymph nodes, and free intraperitoneal air in cases of perforation. Barium contrast studies used to play an important role in assessment of bowel lesions, localizing its extent and site of involvement. Ulcers, strictures, mucosal fold thickening, matted bowel loops, and fistulas are common findings. However, extramural pathology cannot be evaluated by barium studies.[22] Ultrasound can provide diagnostic clues in certain cases of hepatosplenomegaly, abdominal lymphadenopathy, bowel wall thickening, ascites, and omental stranding. Internal vascularity, bowel peristalsis can be well demonstrated with ultrasound. It is a useful follow-up imaging modality with no risk of radiation. CECT abdomen/CT enterography is the most commonly used cross-sectional imaging investigation in evaluation of abdominal TB. It provides crucial information regarding mural and extraluminal

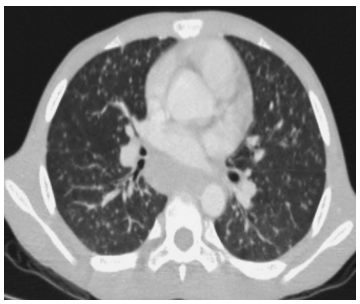

Figure 5. CT chest axial section (lung window) showing multiple centrilobular nodules in bilateral lungs in a 15-year-old immunocompromised boy with disseminated TB.

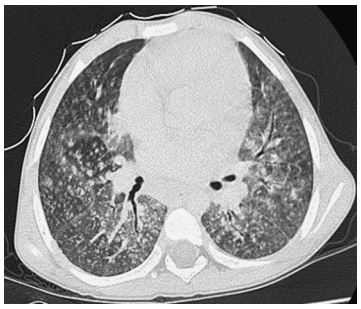

Figure 6. Axial CT chest (lung window) showing multiple miliary nodules in both lungs in a 6-year-old child with HIV infection, chronic cough, and fever (CD4 count 253).

findings. Abdominal MRI, a radiation-free alternative modality with superior soft tissue contrast resolution compared with CT, can be used to evaluate abdominal TB. MRI also can provide extra characteristics, including diffusion restriction, which can help differentiate active versus inactive disease. Also, real-time evaluation of bowel peristalsis is now possible with ultrafast sequences (TruFISP-CINE MR).[23]

Lymphadenopathy is characteristically found in periportal, peripancreatic, mesenteric, and upper retroperitoneal location, with more than one group involved simultaneously. Enlarged lymph nodes can be single, homogeneous, but are more commonly clustered or conglomerated with central necrosis with or without calcification[8] (Figure 7). The necrotic lymph nodes have central anechoic component on US, hypoattenuating on CT, and shows central diffusion restriction on MRI. There is peripheral rim enhancement on postcontrast images. Necrotic lymph nodes are quite specific for TB and should point toward the diagnosis.[24]

Peritoneal TB involves the peritoneal cavity and its reflections, including the mesentery and omentum. It is commonly classified into three types based on imaging features and clinical manifestations: wet type, fibrotic fixed type, and dry plastic type, of which wet type is the most common. The imaging features are often overlapping, and it is difficult to classify into a particular class in certain cases.[25]

Wet type is the most common type of peritoneal TB and is characterized by ascites, peritoneal thickening, and enhancement. Ascites is the most common manifestation of peritoneal disease, and can be free or encapsulated with multiple septations and fine internal echoes on US. It is usually higher in attenuation (25–40 HU), which can show diffusion restriction and postcontrast enhancement on MRI depending on the composition. Peritoneal thickening is hypoechoic on ultrasound and shows intense enhancement on CT. The enhancement is typically regular and uniform, with nodularity less commonly found.

Fibrotic fixed type peritoneal TB is characterized by omental and mesenteric masses with thickened, fixed matted bowel loops and enhancing peritoneum. There is nodular thickening of the peritoneum with low-attenuation masses in the omentum and mesentery. There can also be omental thickening, stranding of the omental fat, thickening of the mesenteric leaflets with loss of normal mesenteric configuration. Small amount of loculated ascites is often seen.

A third type of peritoneal TB, dry plastic type, is defined by fibrous thickening of the peritoneum and mesentery with caseous nodules and dense adhesions. Omental changes as described earlier can be seen. Diffuse infiltration by soft tissue masses in mesentery with multiple mesenteric nodules can be seen.

Abdominal cocoon, also known as sclerosing encapsulating peritonitis, is a rare manifestation of peritoneal TB characterized by encapsulation of the bowel loop in a fibrous membranous sac with associated small bowel obstruction.[26]

Ileocecal junction is the most common site for GIT TB.[8] Involvement of the esophagus, stomach, proximal small bowel, and large bowel is rare. Depending on the host immune response, bowel TB can be ulcerative, ulcerohypertrophic, or hypertrophic type with good host response manifesting as a hypertrophic lesion.[8] Earliest manifestations on barium studies include the hypermobility, spasmodic, thickened, and incompetent ileocecal valve. As the disease progresses, the cecum becomes contracted and irregular, pulled up with the narrowed, rigid terminal ileum. In ulcerative disease, multiple ulcers are seen, which are characteristically stellate or linear shaped with multiple associated short segment strictures. The hypertrophic form is characterized by matted bowel loops with multiple adhesions, resembling a mass. The signs of historic importance described on barium studies include Fleischner's sign, Sterlin's sign, goose neck deformity, and purse string sign.[27] On US, the bowel loop is thickened, heterogenous in echotexture with increased vascularity suggestive of active disease. There is mural thickening with or without mural stratification of the involved bowel on CECT/MRI in active disease (Figure 8). As the disease becomes chronic or latent, the enhancement pattern changes from mural stratified pattern to poor or delayed enhancement of the bowel. In active disease, there can be diffusion restriction on MRI. Complications including fistulas, obstruction, and perforation can be seen on CT/MRI. Other features of abdominal TB including lymphadenopathy and mesenteric edema are commonly present.[8,11]

The most common manifestation of solid visceral involvement includes hepatosplenomegaly with no focal parenchymal abnormality. There can be diffuse heterogenous echotexture or attenuation of the liver and spleen. The other less common pattern includes multiple micronodules scattered in the liver and splenic parenchyma, which vary in size (0.5–2 mm) and number. The lesions can be hypoechoic, isoechoic, or hyperechoic on US. They are usually hypodense on CT (Figure 9), hypointense on T1-weighted images, and hyperintense on T2-weighted sequences. Peripheral rim

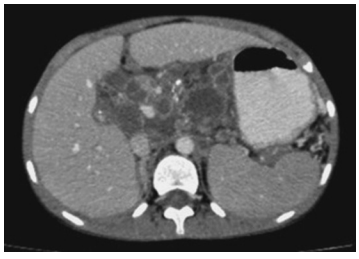

Figure 7. Axial CT section showing multiple enlarged conglomerated necrotic lymph nodes with areas of calcification in an 8-year-old boy with abdominal TB.

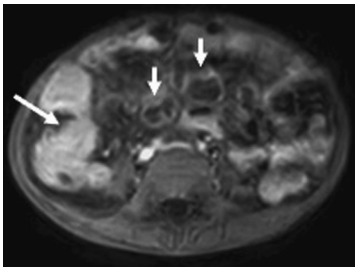

Figure 8. Postcontrast T1-weighted axial image showing a thickened and enhancing ileocecal junction (*long arrow*) in a 12-year-old girl with abdominal TB. Also seen are peripherally enhancing necrotic lymph nodes (*short arrows*).

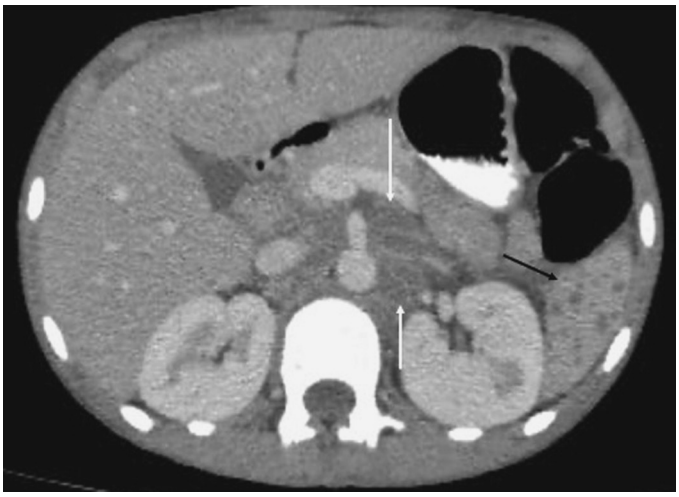

Figure 9. Axial CECT section of upper abdomen in a child with disseminated TB showing multiple small hypodense lesions in the spleen (*black arrow*) with conglomerate retroperitoneal necrotic lymphadenopathy (*white arrows*).

enhancement can be seen. They eventually calcify leading to calcified granulomas. The macronodular form is uncommon.[28] Pancreatic involvement is rare. Imaging features are nonspecific and range from diffusely enlarged pancreas, cystic lesion, and multiple hypodense lesions.[21]

Genitourinary Tuberculosis

Genitourinary involvement is rare in childhood, with often delayed diagnosis due to nonspecific clinical features. The radiologic abnormalities include cortical scarring, calyceal cavitation, infundibular stenosis, calyceal destruction, fibrosis at the renal pelvis ("hiked-up pelvis"), and the calcified kidney. In some cases, the kidney may not be visualized. Irregular thickening of ureters with ureteric stricture can be seen with or without vesicoureteric reflux. The urinary bladder is small in capacity with wall thickening and irregularity.[29]

Musculoskeletal Tuberculosis

Tubercular infections of bone and joint form an important spectrum of tubercular disease. Several forms of musculoskeletal TB include tubercular spondylitis, osteomyelitis, septic arthritis, dactylitis, tenosynovitis, and soft tissue infections.[28,30]

Radiography is usually the initial investigation; however, it can be normal in early disease. Ultrasound can demonstrate superficial abscesses, joint effusions, and soft tissue changes. CT demonstrates erosions and bone changes better than plain radiographs; however, it has a limited role. MRI is the preferred imaging modality for musculoskeletal TB due to higher sensitivity and changes early in the course of disease. Abscess can also be better demonstrated on contrast-enhanced MRI.

Spinal TB is the most common bone manifestation, with most common site being thoracolumbar junction. The vertebral body is most commonly affected with involvement of posterior elements being rare. Three patterns of vertebral body involvement are seen—paradiscal form (most common), anterior lesions, and central lesions. Paradiscal disease is characterized by subchondral bone disease with involvement of the adjacent intervertebral disc. Anterior lesions tend to spread underneath the periosteum and anterior longitudinal ligament leading to compromised blood supply with necrosis of vertebrae. There is collapse of the vertebral body in cases of the central lesion.

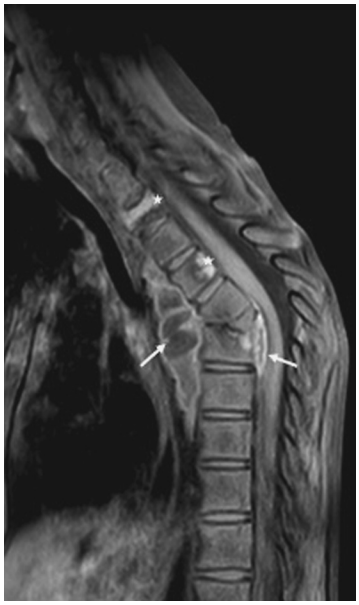

Figure 10. MRI of the cervicodorsal spine in an 8-year-old girl with tubercular spondylitis. Postcontrast, T1-weighted sagittal image showing spondylodiscitis with focal gibbus deformity at T5-T6 vertebral level seen with peripherally enhancing prevertebral and epidural collections (*arrows*). Enhancement is seen in other vertebrae also (*asterisk*).

Contiguous vertebral involvement and skip lesions are common, after spread along the anterior longitudinal ligament. Intraosseous and paraspinal abscesses occur commonly with surrounding inflammatory changes (Figure 10). A prevertebral abscess can result in vertebral body scalloping. Calcification within the abscess is pathognomonic for tubercular abscess. In chronic untreated cases, there are vertebral collapse, kyphosis, and gibbus deformity ultimately leading to bony ankylosis (Figure 11). Sclerosis is uncommon.[8,11,28,30]

Tubercular arthritis usually is monoarticular disease, with the hip joint most commonly involved. Joint disease is more common than isolated bone involvement. The disease usually starts as a bone infection in the metaphysis, which then spreads to the adjacent epiphysis, joint, and overlying soft tissue[30] (Figure 12). There is rarefaction of the bone, trabecular destruction, demineralization, cortical, and cartilage destruction. Poorly defined lytic or sclerotic lesions seen at the site of disease suggest intraosseous abscess, which can be associated with surrounding mild periosteal reaction, sinus formation, and paraosseous abscesses. "Phemister triad" consisting of periarticular osteopenia, bone erosions, and gradual narrowing of the joint space is classically seen with TB.[30] As the disease progresses, there is ankylosis with progressive destruction of the joint. Tubercular dactylitis is more common in children and is characterized by fusiform swelling of short tubular bones, with or without periostitis.

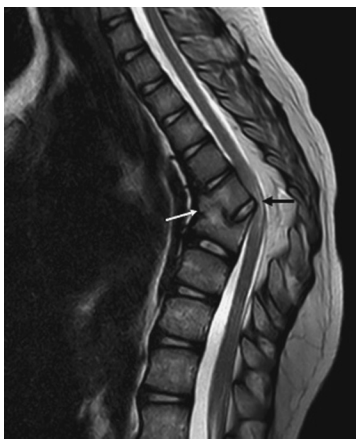

Figure 11. MRI of the spine in a 13-year-old girl under treatment for TB of spine with neurologic symptoms. T2-weighted sequence (sagittal section) shows focal gibbus deformity at D6-D8 vertebral level with partial bony ankylosis of anterior vertebral body of D7-D8 vertebrae. Retropulsion of the D7 intervertebral disc (*white arrow*) causes spinal cord compression with myelomalacia (*black arrow*).

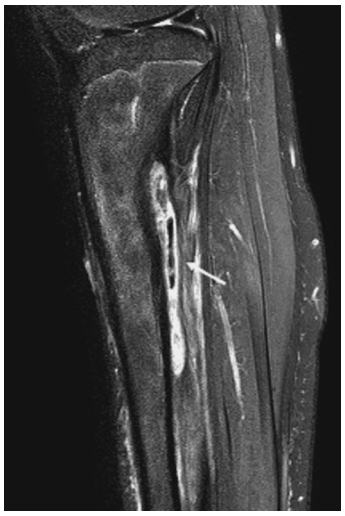

Figure 12. T2-weighted, fat-suppressed sagittal image in a 12-year-old girl with tubercular osteomyelitis who presented with difficulty in walking with the right leg and chronic discharging sinus. Altered signal intensity of marrow of right tibia is noted, with heterogenous collection along the posterior aspect and surrounding muscle edema (*arrow*).

Conclusion

TB can affect any organ, with varied radiologic manifestations in immunocompetent and immunocompromised children. Knowledge of typical and atypical radiologic findings can help to raise suspicion of this infection in children, especially in endemic regions.

References

1. World Health Organization. *Global Tuberculosis Report*. Geneva, Switzerland: World Health Organization; 2019.
2. Jakhar S, Bitzer AA, Stromberg LR, et al. Pediatric tuberculosis: the impact of "omics" on diagnostics development. *Int J Mol Sci*. 2020;21(19):6979.
3. Cruz AT, Starke JR. Clinical manifestations of tuberculosis in children. *Paediatr Respir Rev*. 2007;8(2):107-117.
4. Holmberg PJ, Temesgen Z, Banerjee R. Tuberculosis in children. *Pediatr Rev*. 2019;40(4):168-178.
5. Concepcion NDP, Laya BF, Andronikou S, et al. Standardized radiographic interpretation of thoracic tuberculosis in children. *Pediatr Radiol*. 2017;47(10):1237-1248.
6. Thomas TA. Challenges in diagnosing childhood tuberculosis. *Infect Dis J*. 2016;25(2):26.
7. Sodhi KS, Bhalla AS, Mahomed N, et al. Imaging of thoracic tuberculosis in children: current and future directions. *Pediatr Radiol.* 2017;47(10):1260-1268.
8. Kritsaneepaiboon S, Andres MM, Tatco VR, et al. Extrapulmonary involvement in pediatric tuberculosis. *Pediatr Radiol*. 2017;47(10):1249-1259.
9. Muzumdar D, Vedantam R, Chandrashekhar D. Tuberculosis of the central nervous system in children. *Childs Nerv Syst*. 2018;34(10):1925-1935.
10. Sanei Taheri M, Karimi MA, Haghighatkhah H, et al. Central nervous system tuberculosis: an imaging-focused review of a reemerging disease. *Radiol Res Pract*. 2015;2015:202806.
11. Dawani A, Gupta AK, Jana M. Imaging in pediatric extra-pulmonary tuberculosis. *Indian J Pediatr*. 2019;86(5):459-467.
12. Moon WK, Han MH, Chang KH, et al. CT and MR imaging of head and neck tuberculosis. *Radiographics*. 1997;17(2):391-402.
13. Shah I, Dani S. Profile of tuberculous cervical lymphadenopathy in children. *J Trop Pediatr*. 2017;63(5):395-398.
14. Naranje P, Bhalla AS, Sherwani P. Chest tuberculosis in children. *Indian J Pediatr*. 2019;86(5):448-458.
15. Rana P, Sodhi KS, Bhatia A, et al. Diagnostic accuracy of 3-T lung magnetic resonance imaging in human immunodeficiency virus-positive children. *Pediatr Radiol*. 2020;50(1):38-45.
16. Veedu PT, Bhalla AS, Vishnubhatla S, et al. Pediatric vs adult pulmonary tuberculosis: a retrospective computed tomography study. *World J Clin Pediatr*. 2013;2(4):70-76.
17. Sodhi KS, Sharma M, Saxena AK, et al. MRI in thoracic tuberculosis of children. *Indian J Pediatr.* 2017;84(9):670-676.
18. Heuvelings CC, Bélard S, Andronikou S, et al. Chest ultrasound compared to chest X-ray for pediatric pulmonary tuberculosis. *Pediatr Pulmonol*. 2019;54(12):1914-1920.
19. Arora A, Bhalla AS, Jana M, et al. Overview of airway involvement in tuberculosis. *J Med Imaging Radiat Oncol*. 2013;57(5):576-581.
20. Obihara NJ, Walters E, Lawrenson J, et al. Tuberculous pericardial effusions in children. *J Pediatric Infect Dis Soc*. 2018;7(4):346-349.
21. Tinsa F, Essaddam L, Fitouri Z, et al. Abdominal tuberculosis in children. *J Pediatr Gastroenterol Nutr*. 2010;50(6):634-638.
22. Nagi B, Sodhi KS, Kochhar R, et al. Small bowel tuberculosis: enteroclysis findings. *Abdom Imaging*. 2004;29(3):335-340.
23. Amzallag-Bellenger E, Oudjit A, Ruiz A, et al. Effectiveness of MR enterography for the assessment of small-bowel diseases beyond Crohn disease. *Radiographics*. 2012;32(5):1423-1444.
24. Ablin DS, Jain KA, Azouz EM. Abdominal tuberculosis in children. *Pediatr Radiol*. 1994;24(7):473-477.
25. Sanai FM, Bzeizi KI. Systematic review: tuberculous peritonitis—presenting features, diagnostic strategies and treatment. *Aliment Pharmacol Ther*. 2005;22(8):685-700.
26. Singal R, Satyashree B, Mittal A, et al. Tubercular abdominal cocoon in children—a single centre study in remote area of northern India. *Clujul Med*. 2017;90(2):179-184.
27. Debi U, Ravisankar V, Prasad KK, et al. Abdominal tuberculosis of the gastrointestinal tract: revisited. *World J Gastroenterol*. 2014;20(40):14831-14840.
28. Burrill J, Williams CJ, Bain G, et al. Tuberculosis: a radiologic review. *Radiographics*. 2007;27(5):1255-1273.
29. Nerli RB, Kamat GV, Alur SB, et al. Genitourinary tuberculosis in pediatric urological practice. *J Pediatr Urol*. 2008;4(4):299-303.
30. Teo HE, Peh WC. Skeletal tuberculosis in children. *Pediatr Radiol*. 2004;34(11):853-860.

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1. The Phemister triad refers to
   - **A.** periarticular osteopenia, bone erosions, and gradual narrowing of the joint space
   - **B.** bone erosions, soft tissue abscesses, and gradual narrowing of the joint space
   - **C.** gradual narrowing of the joint space, bone erosions, and soft tissue abscesses
   - **D.** soft tissue abscesses, periarticular osteopenia, and bone erosions
   - **E.** bone erosions, soft tissue abscesses, and ankylosis

2. Which one of the following is the *most* common pattern of vertebrae involvement in spinal TB?
   - **A.** Paradiscal form
   - **B.** Posterior element involvement
   - **C.** Anterior lesions
   - **D.** Central lesions
   - **E.** Isolated intervertebral disc involvement

3. An 8-year-old child presents with clinical features of intestinal obstruction. A radiograph of the abdomen shows dilated, centrally placed small bowel loops. CT of the abdomen shows dilated small bowel loops encapsulated in a fibrous membranous sac. Which one of the following is the *most* likely diagnosis?
   - **A.** Celiac disease
   - **B.** Pseudomembranous colitis
   - **C.** Sclerosing encapsulating peritonitis (abdominal cocoon)
   - **D.** Ischemic bowel disease
   - **E.** Malignant liver lesion

4. Which one of the following is the *most* common site of gastrointestinal TB?
   - **A.** Esophagus
   - **B.** Jejunum
   - **C.** Colon
   - **D.** Ileocecal junction
   - **E.** Stomach

5. Etiopathologic correlates of centrilobular and miliary nodules in lungs include
   - **A.** endobronchial spread: miliary nodules, hematogenous spread: centrilobular nodules
   - **B.** endobronchial spread: centrilobular nodules, hematogenous spread: miliary nodules
   - **C.** lymphatic spread: centrilobular nodules, hematogenous spread: miliary nodules
   - **D.** lymphatic spread: miliary nodules, hematogenous spread: centrilobular nodules
   - **E.** endobronchial spread: miliary nodules, lymphatic spread: centrilobular nodules

6. The *most* common form and manifestation of CNS TB in children is
   - **A.** meningitis with hydrocephalus
   - **B.** meningitis with intracranial bleed
   - **C.** tuberculomas with cranial nerve palsy
   - **D.** intracerebral abscess with hydrocephalus
   - **E.** tuberculomas with intracranial bleed

7. Characteristics of tubercular lymph nodes include
   - **A.** discrete fat-attenuation lymph nodes
   - **B.** hypervascular discrete lymph nodes
   - **C.** necrotic conglomerate lymph nodes
   - **D.** homogeneous soft tissue attenuation lymph nodes
   - **E.** conglomerating densely calcified lymph nodes

8. Which one of the following is a hallmark of postprimary TB in the lung?
   - **A.** Homogeneous consolidation in lower lobes
   - **B.** Cavities in both upper lobes
   - **C.** Centrilobular nodules in lower lobes
   - **D.** Pleural effusion
   - **E.** Mediastinal lymph nodes

9. The *most* common manifestation of solid visceral involvement in abdominal TB is
   - **A.** hepatosplenomegaly
   - **B.** liver abscesses
   - **C.** splenic abscesses
   - **D.** pancreatitis
   - **E.** splenic calcifications

10. Figure 13 is a CECT head axial section showing characteristic findings of CNS TB, which include
    - **A.** intracranial hemorrhage, midline shift, chinked ventricles
    - **B.** hydrocephalus, tuberculomas, basal meningeal enhancement
    - **C.** venous sinus thrombosis, calcified granuloma, subdural effusion
    - **D.** hydrocephalus, tuberculomas
    - **E.** hydrocephalus, basal meningeal enhancement

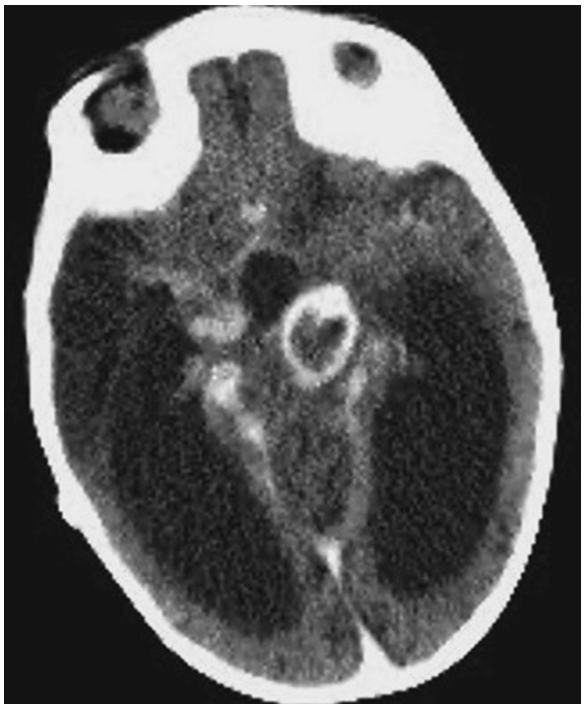

Figure 13.