Review Article

Imaging in Tuberculosis

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Tuberculosis is a leading cause of mortality in our country. Our article highlights the imaging appearances of tuberculosis in various organs with emphasis on its pulmonary involvement. The spectrum of presentation of active and latent tuberculosis infection in the lungs is discussed. The commonly practised algorithms for diagnosis and follow up of tuberculosis in routine clinical practise are summarized. Tuberculosis in cardiac, central nervous system, musculoskeletal and gastrointestinal system is also discussed briefly.

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Tuberculosis (TB) is a common health problem, particularly in our country. Although pulmonary involvement is the most common manifestation, tuberculosis can involve any of the organ systems. Timely diagnosis of the disease with the help of imaging can affect treatment decisions, such as the duration of therapy[1]. Early diagnosis promotes effective treatment and leads to reduced onward transmission of TB[2]. Chest radiographs are used to stratify for risk and to assess for asymptomatic active disease[1]. Though computed tomography scan is frequently employed in the diagnosis and follow-up of TB, it is not a part of the national and international guidelines[3].

Chest Tuberculosis :

It manifests in active and latent forms. Active disease occurs as primary tuberculosis - developing shortly after infection, or postprimary tuberculosis, developing after a long period of latent infection. Primary tuberculosis commonly presents with lymphadenopathy, pulmonary consolidation, and pleural effusion. Postprimary tuberculosis manifests with cavities, consolidations, and centrilobular nodules. Miliary tuberculosis refers to hematogenously disseminated disease that is more commonly seen in immunocompromised patients. Latent tuberculosis is an asymptomatic infection that can lead to postprimary tuberculosis in the future[1,3].

Primary Tuberculosis :

Parenchymal disease — Manifests as consolidation in a segmental or lobar distribution (Fig 1a). Cavitation occurs in a minority of patients with primary tuberculosis- 29% in one series and when cavitation occurs, it is known as progressive primary disease[1]. This cavitation occurs within existing consolidation and thus does not demonstrate an upper lung zone predominance, in contrast to postprimary disease. Resolution of pulmonary consolidation is slow and residual opacities are seen. After resolution, parenchymal scarring can be seen at sites of prior consolidation in 15%–18% of patients and is referred to as a Ghon focus, or Ghon tubercle[1].

Editor's Comment :

- Tuberculosis can involve any organ system and has a wide spectrum of presentation. Imaging plays a key role in the disease course from diagnosis to treatment response and follow up.

Lymphadenopathy — Lymph node and pleural involvement is part of extra-pulmonary tuberculosis (EPTB)[2]. Mediastinal and hilar lymphadenopathy is the most common radiologic manifestation of primary tuberculosis presenting as low attenuation central necrosis with peripheral enhancement on contrast CT scan (Fig 1b). Lymphadenopathy is a common manifestation of tuberculosis in pediatric population. At resolution of lymphadenopathy, calcified normal-sized lymph nodes may be seen[1].

Pleural Effusion — It is seen in approximately 25% of primary tuberculosis cases in adults and are predominantly unilateral. Tuberculous empyemas are loculated and associated with pleural thickening and enhancement (Fig 1c). Tuberculous empyemas may be complicated with bronchopleural fistula or extension into the chest wall (empyema necessitatis). An air-fluid level within an empyema in the absence of intervention is suggestive of a bronchopleural fistula. Residual pleural thickening with calcification can develop, potentially leading to fibrothorax as post treatment sequelae (Fig 1d)[1].

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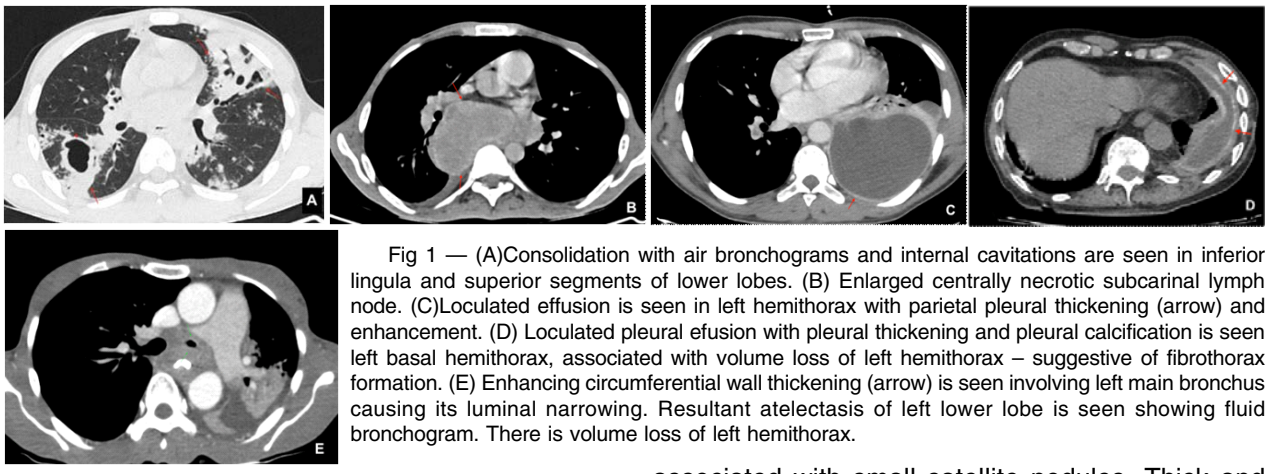

Fig 1 — (A)Consolidation with air bronchograms and internal cavitations are seen in inferior lingula and superior segments of lower lobes. (B) Enlarged centrally necrotic subcarinal lymph node. (C)Loculated effusion is seen in left hemithorax with parietal pleural thickening (arrow) and enhancement. (D) Loculated pleural efusion with pleural thickening and pleural calcification is seen left basal hemithorax, associated with volume loss of left hemithorax – suggestive of fibrothorax formation. (E) Enhancing circumferential wall thickening (arrow) is seen involving left main bronchus causing its luminal narrowing. Resultant atelectasis of left lower lobe is seen showing fluid bronchogram. There is volume loss of left hemithorax.

Airway Disease — Bronchial wall involvement may be seen in primary and postprimary tuberculosis. Bronchial stenosis occurs in 10%–40% of patients with active tuberculosis . The main radiographic features of proximal airway involvement include segmental or lobar atelectasis, lobar hyperinflation and postobstructive pneumonia. At CT, airway involvement can manifest as long segment narrowing with irregular wall thickening, luminal obstruction, and extrinsic compression (Fig 1e)[1].

Postprimary Tuberculosis :

Postprimary tuberculosis result from reactivation of dormant *M Tuberculosis* infection or may result from a second infection with a different strain.

Consolidation and Cavitation — Strong predilection for the apical and posterior segments of the upper lobes as well as the superior segments of the lower lobes in postprimary tuberculosis. In 3%-6% of cases, a noncalcified nodule known as a tuberculoma (ranging from 5 mm to 40 mm in dimension) may be the predominant manifestation associated with small satellite nodules. Thick and irregular walled cavities, often seen within an area of consolidation is a common finding in postprimary tuberculosis (Fig 2a)[1]. Residual cavities may persist after treatment, that predispose to bacterial superinfection, mycetoma formation, or erosion of adjacent vasculature resulting in hemoptysis[3].

Centrilobular Nodules — are seen due to endobronchial spread of infection. At CT, centrilobular nodules are seen in approximately 95% of cases of active tuberculosis, some showing tree-in-bud branching pattern (Fig 2b)[1].

Miliary Tuberculosis :

Hematogenous dissemination , especially in immunocompromised patients, results in miliary tuberculosis. Miliary disease may occur in primary or postprimary tuberculosis. It manifests as diffuse 1–3-mm nodules in a random distribution (Fig 2c)[1].

Imaging findings in sequelae of tuberculosis —

Inactive tuberculosis is characterized by stable fibronodular lesions, fibroatelectatic bands,

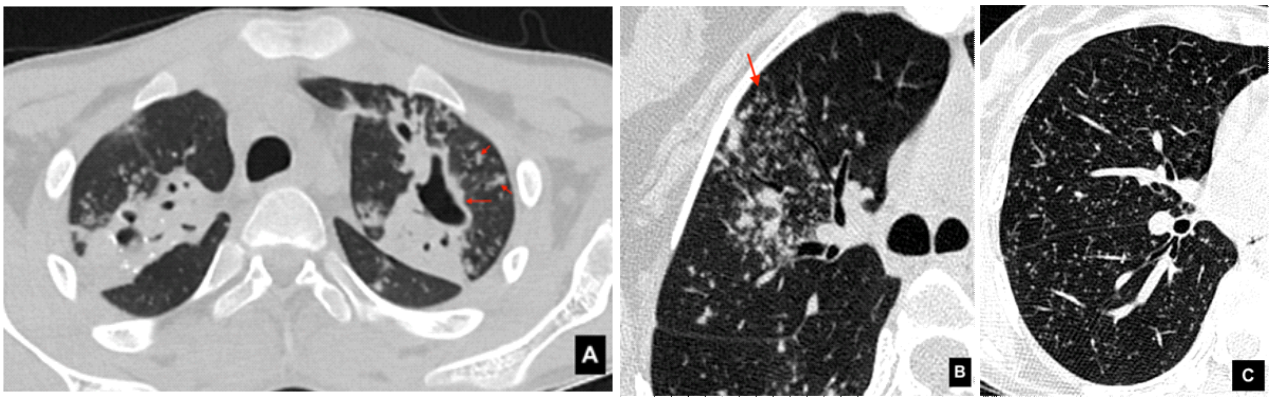

Fig 2 — (A) Consolidation with internal irregular cavity (arrow) is seen in apicoposterior segment of left upper lobe with surrounding discrete centrilobular nodules (short arrow) suggestive of reactivation of tuberculosis. Fibronodular and fibrobronchiectatic changes with calcification are seen in right upper lobe. (B) Multiple discrete centrilobular nodules are seen, some showing tree-in-bud branching pattern representing endobronchial spread of infection. (C) Widespread discrete 2-3 mm sized nodules are seen in random distribution pattern representing miliary tuberculosis.

peribronchial fibrosis, bronchiectasis and architectural distortion (Fig 3)[1]. Thin-walled cavities and well-defined nodules may persist for a long time after completion of antituberculous treatment. Tuberculomas and small calcified lung nodules also suggest prior infection. Calcified lymph nodes and pleural thickening (with/without calcification) are also imaging features of healed TB[3].

Persistent lesions at the end of anti-tuberculous treatment —

The activity of residual lesions needs to be resolved using imaging and/or laboratory parameters (Fig 4a). The residual inactive lesions, which are stable on follow up imaging, do not require further treatment. In case of partial or no response, ATT is prolonged as per guidelines. The persistent lesions may represent drug-resistant TB, in which case drug susceptibility testing is recommended. Appearance of new lesions may represent reactivation of tuberculosis. Stability of radiographic findings for 6 months distinguishes inactive from active disease[1].

Imaging algorithm for diagnosis of pulmonary tuberculosis —

As per the RNTCP guidelines, any person with cough for 2 or more weeks is a Pulmonary TB suspect. In addition to sputum smear examination, all such patients should be subjected to a CXR, wherever feasible.

If the radiographic findings suggest active TB (Fig 4b), ATT may be started in concordance with clinical scenario. In case the X-ray findings are equivocal and not specific for TB, confirmation with non-contrast or contrast enhanced CT scan is needed (preferably contrast enhanced).

If the CXR suggests healed TB, then comparison with prior imaging is required to document stability, failing the availability of previous imaging, a CT is usually done to confirm the absence of active infection.

Based on the CT scan findings, radiologist should categorize the patient into active, healed or indeterminate categories.

In case CT scan is indeterminate for disease activity, other parameters like BAL, lab parameters, or tissue sampling play an important role[3].

Protocol for follow-up of pulmonary tuberculosis —

Follow-up is done at the end of intensive phase/continuation phase (IP/CP). In case of no response to treatment, follow-up is repeated after extension of IP/CP as per the RNTCP guidelines.

(1) CXR is done at the completion of IP of the treatment regimen. If there is significant / near

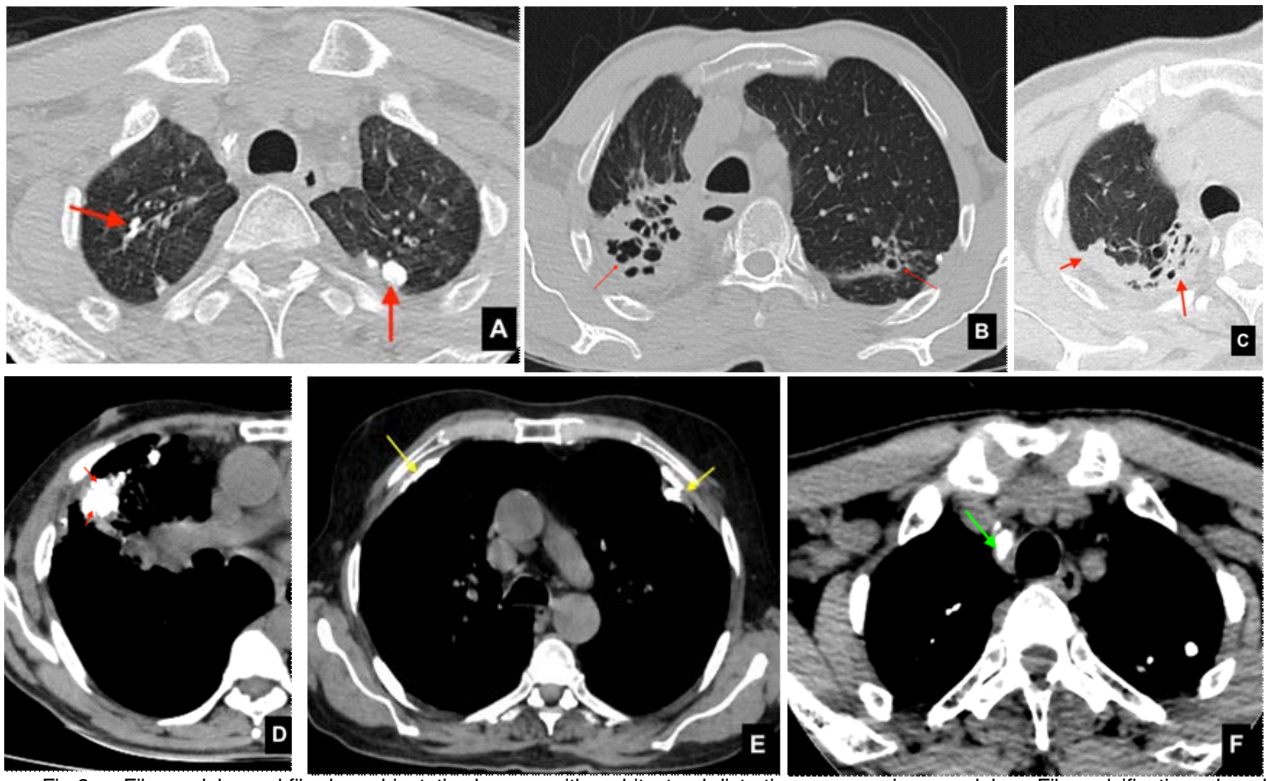

Fig 3 — Fibronodular and fibrobronchiectatic changes with architectural distortion are seen in upper lobes. Fibrocalcification, pleural thickening with calcification and calcified lymph nodes are also seen as sequelae of old tuberculous infection.

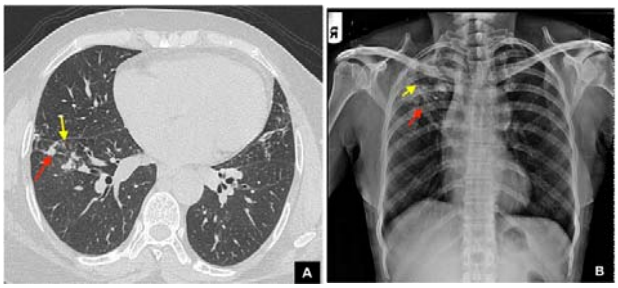

Fig 4 — (A) Fibronodular changes (red arrow) are seen in right lower lobe with surrounding discrete nodules (yellow arrow), the activity of the nodules cannot be decided in absence of previous imaging . Clinical workup and follow up CT scan after 3 months is indicated in such cases. (B) Consolidation with internal cavitation is seen in right upper zone. There is widening of bilateral paratracheal strip (due to mediastinal lymphadenopathy). These findings are in favour of active tuberculosis.

complete resolution of findings or CXR depicts only sequelae of prior infection, then no further imaging is needed at the end of treatment regimen, provided there is clinical improvement as well.

(2) If the CXR shows residual findings or is indeterminate and the patient is improving clinically, then a follow up CXR is recommended at the end of ATT course. The follow up CXR if shows findings as mentioned above, then ATT can be stopped. However, if the follow up CXR still shows residual findings, ATT maybe prolonged depending on clinical scenario.

(3) In case of no definite response on CXR and absence of clinical improvement, CT may be done to assess disease activity. Non-contrast CT is sufficient for follow-up of parenchymal lesions, but contrast administration is required for follow-up of nodal disease. ATT may be prolonged in case CT suggests residual active disease or if CT is indeterminate but clinical and laboratory parameters do not suggest any treatment response[3].

Apart from lungs, tuberculosis affects other organs presenting in various typical and atypical manifestations. A brief overview of extra-pulmonary tuberculosis is as mentioned below:

Cadiac Tuberculosis :

This accounts for only 0.5% cases of extrapulmonary tuberculosis. Pericardial involvement is more common than myocardial involvement.

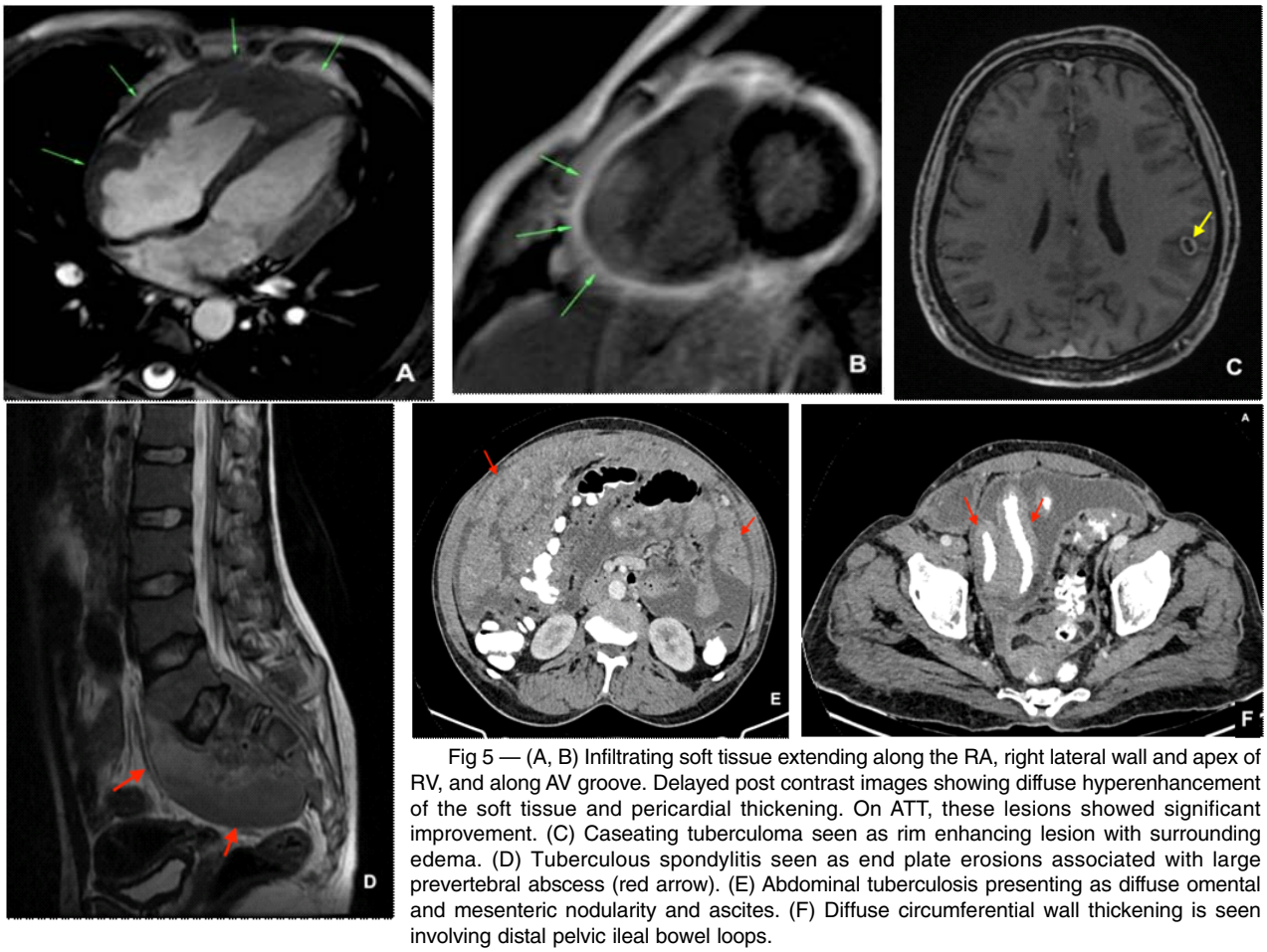

Fig 5 — (A, B) Infiltrating soft tissue extending along the RA, right lateral wall and apex of RV, and along AV groove. Delayed post contrast images showing diffuse hyperenhancement of the soft tissue and pericardial thickening. On ATT, these lesions showed significant improvement. (C) Caseating tuberculoma seen as rim enhancing lesion with surrounding edema. (D) Tuberculous spondylitis seen as end plate erosions associated with large prevertebral abscess (red arrow). (E) Abdominal tuberculosis presenting as diffuse omental and mesenteric nodularity and ascites. (F) Diffuse circumferential wall thickening is seen involving distal pelvic ileal bowel loops.

Tuberculous pericarditis presents as pericardial thickening of more than 3 mm, frequently associated with mediastinal lymphadenopathy. Tumefactive tubeculosis of myocardium (Fig 5a,b) presents as infiltrating masses which need to be differentiated from other lesions such as angiosarcoma and lymphoma[2].

CNS Tuberculosis :

CNS tuberculosis can manifest in a variety of forms, including tuberculous meningitis, tuberculomas, tuberculous abscesses, tuberculous cerebritis, and miliary tuberculosis. TB meningitis presents as abnormal meningeal enhacement (more marked in basal cisterns and within the sulci). The common complications are communicating hydrocephalus and ischemic infarcts.

A noncaseating tuberculoma is hypointense relative to gray matter on T1-weighted images and hyperintense on T2-weighted images, with homogeneous enhancement while caseating tuberculomas are isointense to hypointense on both T1- and T2-weighted, have a variable amount of surrounding edema and show rim enhancement on post contrast study (Fig 5c).

Head and Neck Tuberculosis :

Most commonly it presents as cervical lymphadenitis, initially homogeneous but later undergo central necrosis. 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. Extranodal tuberculous disease in neck most commonly involves the larynx, temporal bone, and pharynx[2,4].

Musculoskeletal Tuberculosis :

Tuberculous spondylitis most commonly affects the lower thoracic and upper lumbar vertebrae. Infection usually begins in the anterior part of the vertebral body adjacent to the end plate with subsequent end plate erosion and involvement of intervertebral disc. There may be associated prevertebral, bilateral paravertebral and epidural abscess as well (Fig 5d). Psoas abscess are also commonly known to occur. If left untreated, the infection eventually results in vertebral collapse , anterior wedging, leading to kyphosis and gibbus formation. With healing, ankylosis of the vertebral bodies occurs, with obliteration of the intervening intervertebral disc[6].

Tuberculous osteomyelitis usually affects the metaphyses of involved bones. It presents as poorly defined lytic lesions with surrounding sclerosis and may be associated with abscess formation.

Tuberculous arthritis is characteristically a monoarthritis affecting large weight-bearing joints. It presents as osteopenia, synovitis, marginal erosions, and varying degrees of cartilage destruction. Joint space narrowing is usually delayed. The end result is usually fibrous ankylosis of the joint[2,6].

Abdominal Tuberculosis :

Abdomen is the most common site of extra-pulmonary tuberculosis. There is a varied presentation of abdominal tuberculosis dependenting on the organ of involvement. It may present as an isolated organ involvement or as a combination of findings.

Abdominal lymphadenopathy —

Abdominal lymphadenopathy is the most common manifestation of abdominal tuberculosis, presenting as enlargement of mesenteric and peripancreatic lymph nodes. The enlarged lymph nodes show homogeneous attenuation or are centrally necrotic. Calcifed mesenteric lymph nodes are seen in healed stage of disease.

Tuberculous peritonitis —

Wet type peritonitis is the most common presentation, seen as free or locualted ascites. Fibrotic peritonitis presents as omental and mesenteric cake-like masses with matted bowel loops. Peritoneal thickening and fibrous adhesions are also seen (Fig 5e).

Gastrointestinal tuberculosis —

The most common imaging finding is concentric mural thickening showing enhancement. Ileo-cecal junction is commonly involved. Skip areas of concentric mural thickening with associated luminal narrowing with or without proximal dilatation can also occur elsewhere in the small bowel (Fig 5f).

Hepatosplenic and adrenal tuberculosis —

Hepatosplenic tuberculosis is common in patients with disseminated disease and presents as innumberable 0.5-2.0 mm nodules, often not detected at CT scan and appear hypointense on MRI images. The CT signs of active tuberculous involvement of adrenals are bilateral enlarged glands associated with large, necrotic areas, with or without calcification.

Genitourinary tuberculosis —

Renal tuberculous involvement presents as “moth-eaten” calyx which progresses to papillary necrosis. Dilated calices may be due to infundibular stricture within the collecting system. Calcifications in a lobar distribution are often seen in end-stage tuberculosis.

Ureteric tuberculosis is characterized by a thickened ureteric wall and strictures. Urinary bladder tuberculosis commonly manifests as reduced bladder volume with wall thickening, ulceration, and filling

defects due to granulomatous material.

Genital tuberculosis involves the fallopian tubes in women usually causing bilateral salpingitis with obstruction and multiple constrictions of the fallopian tubes and endometrial adhesions or deformity of the cavity.

Summary :

Imaging plays an important role in the diagnosis and follow up of tuberculosis. Categorisation into active, healed and indeterminate disease activity can be confidently done with imaging. Good response to anti-tuberculous treatment and suspicion of drug- resistant tuberculosis can also be commented upon with the help of follow up scans, which affects treatment regimen and duration. Radiological modalities go hand in hand with clinical and pathological workup for evaluation of tuberculosis, however there are no imaging guidelines for the diagnosis and follow up of such cases.

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