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REVIEW

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Unusual patterns of tuberculosis on cross-sectional imaging: a pictorial review

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Abstract

Background: Tuberculosis is routinely encountered on imaging throughout the world. Radiologists are usually familiar with the common patterns of tuberculosis on cross-sectional imaging. However, tuberculosis frequently presents with unfamiliar imaging patterns and/or uncommon anatomic sites which still pose a diagnostic challenge.

Discussion: Rapid improvements in the management of acquired immunodeficiency syndrome patients leading to increased survival as well as advances in chemotherapy and immunosuppressive medication have complicated the presentation of tuberculosis by increasing the probability of unconventional sites and patterns of involvement in tuberculosis leading to unfamiliar imaging appearances. In this review, we describe these unfamiliar imaging patterns of tuberculosis and provide a diagnostic protocol for arriving at the right diagnosis and differentiating these from other pathologies with similar imaging appearances. These are described through case-based illustrations of unusual patterns and anatomic locations of tuberculosis.

Conclusions: It is important for radiologists to be aware of such unusual imaging patterns of tuberculosis in order to facilitate early and accurate diagnosis for appropriate patient management in the face of changing patient demographics and pathological profile of tuberculosis in the current era of scientific advancements.

Keywords: Tuberculosis, Cross-sectional imaging, Unusual patterns, Uncommon anatomic sites

Background

Tuberculosis (TB) is a global health problem with the lungs being the most common site of involvement [1]. The role of imaging in the early diagnosis and management of tuberculosis has remained paramount from the era of conventional radiographs to the modern era of cross-sectional imaging. The usual imaging patterns of pulmonary and common extrapulmonary tuberculosis are well known [2]. As tuberculosis can virtually affect any anatomical site, it is important to be aware of not only the imaging patterns of TB at uncommon anatomic sites but also the unusual imaging patterns at common anatomic sites. These unusual imaging patterns of TB are relatively less well known; as TB can masquerade several other pathologies, such unusual imaging patterns often lead to incorrect or missed diagnosis [3]. In this article, we have briefly touched upon individual factors predisposing to such unusual patterns of TB, following which various unusual imaging patterns on cross-sectional imaging throughout the body have been described in detail through case-based illustrations. Imaging features which point to the diagnosis of TB in individual cases and differentiating features from close differentials have been described. We have also proposed a protocol for reporting such challenging cases that can subsequently help in the follow-up process and aid the clinician in planning appropriate treatment regimens.

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Discussion

Individual factors predisposing to unusual imaging patterns of TB

There are certain factors which predispose an individual to the disseminated forms and unusual sites/unusual

imaging patterns of TB. As tubercular infection initiates a cell-mediated immune response, any process which affects cell-mediated immunity not only predisposes to tubercular infection but can also cause exacerbation of pre-existing TB or reactivation of latent TB [4, 5].

Immunosuppression

Human immune deficiency virus (HIV) infection

HIV reduces the cell-mediated immune response, especially $CD4^+$ T helper cells predisposing to TB.

Immune-mediated inflammatory diseases (IMIDs)

Tumour necrosis factor-alpha (TNF-α) is a major cytokine in cell-mediated immune response and plays a crucial role in controlling the mycobacterial infection. TNF-α inhibitors are used in the treatment of IMIDs and increase the risk of TB reactivation and exacerbation.

Steroid use

A substantial dose of glucocorticoids for a long duration causes suppression of cell-mediated immune response.

Organ transplant recipients

Immunosuppressant use in transplant recipients predisposes to TB infection.

Malnutrition and low body mass index (BMI)

Both macronutrient and micronutrient deficiency impair the immune response. Jick et al. found that individuals diagnosed with TB are 2.8 times more likely to have a BMI of < 20 with low BMI predating tubercular infection by an average of 2.6 years [4].

Diabetes

It causes impairment of the innate and adaptive immunity, resulting in decreased levels of cytokines and interferon-γ and a reduction in neutrophil chemotaxis. All these factors contribute to the proliferation of tubercular bacilli.

Smoking

It interferes with mucus clearance and phagocytic function of alveolar macrophages and results in a reduction in $CD4^+$ T helper cell count, thereby increasing the risk of developing TB.

Alcohol

It interferes with the signalling pathways responsible for cytokine production in cell-mediated immunity [4, 5].

Unusual imaging patterns of TB

Central nervous system (CNS)

Pituitary and hypothalamic tuberculomas In the brain parenchyma, the most common sites of tuberculoma formation are the parietal and frontal lobes [2]. Imaging characteristics of a tuberculoma on MRI (magnetic resonance imaging) depend on the presence of caseation and central necrosis:

- Non-caseating granuloma appears iso-hypointense on T1 and hyperintense on T2 and shows homogenous enhancement.
- Caseating granuloma is isointense on T1 with a hyperintense rim, appears hypointense on T2 and shows homogenous or ring enhancement.
- Caseating granuloma with central necrosis has a hyperintense rim on T1 similar to a caseating granuloma. Due to necrosis, it appears centrally hyperintense on T2 with a hypointense rim and shows ring enhancement.
- Calcified granuloma is hypointense on all sequences [6].

Isolated pituitary and hypothalamic involvement is rare in TB [2]. We have described a case of TB where enhancing granulomas were seen in the hypothalamus, pituitary and midbrain (Fig. 1). Cerebrospinal fluid (CSF) evaluation confirmed tubercular aetiology. The presence of conglomerate lesions rather than a single mass should lead to suspicion of tuberculosis over more common pituitary lesions/masses (such as adenoma, craniopharyngioma or Rathke cleft cyst). Other differentiating features of pituitary tuberculomas are peripheral or ring enhancement and involvement of pituitary stalk [7]. Additional evidence of TB such as exudates in basal cisterns and leptomeningeal enhancement, as seen in our case, is helpful.

Tuberculosis with secondary moyamoya vasculopathy Vasculitis is commonly encountered in tubercular meningitis (TBM). However, the moyamoya pattern of vasculopathy in TBM is very rare. We came across a known case of CNS TB on anti-tubercular therapy who presented with weakness in the right half of the body. Imaging showed infarction in left MCA (middle cerebral artery) territory with attenuated supraclinoid ICA (internal cerebral artery) (Fig. 2). Imaging of another known case of CNS TB, who presented with multifocal neurological deficits, showed bilateral ACA (anterior cerebral artery) and right MCA infarcts with attenuated supraclinoid ICA and multiple collateral vessels (Fig. 3). Moyamoya vasculopathy usually occurs as a late complication in a known case of CNS TB. The presence of multi-territorial ischaemic infarcts, ivy sign (FLAIR hyperintensity and contrast

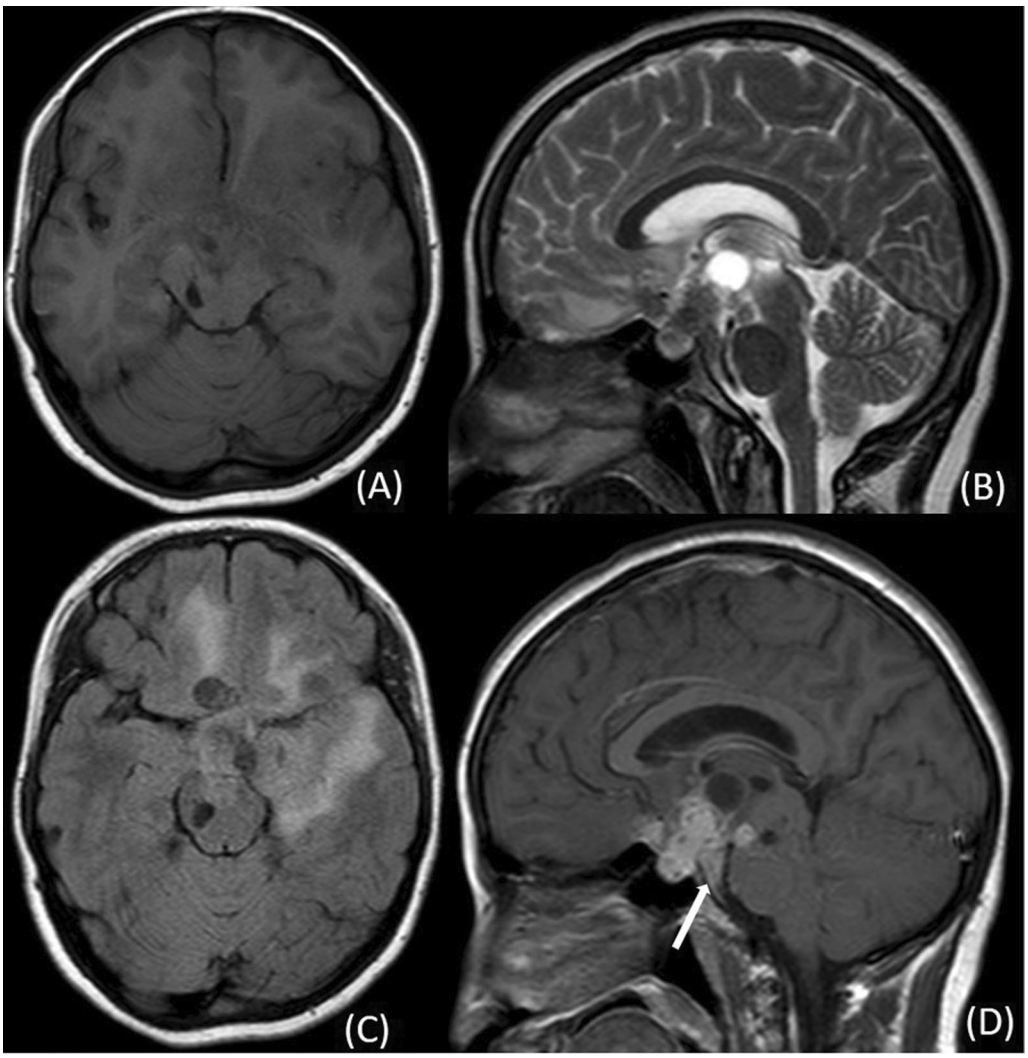

Fig. 1 Pituitary and hypothalamic tubercular granulomas. MRI of a 24-year-old male shows multiple conglomerate lesions in pituitary, hypothalamus and midbrain which appear isointense with hyperintense rim on axial T1W image (**A**), variable signal intensity on T2W image (**B**), extensive surrounding white matter oedema is seen on FLAIR (**C**), on T1 contrast-enhanced image the lesions show peripheral enhancement with basal exudates in prepontine cistern (arrow in **D**)

enhancement due to prominence of leptomeningeal collateral vessels) and attenuation of supraclinoid ICA with prominent collateral vessels on angiography point to the diagnosis of moyamoya disease [8]. Vasculitis due to CNS TB, in contrast, affects small- and medium-sized vessels (thalamo-perforating and lenticulostriate arteries) causing lacunar infarcts in ganglio-thalamic regions [9].

Intramedullary spinal tubercular granuloma It is extremely rare, accounting for two per 100,000 cases of TB and two per cent cases of CNS TB. It can mimic other intramedullary lesions in the spinal cord. We have described a case who presented with paraplegia, cough and fever. MRI spine showed a ring-enhancing intramedullary lesion in the cord (Fig. 4). Chest CT revealed miliary nodules and sputum analysis revealed acid-fast bacilli (AFB)-positive tubercular bacilli. Weakness of bilateral lower limbs improved 15 days after starting antitubercular therapy. The presence of target sign on T2W images (hypointense central signal due to caseation and hyperintense peripheral rim due to granulation tissue) helps in differentiating tubercular granuloma from other intramedullary cord lesions [10]. Tuberculoma causes focal involvement of the spinal cord, whereas common intramedullary tumours like ependymoma and astrocytoma cause relatively long segment cord involvement. Demyelinating diseases also cause a longer segment of cord involvement than tubercular granulomas. Vascular

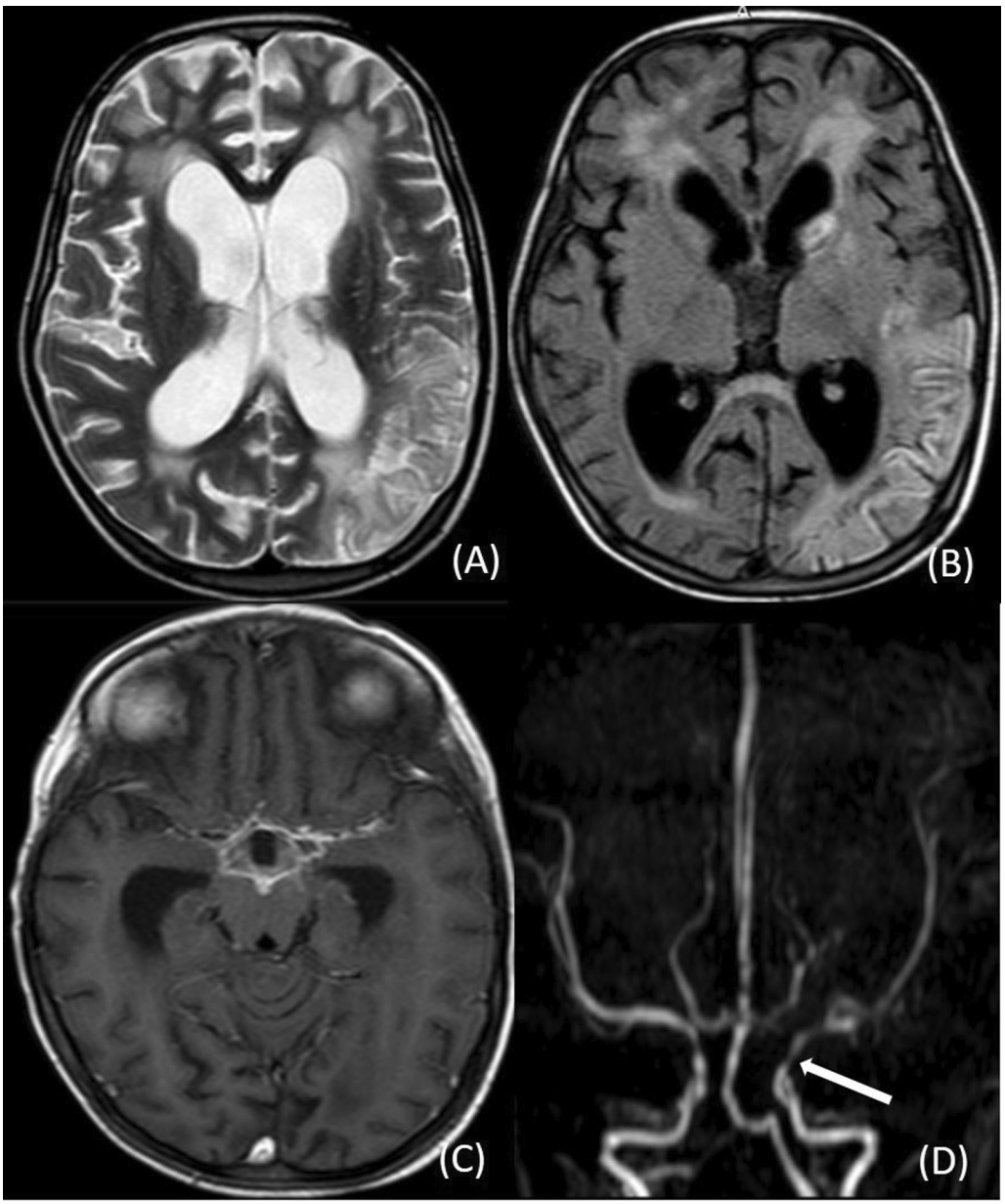

Fig. 2 CNS TB with moyamoya vasculopathy and infarction in left MCA territory. MRI of a 5-year-old girl shows hyperintensity involving both grey and white matter in left MCA territory on T2W (**A**) and FLAIR image (**B**) with communicating hydrocephalus (**A** and **B**), T1 contrast-enhanced image shows exudates in basal cistern and left sylvian fissure (**C**), MR angiography shows attenuated supraclinoid segment of left ICA and left MCA (white arrow in **D**)

intramedullary lesions show flow voids, which are not seen in granulomas. Differentiation from intramedullary metastasis is challenging [11]. The presence of systemic symptoms (anorexia, weight loss, night sweats, fever), past history of TB or typical TB findings at other sites are other useful characteristics that help in the differentiation of tubercular granulomas from other intramedullary cord lesions.

Extramedullary intradural tubercular granuloma Tuberculoma at this location is also extremely rare. It can mimic an extramedullary spinal tumour and poses a diagnostic challenge in the absence of other features of tuberculosis [12]. We encountered a case who presented with symptoms of compressive myelopathy along with systemic symptoms of tuberculosis. The presence of peripherally enhancing granuloma on the MRI

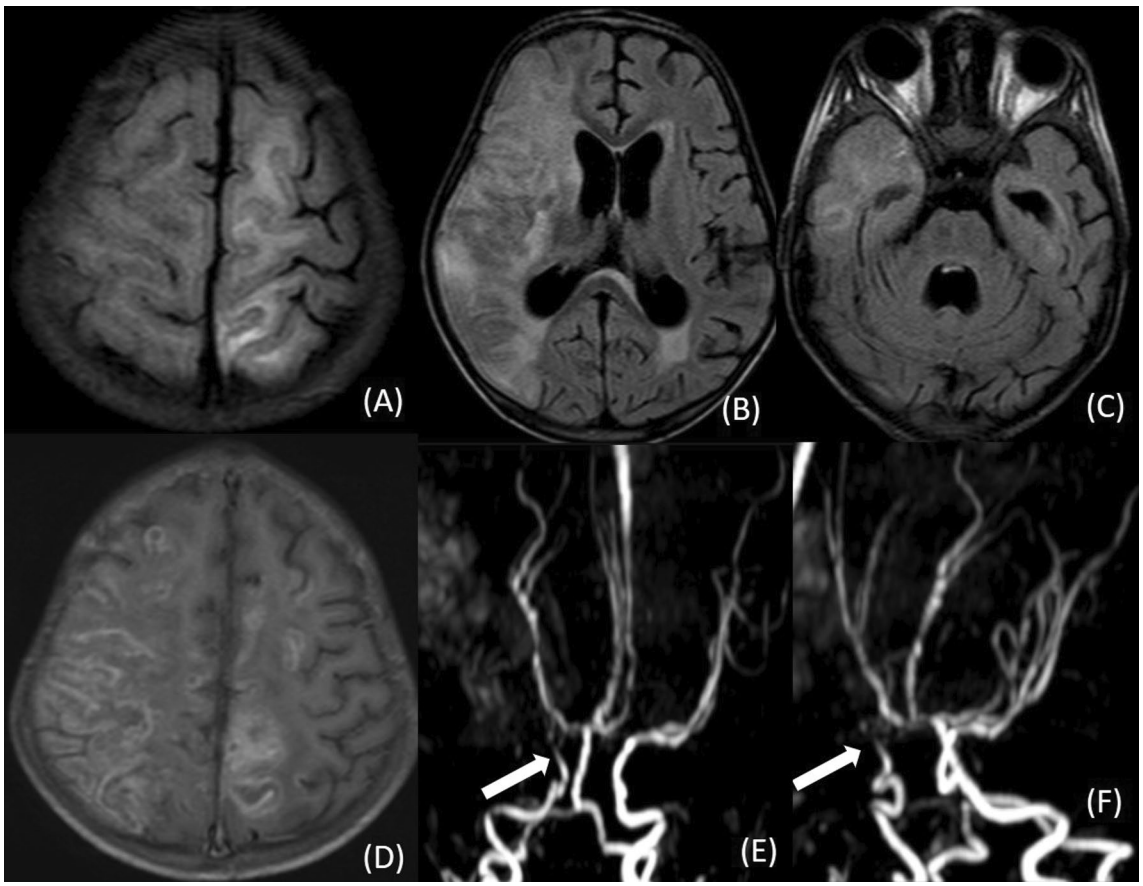

Fig. 3 CNS TB with moyamoya vasculopathy. MRI of a 4-year-old boy shows hyperintensity involving both grey and white matter in bilateral ACA and right MCA territory on FLAIR images (**A**–**C**), T1 contrast-enhanced image shows leptomeningeal enhancement in bilateral ACA territory- ivy sign (**D**). MR angiography (**E**, **F**) shows near-complete occlusion of supraclinoid segment of right ICA and attenuation of supraclinoid segment of left ICA

spine led to suspicion of tubercular granuloma (Fig. 5) due to the presence of associated systemic symptoms. CSF examination showed the presence of tubercular bacilli. Imaging features can help in differentiation from the more common extramedullary intradural lesions like meningiomas (which show vivid enhancement, calcification and dural tail sign) and neurogenic tumours (which are usually dumb-bell shaped and cause neural foraminal widening) [13]. However, these features are not specific and can occasionally be seen in tuberculoma as well [12]. In cases of extramedullary intradural granulomas, a CSF examination is necessary for confirmation of the diagnosis.

Cardiac

Tubercular pericarditis and myocarditis The pericardium is most commonly affected in cardiac TB, resulting in pericarditis, other findings being myocarditis and aortitis. Tubercular pericarditis can present with features of constrictive pericarditis or pericardial effusion or a combination of the two. CT shows irregular or nodular pericardial thickening (> 3 mm). Pericardial calcification and associated pleural effusion may be seen [2]. Diffuse infiltrative, diffuse miliary and confluent masses (tuberculomas) are the various patterns of myocardial involvement in TB. Tuberculomas are usually T1 and T2 iso-hypointense (w.r.t myocardium) and show heterogeneous late gadolinium enhancement. This is a useful differentiating feature as most other infiltrative disorders of the myocardium are T2 hyperintense [14]. We have described a case of cardiac TB with pericardial and myocardial involvement along with lung nodules (Fig. 6). Endomyocardial biopsy confirmed tubercular carditis. The presence of concurrent TB findings in the lungs and mediastinal lymphadenopathy may aid in the diagnosis of cardiac TB if they are present, as in our case.

Respiratory

Tubercular tracheitis Tracheal TB is usually characterized by long segment (> 3 cm) involvement of the trachea. The distal trachea is usually involved. CT shows circum-

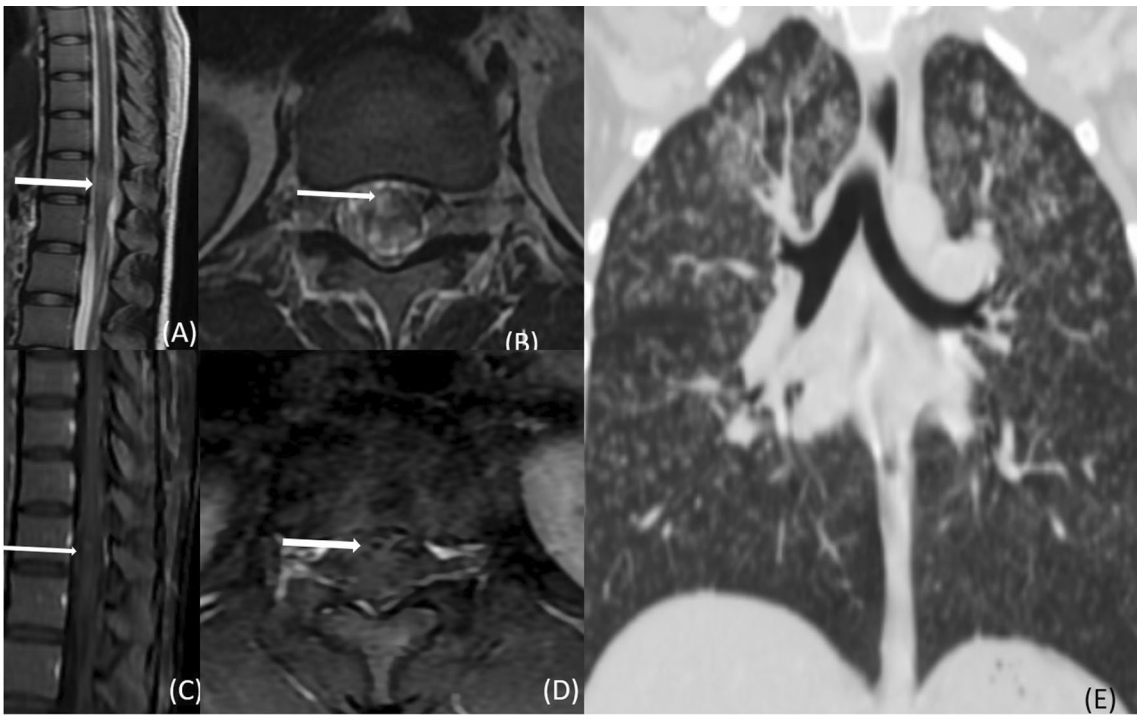

Fig. 4 Intramedullary tubercular granuloma. MRI of a 46-year-old male shows focal ill-defined hyperintensity in cord with cord expansion at T12 vertebral level on sagittal T2W image (arrow in **A**), axial T2W image shows cord expansion with ill-defined hyperintense lesions in the cord (white arrow in **B**), T1W contrast-enhanced sagittal image shows subtle enhancement (arrow in **C**) and axial image shows ring enhancement (arrow in **D**), chest CT shows miliary tubercular nodules in both lungs (**E**)

ferential thickening of the tracheal wall with irregular luminal narrowing. Tubercular tracheitis is frequently associated with bronchial TB. There may be surrounding mediastinitis, seen as mediastinal fat stranding on CT [15]. A common sequela of chronic tubercular tracheitis is multifocal tracheal stenosis due to mural fibrosis. Imaging in our case showed tracheal wall thickening, with concurrent TB features in lung and mediastinal lymphadenopathy, which enabled accurate diagnosis of acute tubercular tracheitis on imaging (Fig. 7). Sputum analysis showed acid-fast bacilli confirming the diagnosis. Differential diagnosis includes GPA (granulomatosis with polyangiitis) and amyloidosis; both of these can cause circumferential tracheal thickening. The presence of upper respiratory involvement, sub-glottic tracheal stenosis, cavitatory lung lesions and ill-defined airspace opacities (due to alveolar haemorrhage) suggests GPA, whereas irregular tracheal wall calcification and associated chronic renal disease favour amyloidosis [16].

Musculoskeletal

Multifocal skeletal tuberculosis It is defined by the presence of osteoarticular lesions at ≥ 2 locations. In endemic countries, it accounts for $< 5\%$ of the cases of skeletal TB. It presents with vague systemic symptoms like fever, anorexia and weight loss. On imaging, this can mimic metastasis or haematological malignancy, especially in the absence of pulmonary involvement. MRI helps in better lesion characterisation. The lesions usually appear iso-hypointense on T1 and hyperintense on T2 and show heterogeneous post-contrast enhancement [17]. We have described a case of multifocal skeletal tuberculosis in a young patient. Imaging showed multiple lesions in lumbar vertebrae, sacrum, ilium and ischium (Figs. 8 and 9). CT-guided biopsy from an ischial lesion showed tubercular granulomas. In such cases of isolated multifocal skeletal tuberculomas, accurate diagnosis on imaging is not possible. Differentials include haematological malignancies and metastasis. In haematological malignancies, the pattern of marrow involvement is usually diffuse but occasionally it can be multifocal as well. The presence of primary malignancy and older age can point to metastatic involvement. However, in many cases of multifocal skeletal lesions a definitive diagnosis is not possible on the basis of imaging alone and confirmation requires biopsy.

Tubercular chondro-sternal arthritis While articular involvement of tuberculosis is a relatively common form of musculoskeletal TB and has been well described in the literature, tubercular involvement of chondro-sternal joint

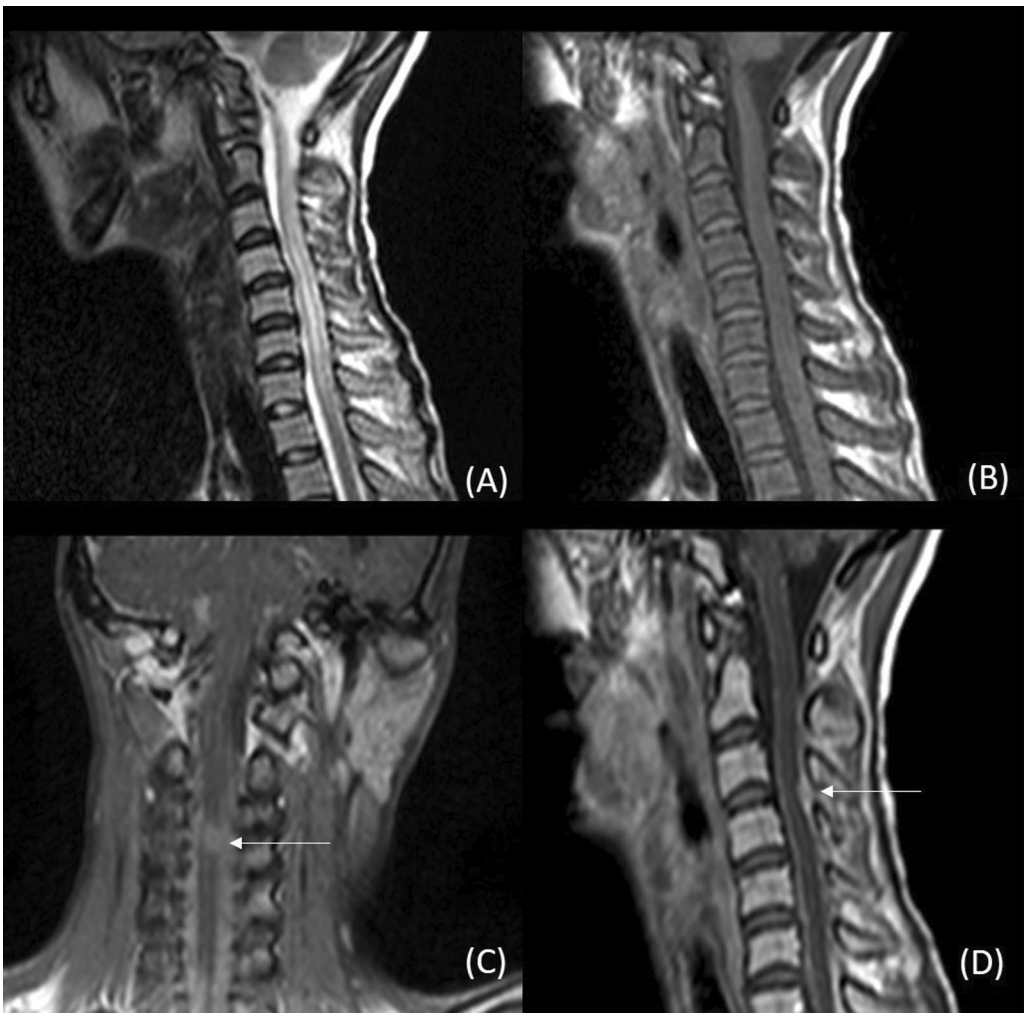

Fig. 5 Extramedullary intradural tubercular granuloma with cord oedema. MRI of a 32-year-old female shows cord expansion with high signal intensity in the cord from C3 to C7 vertebral levels, suggestive of cord oedema on sagittal T2W image (**A**), T1W image also shows cord expansion at the same level (**B**), T1 contrast-enhanced images show a well-defined peripherally enhancing extramedullary intradural lesion posteriorly at C3–C4 vertebral level (white arrow in **C** and **D**)

is unusual. Infection occurs due to haematogenous dissemination. We encountered a case of tubercular chondro-sternal joint arthritis; imaging showed widening of chondro-sternal joint space with effusion and extension of the joint collection focally into the overlying anterior chest wall. The collection also showed diffusion restriction. Associated necrotic mediastinal lymphadenopathy was also present (Fig. 10). Ultrasonography-guided fine needle aspiration cytology (FNAC) from the collection confirmed tubercular aetiology. The presence of concurrent TB features in the lungs and mediastinal lymphadenopathy may aid in the diagnosis in such challenging cases.

Tubercular knee arthritis mimicking JRA (juvenile rheumatoid arthritis) In young patients, knee joint TB can mimic JRA and differentiation between the two may be difficult in cases of monoarticular involvement with no previous or family history of TB. Both the conditions can cause juxta-articular osteopenia, marginal erosions, bone marrow oedema, joint effusion and synovial thickening with preservation of joint space till the late stages. Few differentiating features have been described on MRI. TB causes larger bone erosions with rim enhancement and thin uniform synovial thickening (Fig. 11), whereas the presence of smaller bone erosions and irregular synovial thickening favours JRA [18]. These features can lead to suspicion of one pathology over the other but are not definitive, and synovial aspiration or preferably synovial biopsy is usually necessary for differentiation. Rice bodies in joint space may be seen in both conditions; these are

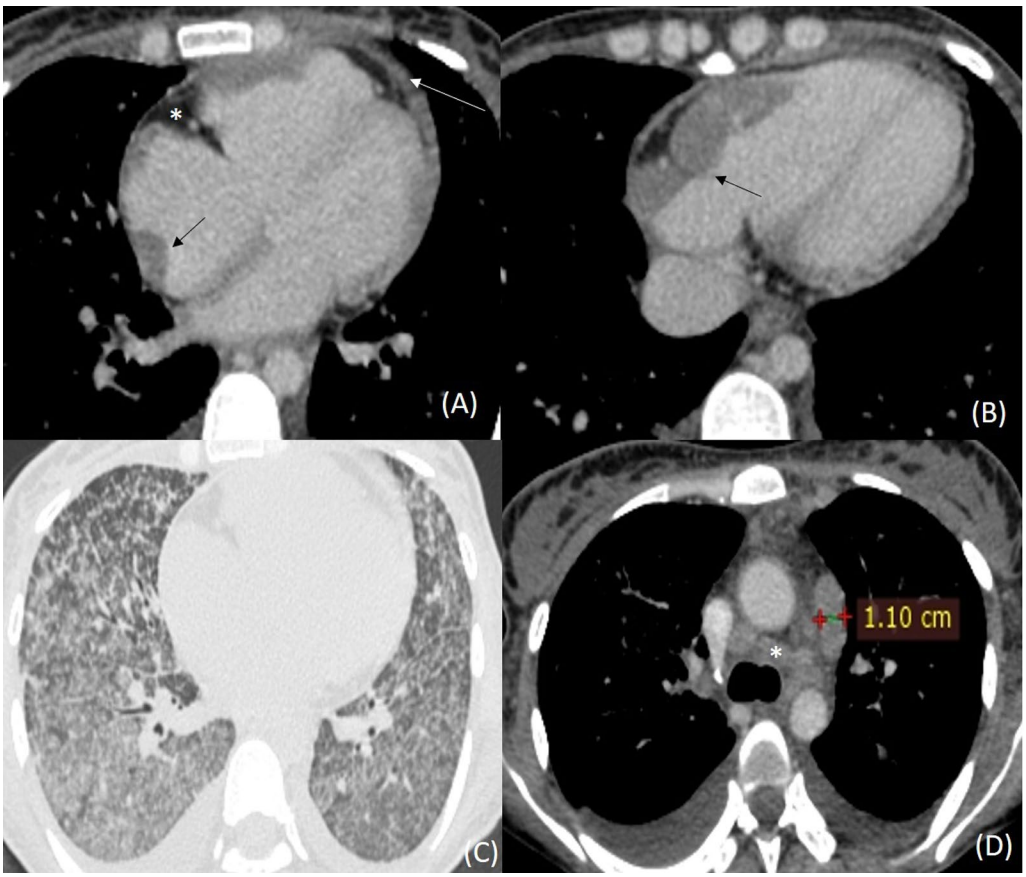

Fig. 6 Cardiac tuberculosis with myocardial and pericardial involvement. CT images of a 42-year-old female show nodular thickening of the right atrial and ventricular myocardium (black arrow in **A** and **B**), thickening of pericardium (white arrow in **A**) and epicardial fat pad (* in **A**), lung window shows miliary nodules in bilateral lungs (**C**) and mediastinal window shows multiple enlarged mediastinal nodes (* in **D**) and bilateral pleural effusion (**D**)

loose bodies and appear hypointense on T1 and T2 without post-contrast enhancement [19]. An extra-articular cold abscess associated with joint arthritis is seen only in TB and, if present, would be diagnostically helpful [18]. Diagnosis in our case was confirmed on synovial biopsy, which showed tubercular granulomas.

Genitourinary

Tuberculosis of urachal cyst The urachal cyst develops because of failure of obliteration of the urachal lumen. Tuberculous infection of urachal cyst is extremely rare with only a few cases described in the literature. It usually occurs because of dissemination from another site [20]. It presents with non-specific imaging features like irregular wall thickening (Fig. 12) of the urachal cyst and is similar to an infected urachal cyst due to any other cause. The indolent nature of the infection with vague pain and longer duration of symptoms as compared to most other causes of infected urachal cyst can point to tubercular aetiology, although the possibility of occult malignancy becomes a major concern in such presentations. Histopathological examination becomes mandatory in most cases for this reason. FNAC of the lesion confirmed tubercular aetiology in our case.

Female genital tuberculosis The fallopian tube is the most commonly affected organ in female genital TB. In the initial stages, TB of the female genital tract presents with pyosalpinx or tubo-ovarian abscess, endometritis, necrotic pelvic lymphadenopathy, ascites and/or irregular peritoneal thickening, as shown in the cases (Figs. 13 and 14). Chronic cases show multiple areas of constriction along fallopian tubes, shrunken irregular uterine cavity secondary to chronic endometritis and calcified pelvic nodes [21]. Endometrial biopsy is necessary for the confirmation of diagnosis as similar features can be seen in pelvic inflammatory disease due to any cause. The presence of complex ascites, irregular peritoneal thickening, necrotic and/or calcified lymphadenopathy, if present, may help in differentiation from other causes of pelvic

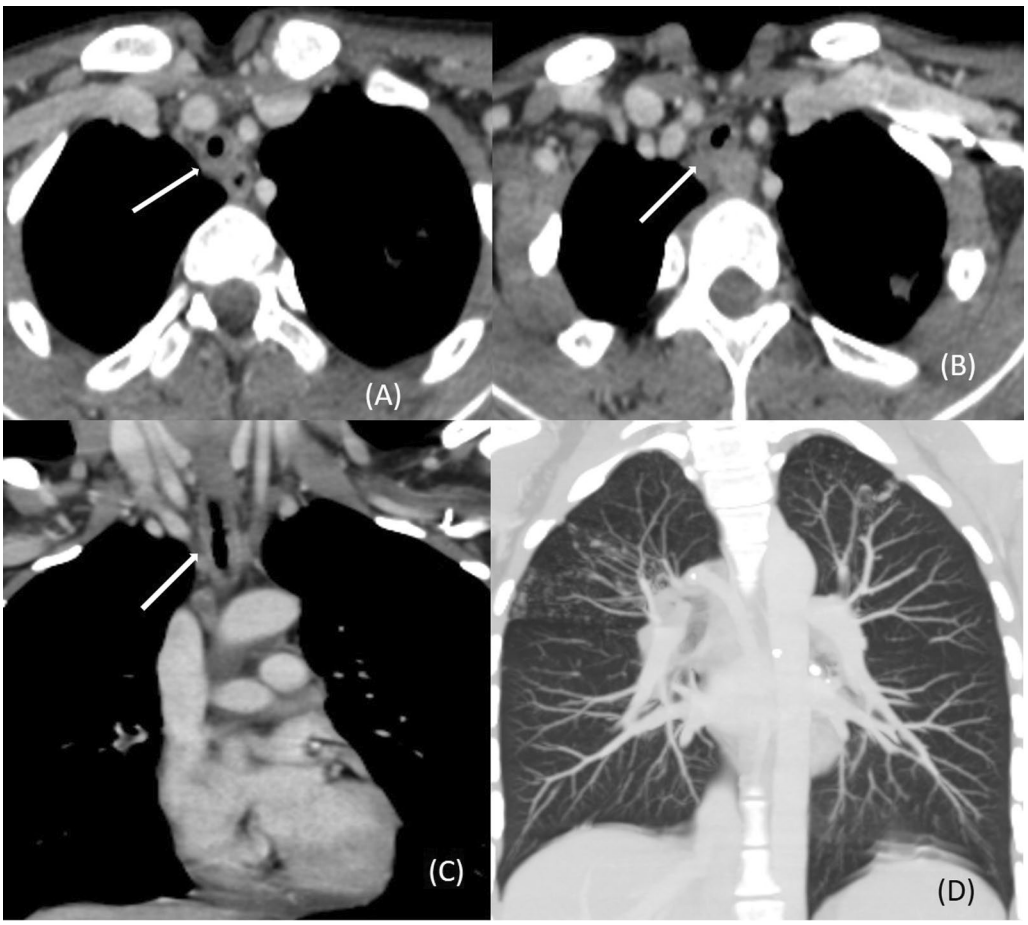

Fig. 7 Tubercular tracheitis. CT images of a 23-year-old female show circumferential wall thickening of distal trachea (white arrow in **A**–**C**) and lung window shows tree-in-bud nodules in the right upper lobe (**D**)

inflammatory disease. Untreated cases often result in chronic pelvic pain with infertility.

Urinary tuberculosis Urinary tract involvement occurs via haematogenous dissemination. The most common site of urinary involvement is the pelvicalyceal collecting system. CT urography shows uneven caliectasis often with infundibular stenosis, moth-eaten calyx, papillary necrosis, irregular wall thickening of renal pelvis, contracted renal pelvis with elevated position due to fibrosis ('hiked-up pelvis'), thickened and straightened ureter ('pipestem ureter'), ureteral stricture, beaded ureter and shrunken bladder ('thimble bladder'). Renal parenchymal involvement may result in non-enhancing tubercular granulomas, cortical abscesses, cortical scarring, parenchymal atrophy and calcification. Tubercular involvement of lymph nodes and adrenal calcification may also occur [22, 23]. These features have been widely described on intravenous urography but not well described on cross-sectional imaging in spite of being more easily appreciated on the same, primarily due to the decline in the incidence of tuberculosis coinciding with the timeline of the development of cross-sectional modalities in the last few decades. However, with the re-emergence of tuberculosis in recent years due to increased survival of patients with HIV/AIDS and advancements in chemotherapy and transplant-related immunosuppressive medication, we believe it is vital that radiologists learn to identify features of genitourinary tuberculosis on cross-sectional imaging in order to avoid diagnostic delays in such patients. The above-mentioned features are well illustrated in CT urography in our cases (Figs. 15 and 16). These features are relatively specific and help in differentiation from other infective causes. Diagnosis in our cases was confirmed by the presence of tubercle bacilli in the morning urine sample. Putty kidney is the sequelae of untreated urinary TB. It is characterized by small, shrunken, non-functional, calcified kidneys due to extensive dystrophic calcifications [15].

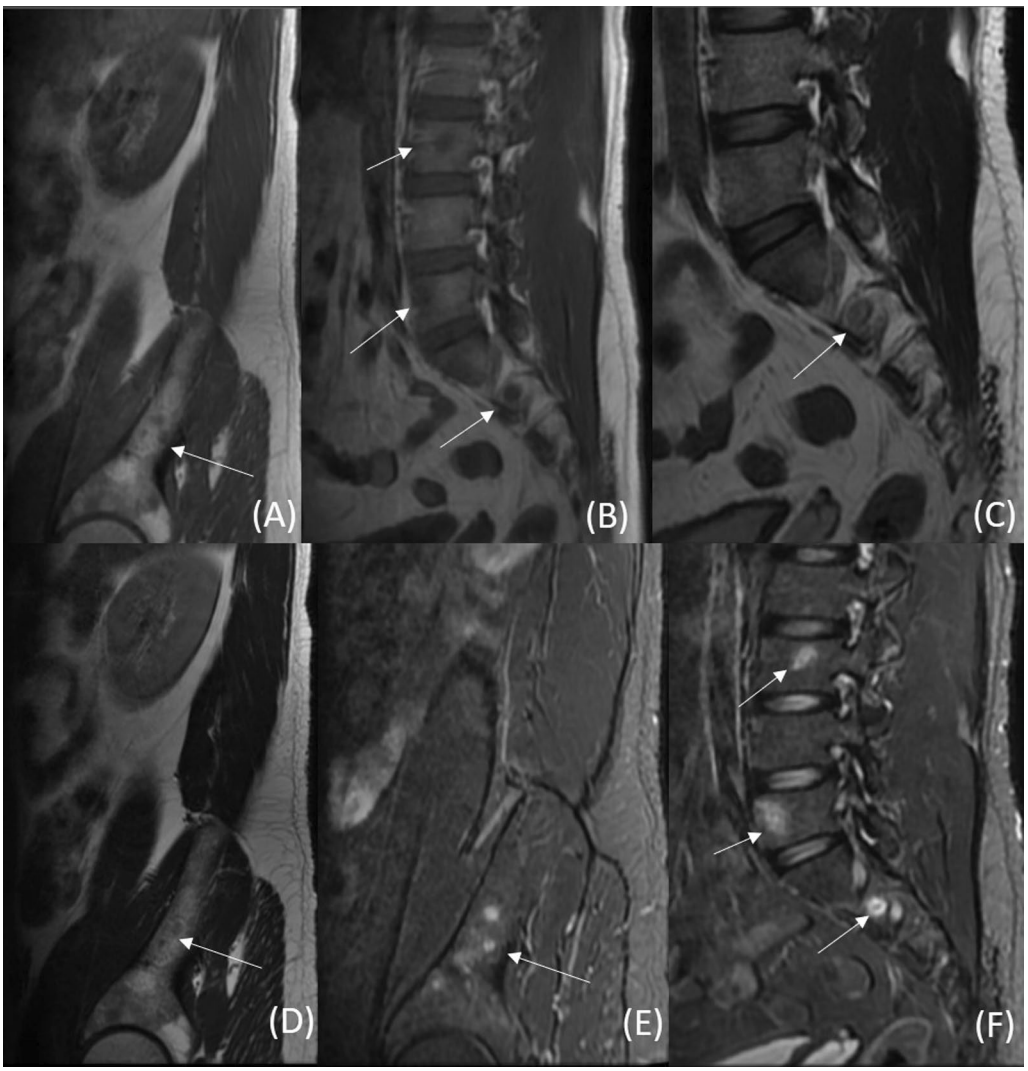

Fig. 8 Multifocal skeletal tuberculosis with subcutaneous tubercular deposits. MRI spine of a 35-year-old male shows multiple lesions (white arrows) in lumbar and sacral vertebrae and iliac bone. The lesions are hypointense on T1W (**A** and **B**) and T2W images (**C** and **D**) and hyperintense on STIR (**E** and **F**)

Gastrointestinal

Abdominal tuberculosis with abdominal wall deposits Abdominal muscle involvement may occur via haematogenous route or direct extension from tubercular lymphadenopathy. There are some case reports of isolated cold abscess involving the anterior abdominal wall [24]. While the extension of abdominal TB to the anterior abdominal wall is relatively well described, the presence of simultaneous deposits in the abdomen and anterior abdominal wall in the absence of contiguous spread is, however, exceedingly rare. We encountered such a case (Fig. 17). Ultrasound-guided FNAC from the lesion in the left rectus abdominis muscle revealed tubercular aetiology. Omental biopsy confirmed intra-abdominal tuberculosis. The presence of imaging features of abdominal TB like ascites, irregular peritoneal and bowel wall thickening, necrotic lymphadenopathy, omental thickening and intra-abdominal tubercular deposits associated with abdominal wall lesions would clearly point to the diagnosis in such cases, although histopathological confirmation is often warranted simply because of the extreme rarity of non-contiguous tubercular involvement of the abdominal wall.

Abdominal cocoon Abdominal cocoon, also known as EPS (encapsulating peritoneal sclerosis), is characterized by fibro-collagenous thickening of peritoneum with resultant bowel encapsulation. It presents with features

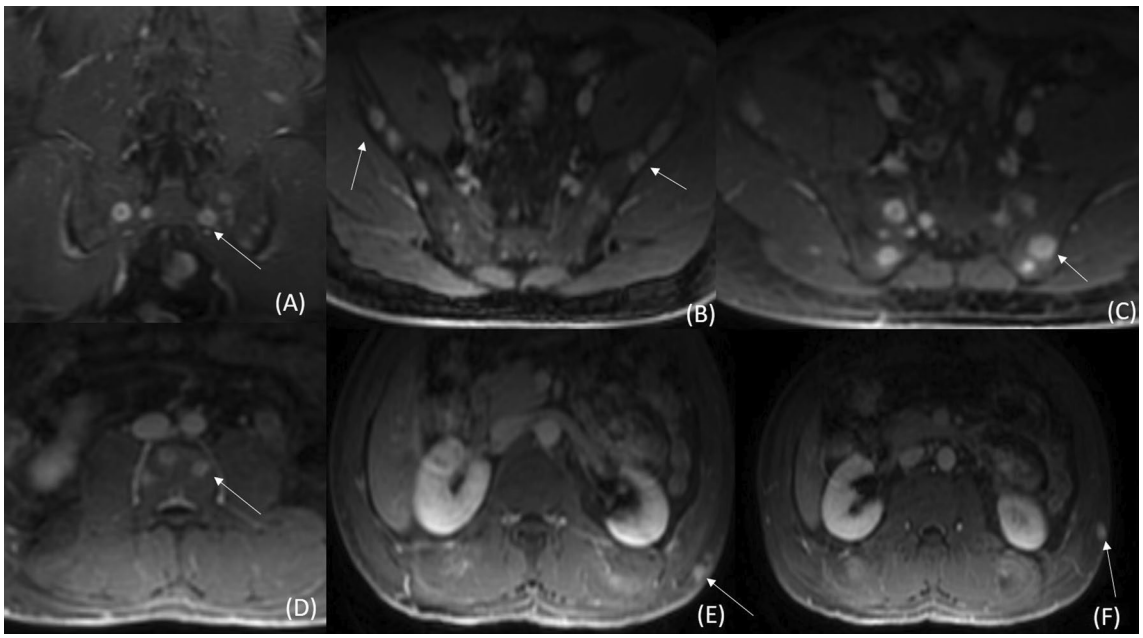

Fig. 9 Multifocal skeletal tuberculosis with subcutaneous tubercular deposits continued. T1 contrast-enhanced images of the same patient show multiple peripherally enhancing lesions (white arrows) in bilateral sacral ala (**A**), iliac blades (**B**), ischium (**C**) and lumbar vertebrae (**D**), homogeneously enhancing lesions are also seen in subcutaneous plane in left flank (white arrow in **E** and **F**)

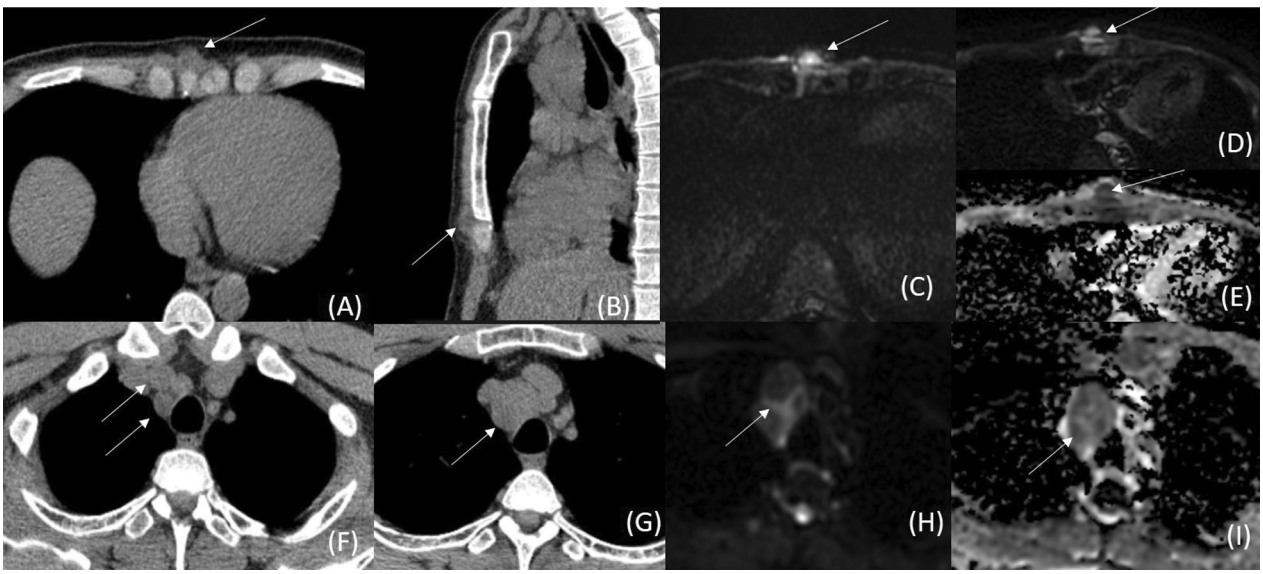

Fig. 10 Tubercular chondro-sternal arthritis with necrotic mediastinal lymphadenopathy. CT images of a 41-year-old male show joint widening and effusion in right 6th chondro-sternal joint with collection extending into subcutaneous plane (white arrow in **A, B**), MRI shows hyperintense collection on STIR (**C**) with diffusion restriction on DWI (**D**) and ADC (**E**). CT images show multiple enlarged mediastinal lymph nodes (arrows in **F**, **G**), MRI shows diffusion restriction in the nodes (arrows in **H**, **I**)

of small bowel obstruction. Peritoneal TB is an important risk factor for EPS. Imaging shows clumped bowel loops with surrounding thickened peritoneum encapsulating the bowel loops [25]. This is demonstrated in our case (Fig. 18). Other common causes of EPS include peritoneal dialysis, ventriculo-peritoneal shunt, meconium peritonitis, malignancies (gastric, pancreatic, renal, ovarian) and previous laparotomy. The clinical history of the patient, as

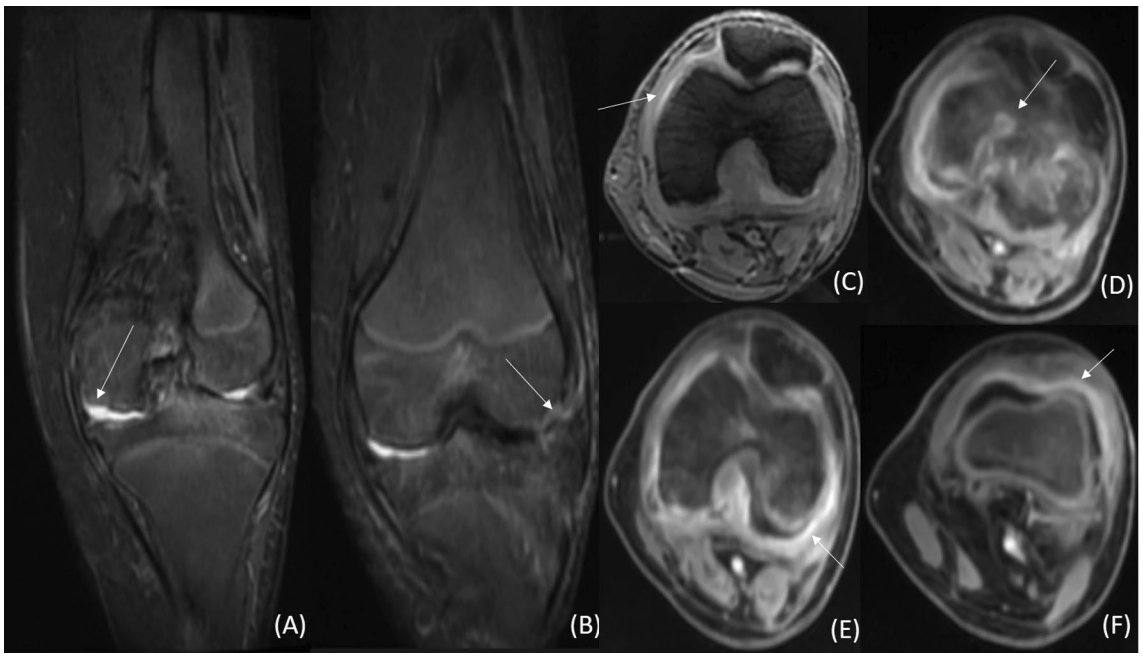

Fig. 11 Tubercular knee arthritis. MRI of a 16-year-old girl shows erosions in medial and lateral femoral condyles on coronal STIR images (white arrow in **A** and **B**), there is mild joint effusion and marrow oedema in tibial plateau and intercondylar notch. Synovial thickening is seen on T2*GRE image (white arrow in **C**), T1 contrast-enhanced axial images show rim enhancement of the erosion (white arrow in **D**) and uniform synovial enhancement and thickening (white arrow in **E** and **F**)

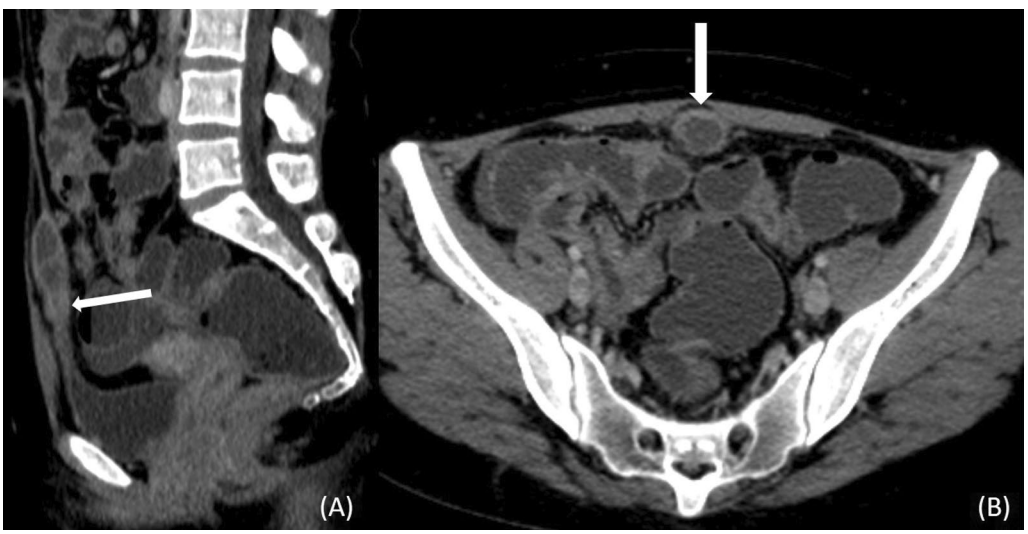

Fig. 12 Tuberculosis of the urachal cyst. CT images of a 26-year-old female show irregular thickening and enhancement of urachal cyst wall (white arrow in **A** and **B**)

well as the presence of other features of abdominal TB like necrotic lymphadenopathy, irregular bowel wall thickening, omental deposits and ascites are useful in differentiation from other causes of EPS. Cytologic evaluation of ascitic fluid provides a definitive diagnosis.

Vascular

Takayasu arteritis associated with tuberculosis Both TB and Takayasu arteritis are chronic inflammatory conditions and are characterized by granulomatous inflammation [26]. TB infection is known to trigger Takayasu

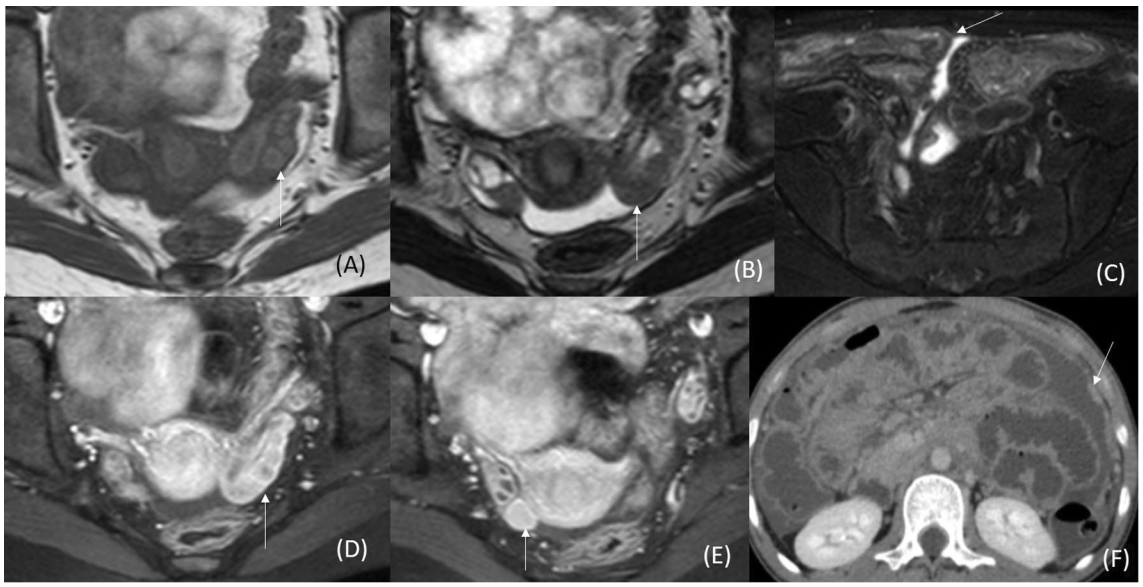

Fig. 13 Female genital tuberculosis—bilateral fallopian tube involvement, ascites and peritoneal thickening. MRI pelvis of a 25-year-old-female shows dilatation of bilateral fallopian tubes with hyperintense wall on T1W image (**A**), wall is hypointense on T2W (**B**), free fluid in pouch of Douglas and endometrial cavity (**B**). T2 fat-supressed image shows ascites (**C**), T1 contrast-enhanced images show peripheral enhancement of bilateral fallopian tubes (**D**, **E**), CT shows irregular peritoneal thickening (**F**)

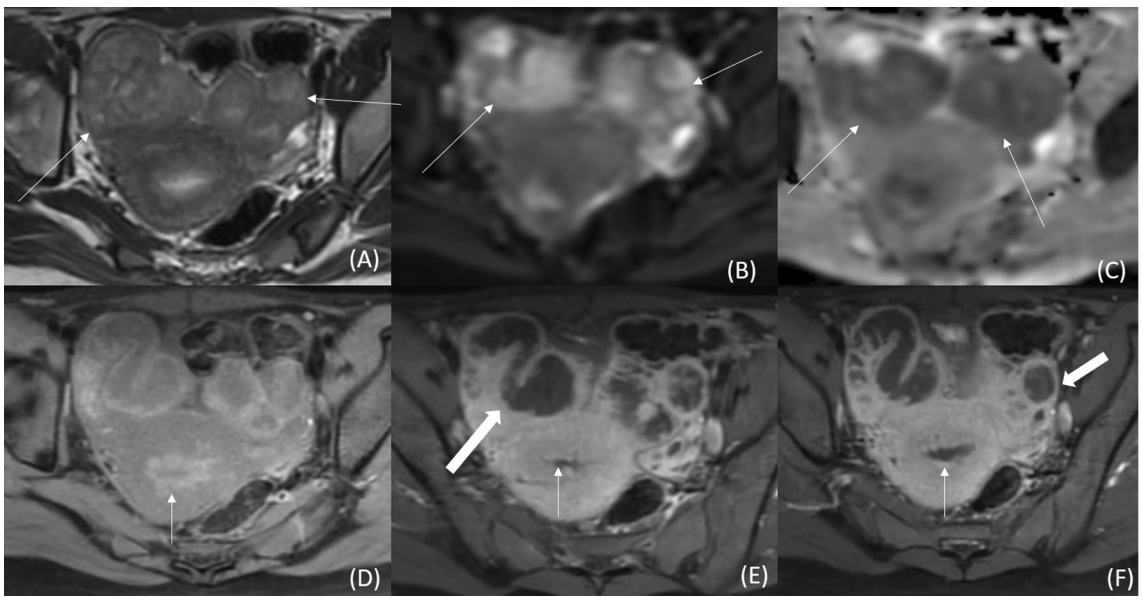

Fig. 14 Female genital tuberculosis—endometritis and bilateral pyosalpinx. MRI pelvis of a 16-year-old girl shows dilatation of bilateral fallopian tubes with hyperintense contents and fluid in endometrial cavity on T2W image (**A**), contents of bilateral fallopian tubes show restriction (**B** and **C**), T1W fat-supressed (**D**) and T1W contrast-enhanced images (**E**, **F**) show irregularity of endometrial lining (thin arrow) and thick peripheral enhancement of bilateral fallopian tubes (solid arrow in **E**, **F**)

arteritis, particularly in young females with a genetic predisposition. A retrospective study by Zang et al. in Chinese population demonstrated that approximately half of Takayasu arteritis patients had TB before the onset of symptoms of Takayasu arteritis, with pulmonary tubercular involvement being the most common [27]. This might

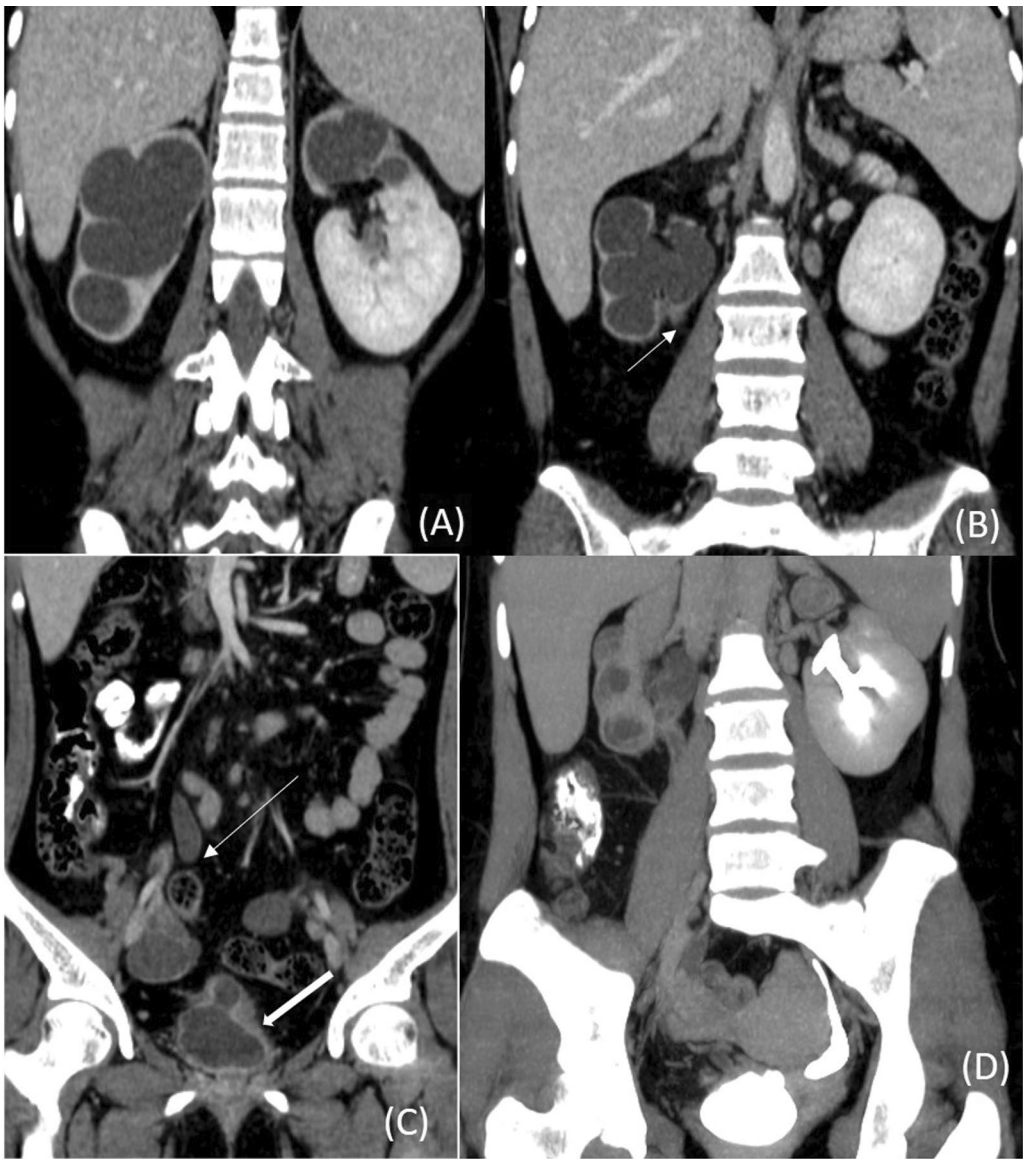

Fig. 15 Urinary tuberculosis involving bilateral kidneys, right pelvi-ureteric junction, right ureter and bladder. CT images of a 44-year-old male show gross hydronephrosis on right and upper pole caliectasis on left side (**A**), there is thickening of right pelvi-ureteric junction (white arrow in **B**) and dilatation of right ureter (thin white arrow in **C**), bladder is contracted, shows irregular wall thickening and a diverticulum (solid white arrow in **C**), there is no contrast excretion on the right side (**D**)

also partly explain the high incidence of Takayasu arteritis in Asia. Although the exact role of TB in the pathogenesis of Takayasu arteritis is unclear, some studies have suggested that cross-reaction between human heat shock protein and tubercular bacilli could result in the autoimmune large vessel vasculitis. Aortitis in Takayasu arteritis can also be due to tuberculin sensitization [26]. CT angiography is usually required for diagnosis and shows wall thickening and wall enhancement with luminal narrowing of large arteries like aorta, subclavian arteries, pulmonary arteries and major branches of the abdominal aorta. We encountered a case of Takayasu arteritis which had involvement of bilateral subclavian arteries, abdominal aorta, left renal artery and superior mesenteric artery (Fig. 19). Additional findings of tree-in-bud lung nodules and calcified mediastinal lymph nodes indicated the presence of chronic infection with tuberculosis. Imaging features of TB associated with typical features of Takayasu arteritis aids in diagnosis. As the typical CT angiography scan protocol for Takayasu arteritis includes

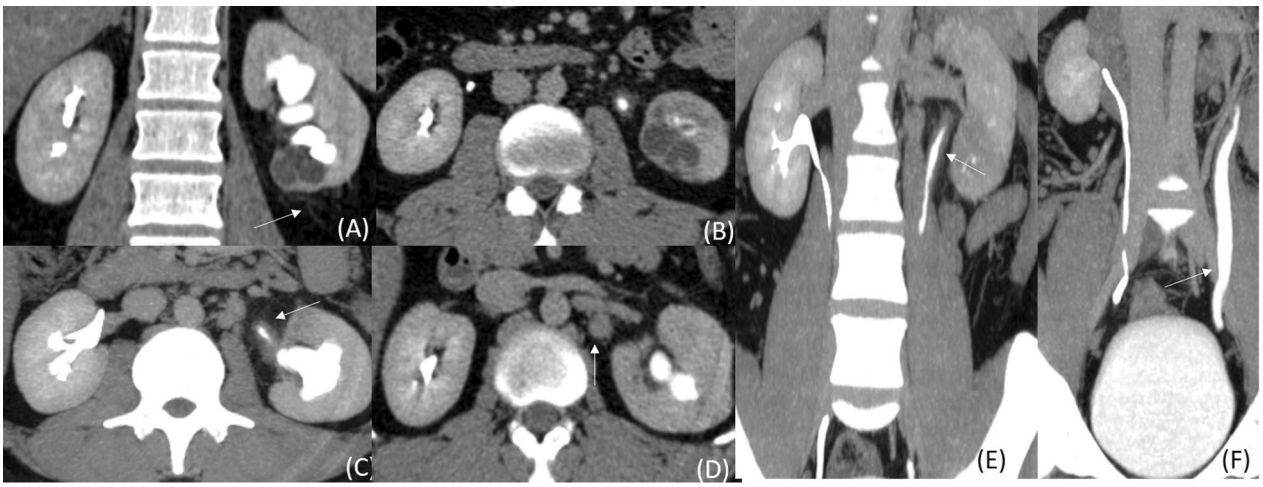

Fig. 16 Urinary tuberculosis involving left kidney, left pelvi-ureteric junction and left ureter. CT images of a 24-year-old male show lower pole caliectasis in left kidney (**A** and **B**) with surrounding fat stranding (white arrow in **A**), there is thickening of pelvi-ureteric junction (white arrow in **C**), an enlarged perinephric lymph node with loss of fatty hilum is seen (white arrow in **D**) with thickening of left upper ureter (white arrow in **E**) and dilatation of lower ureter (white arrow in **F**)

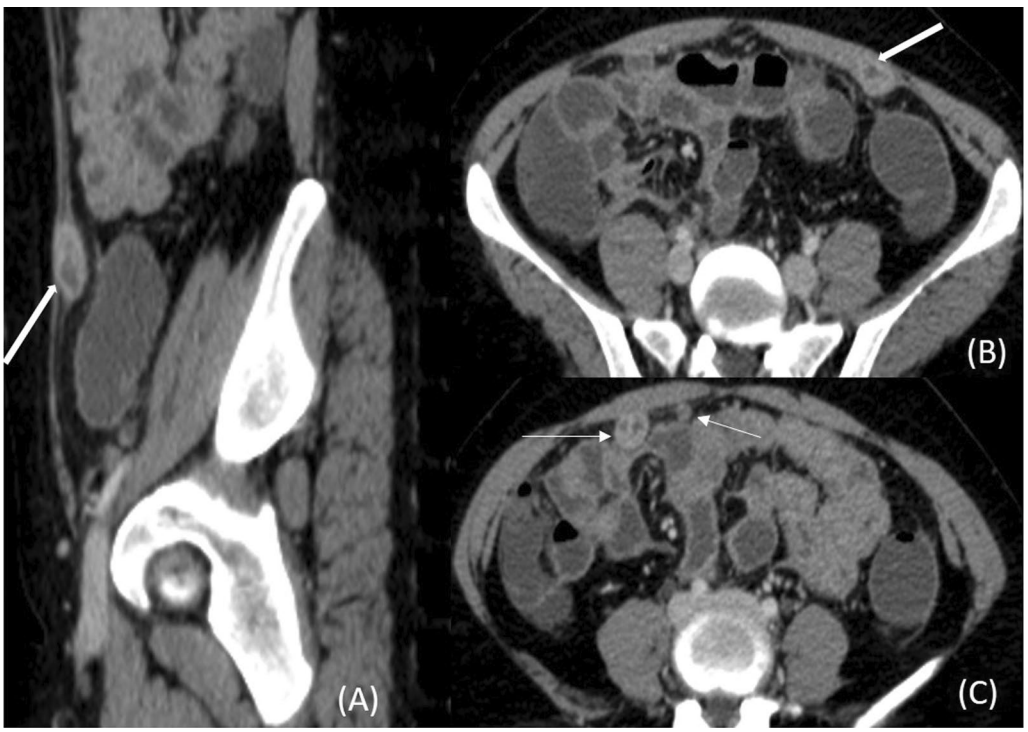

Fig. 17 Abdominal tuberculosis with abdominal wall deposits. CT images of a 28-year-old female show a well-defined peripherally enhancing nodular lesion in left rectus abdominis muscle (solid white arrow in **A** and **B**) with similar morphology lesions in omentum (thin white arrows in **C**)

the lungs and mediastinum, radiologists should always look for evidence of concurrent pulmonary TB as treatment of TB might lead to resolution of the arteritis in such cases.

Miscellaneous

Tubercular paraspinal abscess with fistulous communication with oesophagus and bronchi Tubercular involvement of the oesophagus usually occurs due to the spread of

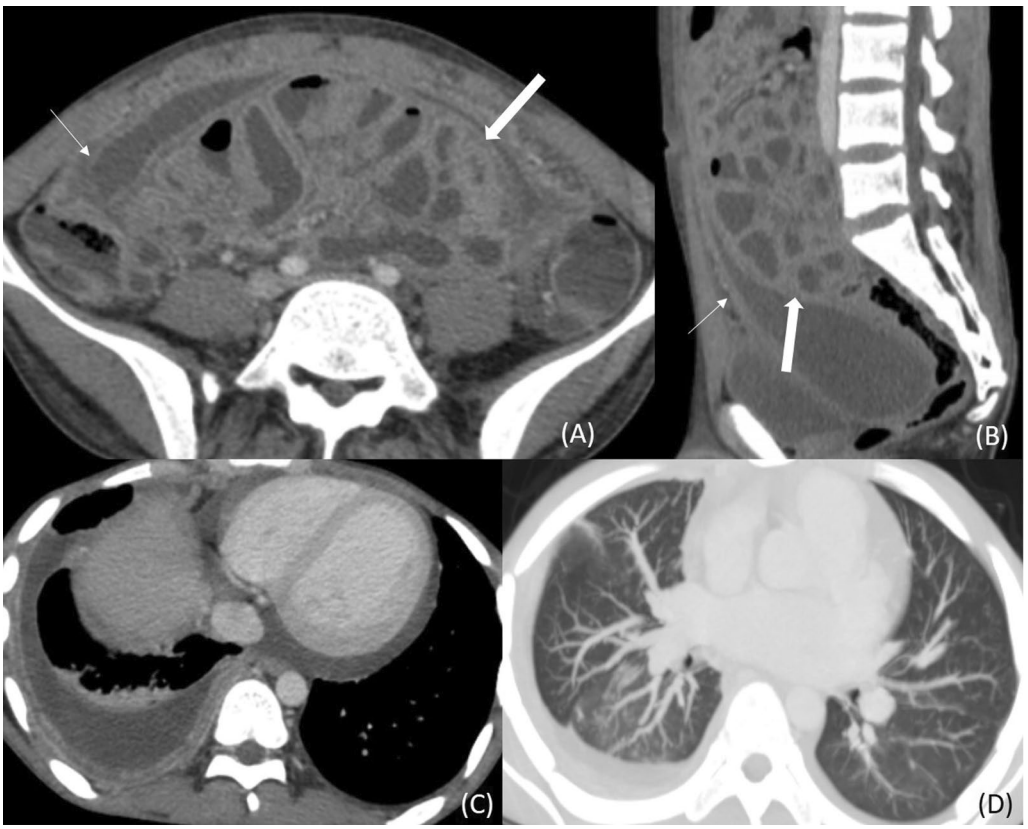

Fig. 18 Abdominal cocoon. Abdominal CT images of 23-year-old male show clumping of small bowel loops in centre of abdominal cavity surrounded by thick and enhancing membrane (solid arrow in **A**, **B**), there is gross ascites and thickening of peritoneal membrane (thin arrow in **A**, **B**), thorax CT shows pericardial effusion and right pleural effusion with thickening of parietal pleura, suggestive of empyema (**C**) and lung window shows randomly distributed nodules in the right lower lobe (**D**)

infection from contiguous structures as primary oesophageal involvement is rare. Usually, this occurs due to mediastinal lymph nodes eroding into the oesophagus, bronchi or trachea. In the case of paraspinal abscesses, there is a tendency towards external fistulization of the abscess into the overlying skin. We encountered a very unusual case of multiple internal fistulizations of a paraspinal cold abscess secondary to TB. Our case initially presented with dysphagia. Imaging showed oesophageal involvement secondary to the formation of a fistula between a paraspinal abscess and the oesophagus. Fistulous communication was also present between the paraspinal abscess and bronchi (Fig. 20), leading to chronic cough. Tubercular paraspinal abscess with simultaneous fistulous communication with the oesophagus and bronchus is, in itself, an extremely rare entity [28]. CT-guided aspiration of paraspinal collection confirmed tubercular aetiology. The presence of spondylodiscitis with paraspinal collection led to the suspicion of Pott's disease in our case with visualization of fistulous involvement of oesophagus and bronchi on cross-sectional imaging.

Reporting protocol for unusual TB cases

As TB is a global problem, it is important for radiologists to be aware of all the necessary information to include in the report of such challenging cases, as this will aid in follow-up and also help the clinician in the selection of the appropriate treatment regime. Therefore, a general and system-wise radiology reporting protocol for TB has been detailed (Fig. 21) outlining essential components to include in the report.

Conclusions

TB can show extremely diverse imaging patterns and can affect any organ. It can mimic various diseases in every organ system and can masquerade as a large number of pathologies. It is important for radiologists to be aware of such unusual patterns and sites of involvement of extrapulmonary tuberculosis in order to facilitate timely and accurate diagnosis of the same for appropriate patient management and prevention of permanent disabling sequelae.

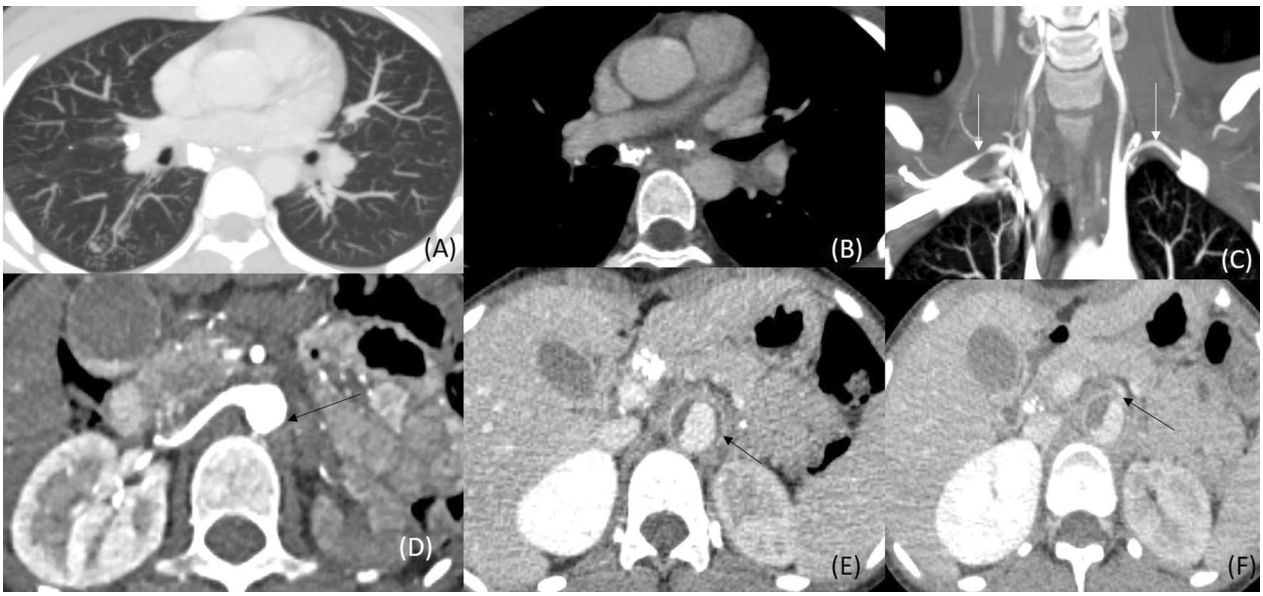

Fig. 19 Takayasu arteritis associated with TB. CT images of a 35-year-old female show tree-in-bud nodules (**A**), calcified mediastinal nodes (**B**), attenuated calibre of bilateral subclavian arteries (**C**), complete luminal obliteration of left renal artery (**D**), wall thickening with luminal thrombus in abdominal aorta (arrow in **E**), calcified periportal nodes (**E**) and narrowing at ostia of superior mesenteric artery (**F**), reduced opacification of left renal parenchyma due to involvement of renal artery (**D**–**F**)

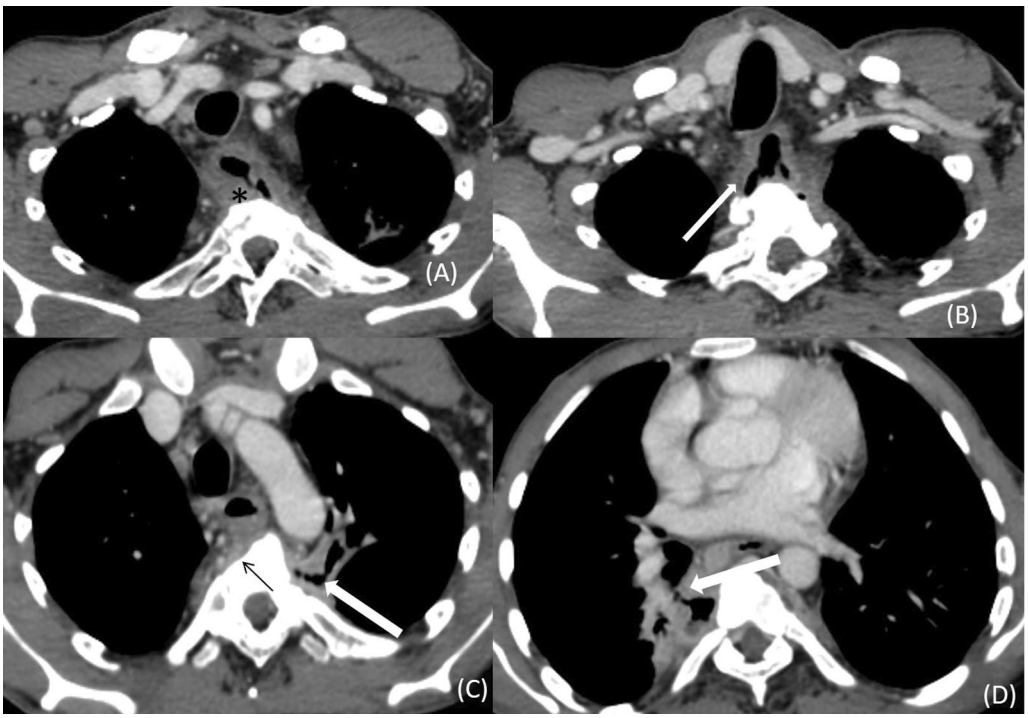

Fig. 20 Tuberculous paraspinal abscess with fistulous communication between oesophagus and bronchi. CT images of a 25-year-old male show a paraspinal abscess (* in **A**) involving multiple vertebral segments, air foci are seen within the abscess at multiple levels (**B**–**D**) with vertebral body irregularity and scalloping (black arrow in **C**), fistulous communication is seen between the paraspinal abscess and cervical oesophagus (white arrow in **B**) and right and left segmental bronchi (white arrow in **C**, **D**)

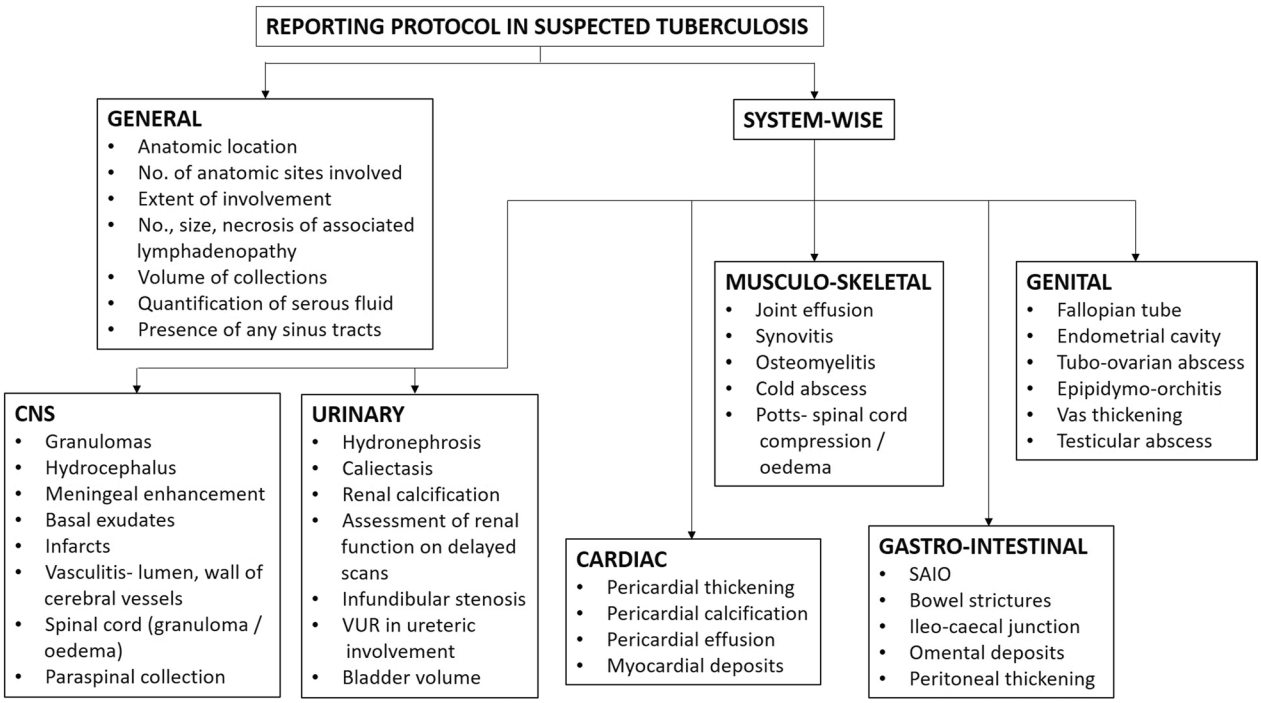

Fig. 21 Flow chart showing general and system-wise reporting protocol in suspected tuberculosis; *VUR* vesico-ureteric reflux, *SAIO* subacute intestinal obstruction

Abbreviations

ACA: Anterior cerebral artery; AFB: Acid-fast bacilli; AIDS: Acquired immunodeficiency syndrome; BMI: Body mass index; CNS: Central nervous system; CSF: Cerebrospinal fluid; CT: Computed tomography; EPS: Encapsulating peritoneal sclerosis; FLAIR: Fluid-attenuated inversion recovery; FNAC: Fine needle aspiration cytology; GPA: Granulomatosis with polyangiitis; HIV: Human immune deficiency virus; ICA: Internal cerebral artery; IMIDs: Immune-mediated inflammatory diseases; JRA: Juvenile rheumatoid arthritis; MCA: Middle cerebral artery; MRI: Magnetic resonance imaging; TB: Tuberculosis; TBM: Tubercular meningitis; TNF-α: Tumour necrosis factor-alpha.

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Author contributions

Dr. A contributed to the conception and design, data acquisition, literature search and manuscript drafting. Dr. V contributed to the conception and design, literature search, manuscript drafting, editing and revision. Dr. A contributed to the literature search, manuscript drafting, editing and revision. All authors approved the final version to be published.

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Availability of data and materials

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Declarations

Ethics approval and consent to participate

Ethics Committee approval was obtained from the Institute Ethics Committee, Vardhman Mahavir Medical College and Safdarjung Hospital. Written informed consent was obtained from all the participants (parents in case of minors) for participation in the study and anonymous publication of data.

Consent for publication

Written informed consent was obtained from all the participants (parents in case of minors) for the anonymous publication of clinical data and study images. Names or other data revealing the patient identity or otherwise compromising anonymity are not present in the manuscript or images.

Competing interests

The authors declare that they have no competing interests.

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