Tumor-like abdominal tuberculosis: a real challenge for radiologist!

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Learning objectives

Abdominal tuberculosis (TB) is an uncommon condition in the western countries, but there has been a resurgence of the disease, becoming an endemic disease in most of the developing countries. Although abdominal tuberculosis is usually secondary to pulmonary tuberculosis, radiologic evaluation often shows no evidence of lung disease making its positive diagnosis difficult. Abdominal TB is a diagnostic challenge, particularly in the absence of evidence of pulmonary infection. It may mimic many other abdominal diseases particularly tumors.

In this educational exhibit and through a 10 patient’s series collected in our institution during three years (2011-2013), we propose to:

• To illustrate ultrasounds and computed tomography findings of tumor-like abdominal tuberculosis with emphasis in the latest.
• To precise its semiological features those help to establish positive diagnosis and differentiate it from other inflammatory and neoplastic diseases.
Background

Abdominal tuberculosis represents 10-15% of all tuberculosis sites. As tuberculosis can affect any abdominal organ, emphasis is placed to lymphadenopathy, peritonitis, ileocecal involvement and solid viscera disease (liver, spleen and pancreas). Historically, it was recognized that abdominal tuberculosis could mimic many other disease, especially tumor-like form. Its diagnosis is difficult, based on imaging, histological and bacterial proofs.

I. EPIDEMIOLOGICAL FEATURES

1. Age and sex:

Abdominal TB tends to affect a population in the third and fourth decades of life and it is more frequent in women.

2. Prevalence:

Abdominal tuberculosis is rare even in endemic areas. Indeed, it is the rarest localization of extra pulmonary TB (only 10-15% of extrapulmonary forms). Actually, its prevalence is clearly increasing; immigration HIV infection, economic recession and increasing resistance to anti-tuberculous treatment are deemed to be the reasons of resurgence of the disease worldwide.

However, the frequency of tumor-like forms in the abdominal tuberculosis is difficult to assess. Indeed, a pseudo-tumoral aspect is reported in 5% of cases.

II. PHYSIOPATHOLOGY:

Tubercle bacilli infect the gastrointestinal tract after ingestion of infected sputum or by ingestion of infected milk. The bacilli penetrate the mucosa and infect the submucosal lymphoid tissue, resulting in the epithelioid tubercle. Mucosal sloughing and ulceration occur 2-4 weeks later and results in the ulcerative form of the disease, which is most common. The ulcers typically are small and multiple with irregular margins. The surrounding mucosa shows considerable thickening. With progression there is granuloma formation, caseous necrosis and cicatrisation.

A less common hypertrophic form is characterized by abundant inflammatory response and reactive tissue producing a multinodular mucosal pattern or neoplasm-like mass.

Disseminated infection to abdominal viscera can also occur by hematogeneous and lymphatic routs from distant source of infection, such as the lungs.
III. CLINICAL FEATURES

Clinically, abdominal tuberculosis and particularly its tumor-like form are characterized by atypical and confusing symptoms. It may be a general condition decline, abdominal pain, fever (inconstant), a palpable abdominal mass or organomegaly. Can also be detected inguinal and epitrochleen lymphadenopathies.

These clinical symptoms are suggestive, particularly in elderly patients, neoplastic disease

IV. Positive diagnosis:

Knowing evoke the diagnosis of TB is the precondition for taking timely appropriate care. In front of highly suggestive aspect of imaging, an etiologic assessment should begin to detect other sites may reinforce the diagnosis.

Confrontation with clinical features (signs of tubercular impregnation, chronic diarrhea ...) and biological (IDR research Mycobacterium tuberculosis in duodenal fluid...) is required. If doubt persists guided biopsy under ultrasound or CT control with histopathological study would permit establishing positive diagnosis. In case of negative puncture, laparotomy is often the only recourse.
Findings and procedure details

Abdominal tuberculosis involves by decreasing order: **intraperitoneal lymph nodes, peritoneum, gastrointestinal tract** and finally **intraperitoneal solid organs (liver, spleen and pancreas)**.

Regardless of involving organ, the most common radiological aspect was **widely necrotic solid mass**.

I. Tuberculous lymphadenopathies

Lymphadenopathy is the most common manifestation of abdominal tuberculosis (seen in 55%-66% of patients), and is usually associated with gastro-intestinal tuberculosis and less commonly with peritoneal or solid organ involvement; however, it can also be the only sign of the disease, especially in the periportal region.

The characteristic pattern is mesenteric, porta hepatis and peripancreatic lymph node group enlargement, with multiple groups affected simultaneously. This distribution is explained by the drainage of the ingested infected material by the lymphatics of the ileocecal region, jejunum, ileum and right side of the colon.

- **At US: (fig. 1)**

  Lymph nodes are either discrete or appear as matted conglomerate masses. Enlarged nodes usually contain central hypoechoic areas underlying its necrotic nature.

- **At CT: (fig. 2, 3, 4 and 5)**

  Lymph node enlargement is better assessed on CT than on US. Involved nodes are usually enlarged with hypoattenuating centers and hyperattenuating enhancing rims at CT; findings that are characteristic of caseous necrosis (fig 2 and 3).

  Other nodal patterns include conglomerate mixed-attenuation masses, enlarged homogeneous-attenuation nodes, and an increased number (more than three) of normal or mildly enlarged homogeneous nodes; lymph node calcifications may be also present and are characteristic for sequelae of old tuberculosis (fig. 4).

Although peripheral enhancement is very characteristic of lymphadenopathy TB, it is not a pathognomonic sign, as it is also noted in other processes particularly **lymphoma and metastatic malignancy**. Also, peripancreatic location especiall peri-cephalic necrotic adenomegalies can be confusing with **cystic pancreatic tumor** such as mucinous cystadenocarcinoma or serous (fig. 5).
For this purpose, some semiotic features are required to establish the differential diagnosis:

- Absence of compression of biliary and pancreatic ducts or portal system,
- Presence of calcifications,
- The characteristic location (peri-pancreatic and porto-mesenteric) and association with other tuberculous locations (ileocaecal or lung).

II. Tuberculous Peritonitis (fig. 6, 7 and 8)

Peritonitis is the second most common clinical manifestation of abdominal tuberculosis, affecting approximately 35% of all patients.

On imaging, peritoneal involvement is subdivided into three main types: wet, fibrotic-fixed, and dry although there is considerable overlap in their CT appearances. Tumor-like peritonitis is characterized particularly by fibrotic-fixed form. Fibrotic type peritonitis accounts for 60% of cases of peritonitis and is characterized by large omental and mesenteric "cakelike" masses with tethered and matting of bowel loops.

At CT, it manifests as mottled low-attenuation masses with nodular soft-tissue thickening and mesenteric infiltration (fig. 6).

Peritoneal irregular thickening and enhancement associated with nodular implants are uncommon and are more suggestive of peritoneal carcinomatosis. In addition, caked type, which is uncommon in tuberculosis, should lead to evoke primordially carcinomatous peritonitis. The clinical and radiological differentiation of tuberculous peritonitis and peritoneal carcinomatosis can be challenging, and has a crucial importance, as peritoneal tuberculosis, if promptly diagnose, has an effective therapy and good survival rate.

Although most analyzed CT findings overlap these diseases, some imaging features increase the ability of distinguishing tuberculous peritonitis from peritoneal carcinomatosis:

- Mesenteric macronodule,
- Relative regularity of infiltrated omentum covered by a thin line,
- Masses with low-density center,
- Association with widely necrotic adenopathy and/or Calcification and hepatosplenic lesions.
III. Intestinal tuberculosis (fig. 9, 10 and 11)

Intestinal tuberculosis is rare; when present, however, it almost always involves the ileocecal region (90% of cases), usually both the terminal ileum and the cecum. This is due to the abundance of lymphoid tissue.

- **At US** (fig. 9 and 10): Ultrasonography usually reveals uniform and concentric bowel wall thickening in most patients. Cecal involvement can be confusing to matted hypoechoic masse and should evoke neoplastic process.

- **At CT** (fig. 11 and 12): CT is an excellent method for evaluation of the pathologic process morphology. Characteristic CT features include asymmetric thickening of the ileocecal valve and medial wall of the cecum, shortening and retraction of ascending colon, exophytic extension engulfing the terminal ileum, widely necrotic pseudomasses and massive lymphadenopathy. Skip areas of concentric mural thickening with associated luminal narrowing with or without proximal dilatation can occur elsewhere in the small bowel, findings that strongly suggest tuberculosis in the presence of ileocecal involvement.

In advanced process, asymmetric thickening of the ileocecal valve and medial wall of the cecum associated with large lymph nodes forms a heterogeneous soft-tissue mass that envelops the terminal ileum. Adherent loops and mesenteric thickening centered at the ileocecal valve can also together form part of this soft-tissue mass.

Some semiotic features help to differentiate ileocecal TB from neoplastic disease:

- **Predominance in the medial wall of the caecum**
- **Regular thickening**
- **Cecal retraction and shortening (better seen with barium enema)**
- **Necrotic nature of the thickening**
- **Association with widely necrotic and lymphadenopathies**
- **Association with other tuberculous location (pulmonary miliary (fig. 13)**

IV. Solid organ tuberculosis
Tuberculous involvement of solid viscera is most likely secondary to hematogenous dissemination of the primary form of the disease. Indeed, hepato-splenic TB is common in miliary form.

- Hepato-splenic TB (fig. 14, 15 and 16)

Hepato-splenic tuberculosis develops under micronodular (miliary) or macronodular form. Miliary hepato-splenic usually occurs in association with miliary pulmonary tuberculosis. On CT scans, the disease manifests as innumerable tiny, low-attenuating foci at CT.

The macronodular form is rare and manifests as diffuse liver or splenic enlargement with multiple low-attenuation lesions or a single tumor-like mass. On contrast-enhanced images, these lesions usually appear as infiltrative widely necrotic mass. At early-stage, lesions may demonstrate central enhancement whereas more advanced lesions may demonstrate calcifications.

The differential diagnosis of the miliary form includes metastases, fungal infection, sarcoïdosis, and lymphoma. The macronodular form can be mistaken for metastases, primary malignant tumor, or pyogenic abscess. Distinctive semiotic elements are:

-Micronodular form

-Presence of calcifications

-Association with peri-portopancreatic necrotic lymphadenopathies

-Association with pulmonary milary

- Pancreatic TB (fig. 17)

Pancreatic tuberculosis is a rare entity and is usually diagnosed as a solid tumor. Although it usually occurs in miliary form of tuberculosis, it may be the only site of reactivation of an old TB.

Pancreatic tuberculous involvement appears as well-defined hypoechoic lesion in US and as hypodense necrotic lesion within an enlarged pancreatic head on CT scan.

The diagnosis of pancreatic TB should be obtained by percutaneous biopsy as pancreatic malignancies. Adenocarcinoma, cystic tumor, abscesses and chronic pancreatitis are considered in the differential diagnosis.
Fig. 1: Abdominal US (a) and Power Doppler (b) images: Tuberculous lymphadenopathies: Conglomerate widely necrotic mesenteric lymph nodes (arrows). Note the tuberculous ileal involvement mimicking malignancy (asterix).

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Fig. 2: Abdominal enhanced CT with axial (a and b) and coronal (c) reconstruction: Tuberculous lymphadenopathies: Conglomerate widely necrotic porta hepatis, peripancreatic and mesenteric lymph nodes (arrows). Note the absence of compression of intra and extra hepatic biliary ducts.
Fig. 3: Abdominal enhanced CT with axial (a and b) and coronal (c) reconstruction: Tuberculous lymphadenopathies: Conglomerate widely necrotic mesenteric lymph nodes (arrows). Note associated peri-portopancreatic lymphadenopathies
**Fig. 4:** Abdominal enhanced CT with axial (a) and coronal (b) reconstruction: Tuberculous lymphadenopathies: Massively calcified peri-portals nodes (arrows) associated with multiple splenic calcified granulomas (arrows head).

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**Fig. 5:** Abdominal enhanced CT: Tuberculous peripancreatic lymphadenopathies: Conglomerate peripancreatic lymphadenetis mimicking a cephalic cystic pancreatic tumor (arrow). Note the association with another peripancreatic necrotic lymphadenetis (arrow head).

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**Fig. 6:** Abdominal enhanced CT: Fibrotic tuberculous peritonitis: Nodular thickening of parietal peritoneum at the right paracolic gutter (arrows) associated with mesenteric infiltration (asterix) and caeacal involvement (arrow head).

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**Fig. 7:** Abdominal enhanced CT: Dry tuberculous peritonitis: Regular thickening of parietal peritoneum at left paracolic gutter (arrows) associated with mesenteric infiltration and thickening (arrows heads) and effusion peritoneal (asterix).

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Fig. 16: Abdominal enhanced CT with axial (a and b) and coronal (c) reconstruction. Hepatosplenic calcified miliary.

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**Fig. 15:** Abdominal enhanced CT with axial (a, b, c and d) and coronal reconstruction (e) Hepatosplenic micornodular tuberculosis: Tiny low-attenuating hepatosplenic foci (arrows heads). Note ileocecal (arrow) and ganglionic involvement (asterix)

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**Fig. 13:** Abdominal enhanced CT (a) and thoracic CT (b): Tumoral-like ileo-cecal tuberculosis associated with pulmonary miliary: Irregular and mixed enhanced thickening of cecal wall (arrows) associated with pulmonary miliary.

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**Fig. 8:** Abdominal enhanced CT: Fibrotic tuberculous peritonitis with omental cake aspect (arrows) and tethered bowel loops (arrows heads). Note peritoneal infiltration (asterix)

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**Fig. 9:** Abdominal US (a) and Power Doppler (b) in transverse section: Tuberculous tumor-like involvement of caecum: Hypoechoic regular thickening of cecal wall mimicking a large solid mass (arrows). At Power Doppler, note hypervascularisation of the involved area and pericaecal soft tissue (arrows heads)

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**Fig. 10:** Abdominal US in transverse section: Tuberculous tumor-like involvement of terminal ileum: Hypoechoic regular and circumferential thickening of ileal wall mimicking a large solid mass (arrows). Note mesenteric infiltration showed as hyperechoic aspect of peri-ileal soft tissue (asterix)

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Fig. 12: Abdominal enhanced CT with axial (a and b) and coronal (c) reconstruction: Tumoral-like ileo-cecal tuberculosis: Irregular and mixed enhanced stenosing thickening of cecal wall predominantly of the valvular area (arrows) with distension of terminal ileum (arrows heads). Note the "feces sign" in the ileal terminal loop demonstrating chronic nature of the stenosis (asterix)

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Fig. 11: Abdominal enhanced CT with axial (a and b) and coronal (c) reconstruction: Tumoral-like cecal tuberculosis: Irregular and large hypoattenuating thickening of cecal wall predominantly of the valvular area mimicking widely necrotic tumoral mass. Note associated retrocecal fascia infiltration (arrows head).

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**Fig. 14:** Abdominal enhanced CT: Hepatic macronodular tuberculosis: Infiltrative widely necrotic hepatic masses mimicking liver cystic or necrotic metastasis (arrows heads). Widely necrotic peri pancreatic lymphadenopathy similar to cystic pancreatic tumor (arrow). Note ileocecal tuberculous involvement (asterix)

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Fig. 17: Abdominal enhanced CT: Tumor-like pancreatic tuberculosis: Hypoattenuating well defined corporeal pancreatic pseudomass with swelling of the pancreatic gland. Note associated peripancreatic widely necrotic lymphadenopathy mimicking an adenocarcinoma perivascular involvement.

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Conclusion

Diagnosis of abdominal tuberculosis is often difficult especially in its pseudo-tumoral form. A high level of clinical suspicion is required, mainly in high-risk population. Although there are no pathognomonic radiological findings, features that suggest the correct diagnosis must be well recognized.
References


