

Chapter 102

Abdominal Tuberculosis — Current Concepts in Diagnosis and Management

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INTRODUCTION

Abdominal tuberculosis is a most common type of extra-pulmonary tuberculosis, comprising of tuberculosis of gastrointestinal tract, peritoneum, omentum, mesentery and its lymph nodes and other abdominal organs such as liver, spleen and pancreas. The extrapulmonary tuberculosis involves 11-16% of all patients of tuberculosis out of which 3 to 4% belong to abdominal tuberculosis¹.

Extrapulmonary tuberculosis is common amongst HIV-infected patients.^{2,3} This co-existence of TB and HIV / AIDS has led to the resurgence of extrapulmonary tuberculosis (EPTB) in the developing and under-developed countries.² In various series, extrapulmonary tuberculosis alone or in association with pulmonary disease has been documented in 40-60% of all cases with HIV co-infected individuals. The pattern of presentation of abdominal tuberculosis has dramatically changed with increasing incidence of HIV coexistence, making the diagnosis of extrapulmonary tuberculosis in HIV-infected persons difficult.

Abdominal tuberculosis can mimic a variety of other abdominal conditions/diseases and only a high degree of suspicion can help in the diagnosis otherwise it is likely to be missed or delayed resulting in high morbidity and mortality.

Pathophysiology of Abdominal Tuberculosis

Abdominal tuberculosis can occur primarily or it can be secondary to a tubercular focus elsewhere in the body. Gastrointestinal tuberculosis occurring due to ingestion of milk or food infected with *Mycobacterium bovis* can result in primary intestinal tuberculosis, but it is now-a-days rare. Infection by *Mycobacterium tuberculosis* causing abdominal tuberculosis is acquired in following ways:

- Dissemination of primary pulmonary tuberculosis in childhood
- Swallowing of infected sputum in active pulmonary tuberculosis
- Hematogenous dissemination from a focus of active pulmonary tuberculosis or military tuberculosis
- Mycobacteria can spread from infected adjacent organs like fallopian tubes
- Intestinal infection can occur by lymphatic spread from infected mesenteric lymph nodes
- Mycobacteria can also get disseminated through bile from tubercular granulomas of the liver.

Sites of Involvement in Abdominal Tuberculosis³

1. Gastrointestinal tract
2. Peritoneum, e.g. ascites
3. Lymph nodes
4. Solid organs, e.g. liver, spleen and pancreas.

Gastrointestinal tuberculosis constitutes 70-78% cases of abdominal tuberculosis⁴. Ileocecal area is the most commonly involved site due to the abundance of lymphoid tissue (Peyer's patches) followed by the colon and jejunum^{4,5}. Rarely tuberculosis may also involve stomach, duodenum and esophagus. The three characteristic intestinal lesions produced in tuberculosis include (i) ulcerative, (ii) hypertrophic and (iii) stricturous or constrictive⁵. A combination of these three morphological forms can also occur such as ulcero-constrictive or ulcerohypertrophic. Strictures are usually produced as a result of cicatricial healing of ulcerative intestinal lesions. Most cases of gastrointestinal tuberculosis have associated lymph node and peritoneal involvement.

Peritoneal involvement occurs in 4-10% patients of extrapulmonary tuberculosis (EPTB)⁶. Tubercular peritonitis follows either the direct spread of tuberculosis from ruptured lymph nodes and intra-abdominal organs or hematogenous seeding. Peritoneal involvement may be in the form of peritoneal adhesions or exudative fluid in the peritoneal cavity (ascites). The coexistence of cirrhosis in patients with tubercular peritonitis complicates the diagnosis.

Tubercular lymphadenitis accounts for about 25% cases of extrapulmonary tuberculosis. The lymph nodes disease is particularly frequent in younger age groups and more frequent in HIV-infected patients. Once caused mainly by *M. bovis*, tubercular lymphadenitis now-a-days is largely to *M. tuberculosis*². The nodal involvement in abdominal tuberculosis is mainly mesenteric (tabes mesenterica) or retro-peritoneal⁵. The lymph nodes may show caseation or calcification. Intestinal, nodal and peritoneal tuberculosis may also occur in varying permutations and combinations.

The involvement of liver and spleen in tuberculosis occurs as a part of disseminated and military tuberculosis and is usually granulomatous. The macronodular form of hepatosplenic tuberculosis is an uncommon form of disseminated tuberculosis (Fig. 1)⁷.

Gastric tuberculosis though rare but is commoner than esophageal, duodenal, appendicular and anal tuberculosis⁶.

CLINICAL MANIFESTATIONS^{4,10}

In order of frequency, abdominal tuberculosis manifests as tubercular lymphadenitis, peritonitis and hepatosplenic or pancreatic tuberculosis. The disease may present at any age but commonly seen in young adults⁸. In children, peritoneal and nodal form of tuberculosis is more common than intestinal tuberculosis⁹. The modes of presentation may vary from asymptomatic disease (an incidental finding on laparotomy) to acute, acute on chronic or chronic symptomatic disease. The clinical manifestations depend on the site and type of involvement. The symptomatology mainly includes (i) constitutional symptoms in about one-third of patients (fever, malaise, anemia, night sweats, loss of weight, weakness), and (ii) local symptoms and signs referable to the site involved. The clinical presentation of intestinal tuberculosis is tabulated (Table 1).

A physical examination of abdomen may show signs of ascites, lump in the abdomen or visible peristalsis with dilated loops of gut. However, abdominal examination may be unrewarding in a large number of cases.

Because of varied clinical manifestations, one or the other form of abdominal tuberculosis may mimic any one of the followings:

1. Malignant neoplasms, e.g. lymphoma, carcinoma
2. Inflammatory bowel disease

Table 1: Clinical presentations of intestinal tuberculosis

Type	Clinical presentations
1. Ulcerative	Chronic diarrhea, malabsorption, intestinal perforation (occasional). Rectal bleeding is rare but reported occasionally in colonic tuberculosis.
2. Hypertrophic	Intestinal obstruction or an abdominal (ileocaecal) lump
3. Stricture / constrictive	Recurrent subacute intestinal obstruction (e.g. vomiting, constipation, distention and colicky pain). There may be associated gurgling sounds or feeling of moving ball of wind in the abdomen and visible distended intestinal loops with visible peristalsis. These symptoms get relieved with passage of flatus / stool. Sometimes, acute intestinal obstruction may develop.
4. Anorectal	Stricture or fistula-in-ano
5. Gastroduodenal	Peptic ulcer with or without gastric outlet obstruction or perforation
6. Liver and spleen	Hepatosplenomegaly usually a part and parcel of disseminated tuberculosis is accompanied with fever, night sweats and decreased or loss of appetite. Microscopic involvement shows granulomatous hepatitis.
7. Peritoneum	Abdominal distention and ascites, sometimes there may be a soft cystic lump due to loculated ascites
8. Lymph node	As a mass or lump of matted lymph nodes in the central abdomen or as vague abdominal pain. There is associated fever, night sweats and malaise.

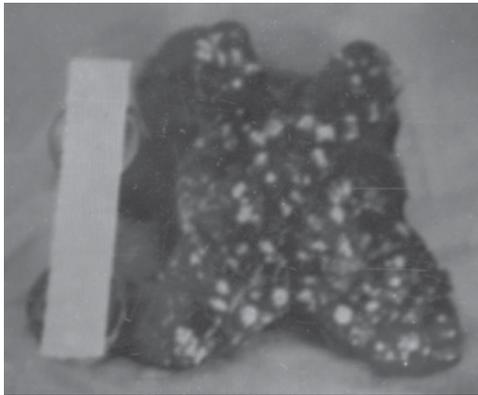


Fig. 1: Splenic tuberculosis: bisected spleen after splenectomy shows multiple tubercles

3. Cirrhosis of the liver especially peritoneal tuberculosis
4. Ileocaecal mass may mimic appendicular lump or malignancy caecum or other conditions.

A high degree of suspicion combined with proper use of diagnostic modalities will help in the timely diagnosis of the disease.

DIAGNOSIS

The isolation of acid fast bacilli (AFB) is the gold standard for diagnosis of pulmonary tuberculosis but may not be possible for establishing the diagnosis of various forms of abdominal tuberculosis. So far the diagnosis of abdominal tuberculosis has been made either on the histological evidence of TB in the tissue (e.g. evidence of tubercles with caseation or demonstration of AFB in a lesion) or typical operative findings suggestive of TB or animal inoculation or tissue culture yielding the growth of *M. tuberculosis*. Now with the advent of better radio-imaging procedures, new criteria for the diagnosis were suggested by Lingenfelter¹¹ as follows:

- i. Clinical manifestations suggestive of TB
- ii. Imaging evidence indicative of abdominal TB
- iii. Histopathological or microbiological evidence of TB and/or
- iv. Therapeutic response to treatment.

INVESTIGATIONS

1. **Blood examination** may show varying degree of anemia, leucopenia and raised ESR
2. **Serum biochemistry:** Serum albumin level may be low. Serum transaminases are normal. A high level of serum alkaline phosphatase may be observed in hepatic tuberculosis.

3. **PPD skin testing/mantoux test:** This gives supportive evidence to the diagnosis of abdominal tuberculosis in 55 to 70% patients if positive, however, a negative tuberculin test may also be observed in one-third of patients. The test is of limited value due to its low sensitivity and specificity. Both false negative and false positive reactions are common. Negative mantoux test in patients of tuberculosis could be due either to (a) immunosuppression or malnutrition producing anergy or (b) recent overwhelming tuberculosis or miliary tuberculosis or (c) rarely circulating mononuclear adherent cells suppressing the sensitized T-lymphocytes in peripheral blood or (d) suppression of PPD-reactive T-lymphocytes. However, a tuberculin test performed later (i.e. after 6-8 weeks) will always be positive in these patients. Positive reactions are also common with quiescent disease or when persons have been sensitized by nontuberculous mycobacteria or following BCG vaccination. The results of anergy testing in HIV-infected populations do not help in the clinical diagnosis and in decision making about preventive therapy.

4. Imaging Techniques:

Plain X-ray abdomen and chest: Plain X-ray of abdomen (erect and supine films) is useful simple investigation. It may show presence of multiple air-fluid levels and dilated loops of gut in case there is subacute or acute intestinal obstruction. Calcification in the abdominal lymph nodes also indicate tuberculosis.

Plain X-ray chest done simultaneously may reveal either healed or active pulmonary tuberculosis in 22 to 80% cases^{7,12}. Although finding of tubercular lesion on chest X-ray supports the diagnosis of abdominal tuberculosis but a normal chest X-ray does not rule it out.

Barium Studies¹²

Barium contrast studies are useful for the diagnosis of intestinal tuberculosis. It has been documented that barium studies are useful in 75% patients with suspected intestinal tuberculosis²¹. *Enteroclysis*, in which a mixture of barium and methylcellulose is infused by a rate-controlled pump into the small intestine with fluoroscopic examination followed by a barium enema may be the best protocol for evaluation of intestinal tuberculosis.

Barium meal follow through is the best diagnostic test for intestinal lesions. The bowel lesions highly suggestive of tuberculosis include multiple strictures, distended caecum or terminal ileum. The other

Table 2: Findings of barium meal follow through study in intestinal tuberculosis¹⁰

Group I	Highly suggestive of intestinal tuberculosis if one or more of the following features are present. <ul style="list-style-type: none"> • Deformed ileocaecal valve with dilated ileum • Contracted caecum with abnormal ileocaecal valve or terminal ileum • Stricture of ascending colon with shortening or involvement of ileocaecal region
Group II	Suggestive of intestinal tuberculosis if one of the following is present: <ul style="list-style-type: none"> • Contracted caecum • Ulceration or narrowing of terminal ileum • Stricture of ascending colon • Multiple sites of narrowing and dilatation leading to formation of small bowel loops
Group III	Non-specific changes Features of adhesions, dilatation and mucosal thickening of small bowel loops
Group IV	Normal study

radiological findings include mucosal irregularity and rapid emptying (ulcerative variety), flocculation and segmentation of barium column (malabsorption pattern), dilated loops and strictures, displaced loops by enlarged lymph nodes and adhesions between gut loops and adherent fixed loops (adhesive peritoneal disease). Barium meal follow through findings in abdominal tuberculosis are divided into 4 groups (Table 2). For tuberculosis of colon and ileocaecal region, the **barium enema studies** are useful. The thickening of ileocaecal valve with triangular appearance, pulled up caecum and/or wide gaping of the valve with narrowing of the terminal ileum (an inverted umbrella sign, or Fleischner's sign, (Fig. 2) have been described in early ileocaecal tuberculosis. **Double contrast barium** studies are more useful for mucosal details and visualizations of ulceration in the early stages of the disease. The tubercular ulcers are shallow with elevated margins and are situated along the circumference of the bowel wall. Rarely, there may be deep ulcers with fistulae. The extreme ulceration of the bowel leads to its irritability and early transition of the barium. Rapid transit and lack of retention of the barium in an inflamed segment of the small bowel constitutes *Stierlin's sign*". A persistent narrowing or stenosis of the bowel leads to consistent narrowing of stream of barium called the "*string sign*". Both the Stierlin's and String signs are also seen in inflammatory bowel disease (Crohn's disease), hence, are nonspecific for tuberculosis. Double contrast barium enema may show a shortened ascending colon, deformed (irregular, shortened, narrowed) caecum,

**Fig. 2:** Barium meal follow through showing stricture of terminal ileum and deformed ileocecal junction with pulled up caecum (an inverted umbrella sign)

incompetent ileocaecal valve with obtuse ileocaecal angle and dilated ileum.

In patients with esophageal tuberculosis, the common radiological features include ulceration, stricture, pseudosinus tract formation and traction diverticulae. In duodenal tuberculosis, barium study may reveal segmental narrowing. The widening of 'C' loop of duodenum may occur due to tubercular lymphadenitis.

Ultrasound¹³⁻¹⁵

The barium studies are sensitive and most useful for diagnosis of intestinal tuberculosis while ultrasonography (USG) is beneficial in extraintestinal (peritoneal, lymph nodes) tuberculosis. The USG of abdomen may show a mass of matted loops of small bowel with thickened walls, rolled up or diseased omentum, and loculated ascites. Fine septae (complete or incomplete), echogenic debris (seen as fine strands and particulate matter) may be seen within tubercular ascites. These septae are due to high fibrin content of the exudative ascitic fluid. However, these findings are not specific to tuberculosis as they may be observed in malignant ascites. Peritoneal thickening and nodularity are the other ultrasonographic findings of peritoneal tuberculosis. Interloop ascites due to localized collection of fluid between radially-oriented bowel loops named as *Club sandwich* or *Sliced bread sign* may be observed.

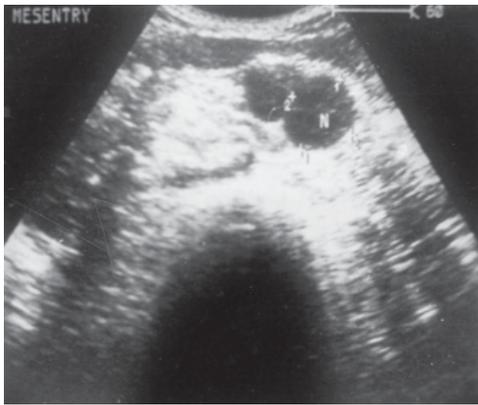


Fig. 3: Abdominal ultrasonography showing enlarged mesenteric lymph nodes (Note the hypoechoic centers due to caseation)

The other conditions that may produce similar findings include mesothelioma, peritoneal carcinomatosis, and sometimes septic peritonitis and hemoperitoneum. Omental cakes and adhesions commonly seen in peritoneal mesothelioma may be observed in peritoneal tuberculosis. Peritoneal tubercles are usually small and rarely seen on USG.

Tubercular lymphadenitis usually involves mesenteric, peri-pancreatic, periportal and para-aortic groups of lymph nodes. These lymph nodes are seen as conglomerate mass and/or as scattered enlarged nodes with hypoechoic center because of necrosis (Fig. 3). This necrosis within lymph nodes may also be seen in metastatic lymphadenopathy. However, caseation with calcification is highly suggestive of tubercular lymphadenitis rather than malignant. The nodes may transiently increase in size once the treatment is started and then gradually diminish in size.

Small bowel mesenteric thickening (15 mm or more) with increased echogenicity combined with mesenteric lymphadenitis is a characteristic ultrasonographic feature of early abdominal (mesenteric) tuberculosis. Omental thickening with altered echogenicity has also been reported. Ultrasonography is also helpful in detecting intestinal tuberculosis. The findings reported include dilated small bowel loops, bowel wall thickening showing a hypoechoic halo measuring >5 mm.

Lastly ultrasound is also helpful for guiding procedures like ascitic fluid aspiration or fine needle aspiration cytology or biopsy from the enlarged lymph nodes or hypertrophic lesions.

*Computed Tomography (CT)*¹⁶

Abdominal CT scan is better than ultrasound for detecting high density ascites, lymphadenopathy with

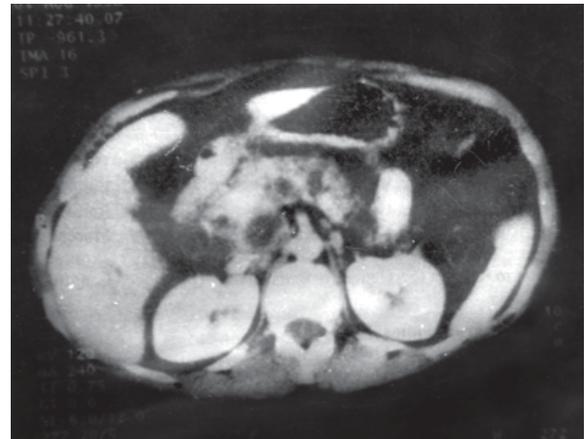


Fig. 4: Abdominal CT scan showing caseous lymph nodes in tubercular lymphadenitis

caseation (Fig. 4), bowel wall thickening and irregular soft tissue densities in the omental area²⁶⁻²⁷. Abdominal lymphadenopathy is the commonest manifestation of tuberculosis on CT. Contrast enhanced CT (CECT) is better than plain CT, shows four patterns of contrast enhancement, i.e. (i) peripheral enhancement, (ii) non homogenous enhancement, (iii) homogenous enhancement and (iv) homogenous non-enhancement. Though not pathognomonic, the pattern of peripheral rim enhancement could be highly suggestive of tuberculosis (Fig. 5). A similar pattern is seen in metastatic lymphadenopathy. The presence of calcification in the lymph nodes in the absence of a known primary tumor suggests tubercular lymphadenitis. Tuberculosis involves predominantly the omental, mesenteric and upper para-aortic lymph nodes; while lower para-aortic lymph nodes are commonly involved in Hodgkin's and Non-Hodgkin's lymphoma.

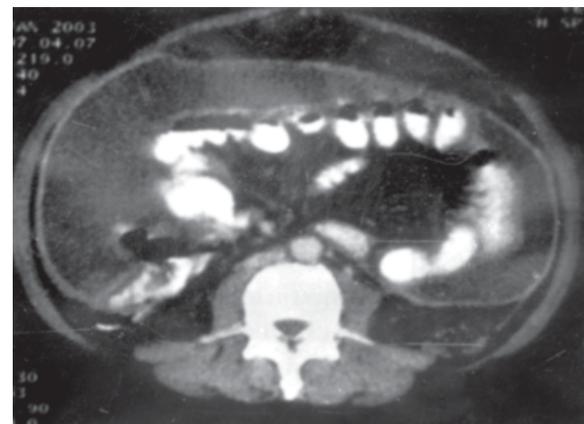


Fig. 5: Tubercular peritonitis. Contrast enhanced CT (CECT) abdomen showing peritoneal reaction with ascites and mesenteric streaking

High density ascites due to high protein and cellular contents of fluid though common in tuberculosis, but can also be seen on CT in mesothelioma and peritoneal carcinomatosis. The CT scan can differentiate between the two, i.e. smooth peritoneum with minimal thickening and marked enhancement on CECT suggest tuberculosis (Fig. 5) while nodular and irregular thickening of peritoneum suggest peritoneal malignancy.

Loculated fluid collections in the presence of omental infiltration, peritoneal enhancement, transperitoneal reaction, i.e. septal, and mesenteric (macronodules >5 mm in diameter) or bowel involvement are important features of abdominal tuberculosis on CT.

The most common CT finding of bowel wall¹³ involvement is a mural thickening affecting the ileo-caecal region either limited to terminal ileum, caecum or both the regions. The other CT findings reported to be highly suggestive of abdominal tuberculosis are irregular soft tissue densities in omental area, low density masses and a disorganized appearance of soft tissue densities, fluid and bowel forming an ill-defined mass.

Tuberculosis of the liver and spleen may appear as tiny low density foci on CT widely distributed throughout the organ. There is a hepatosplenomegaly with areas of calcification within them. The macronodular form of hepatosplenic tuberculosis may be seen as multiple low attenuation, 1-3 cm round lesions or simple tumor like masses. These lesions may show peripheral enhancement on CECT.

The tuberculosis of the pancreas may produce multiple well defined hypoechoic areas on USG and as hypodense necrotic regions on CT (Fig. 6).

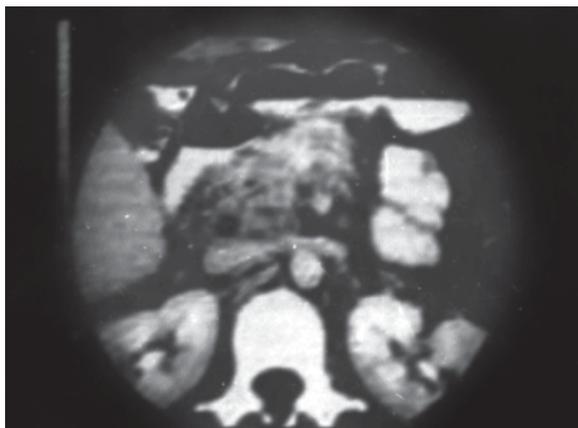


Fig. 6: Pancreatic tuberculosis. CT scan shows multiple nodules in the pancreatic area. In addition, there is enlargement of peripancreatic lymph nodes also

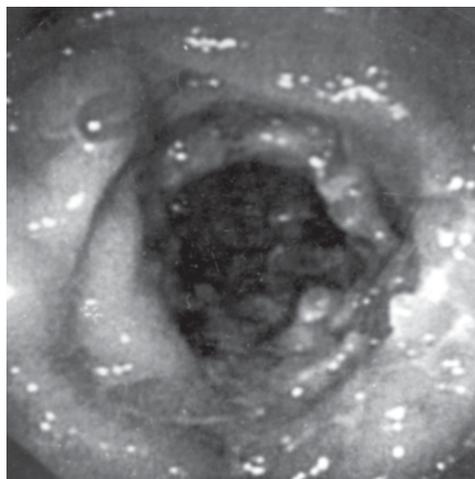


Fig.7: Colonic tuberculosis. Note the multiple colonic ulcers and nodules on colonoscopic view

MRI (Magnetic Resonance Imaging)¹⁷

MRI when compared to CT has no added advantage in the diagnosis of abdominal tuberculosis, hence, its utility in abdominal tuberculosis is limited.

Endoscopy¹⁸

Endoscopy visualizes the tubercular lesion directly, hence, is a useful tool in the diagnosis of colonic and gastro-duodenal tuberculosis; and helps in the confirmation of the diagnosis by obtaining histopathological evidence of tuberculosis. Tuberculosis of the colon involves discrete segments of the colon and produces mucosal nodules and ulcers of varying sizes which are pathognomonic (Fig. 7)¹⁹. Other colonoscopic findings include hyperemic friable mucosa, pseudopolyps and cobblestone appearance of the mucosa simulating Crohn's disease and malignancy. Terminal ileoscopy is the easiest and direct method of establishing the diagnosis of ileocolic tuberculosis. The findings on endoscopy of ileum include submucosal granulomas in all cases from which biopsy may be taken for confirmation of the diagnosis. Endoscopic biopsy specimen may be subjected to PCR for detection of AFB.

Laparoscopy²⁰⁻²¹

Laparoscopy examination is an effective method of diagnosing tubercular peritonitis because (i) it directly visualizes the inflamed thickened peritoneum studded with whitish-yellow miliary tubercles and (ii) biopsy of the peritoneum confirms the diagnosis. Laparoscopy facilitates an accurate diagnosis in 80-90% of patients. Laparoscopic biopsy specimens may reveal AFB in 75%

patients and caseating granulomas in 85-90% patients. The finding of adhesions or fibrotic strands within turbid ascites is virtually diagnostic of tuberculosis. The liver, spleen and omentum can also be examined on laparoscopy, are also studded with tubercles in hepatosplenic tuberculosis. Laparoscopy through open exposure of the peritoneum may be employed in patients with fibroadhesive peritoneal tuberculosis so as to avoid chances of perforation.

Ascitic Tap (Paracentesis)

The ascitic fluid in tuberculosis is exudative (protein >3 g%) with serum-ascites albumin gradient <1.1 g%. Ascitic fluid WBC count is 150-4000 cell/mm³ and consists of predominant lymphocytes. Neutrophils may be seen in early stages of the disease. RBCs, sometimes, may also be seen. Ascitic fluid reveals AFB only in <3% of the cases and culture for *M. tuberculosis* is positive only in 20% of patients.

Adenosine deaminase (ADA) activity in ascitic fluid is a sensitive and specific marker for tuberculosis^{22,23}. ADA is an enzyme present in T-lymphocytes and macrophages, hence, its levels increase due to stimulation of T- lymphocytes in response to CMI to mycobacterial antigens. Dwivedi, et al²¹ have shown a sensitivity and specificity of 100 and 97% respectively when the cut off value of 33U/L was taken. Similarly ascitic fluid to serum ADA ratio >0.985 was also found to be suggestive of tuberculosis. Falsely low levels of ADA can be found in immunocompromised individuals.

Interferon- γ (INF- γ) is an important immune-regulator, is produced by T-lymphocytes in response to stimulation with specific antigens and is capable of activating the macrophages, increasing their bactericidal activity against *M. tuberculosis*. High levels of INF- γ have been found in ascites due to tuberculosis than non-tubercular²⁴. The diagnostic accuracy of this test is yet to be established but combining both ADA and INF- γ estimation in ascitic fluid increase the sensitivity and specificity of the diagnosis of tubercular ascites.

Serodiagnosis

Conventional histological and microbiological methods are often inadequate for the diagnosis of abdominal tuberculosis as it is a paucibacillary disease. A number of serological tests based on the detection of antibody to a variety of mycobacterial antigens developed but all of them have a low predictive value. Polymerase chain reaction (PCR) assay for detection of *M. tuberculosis* in endoscopic biopsy specimens has shown promising results²⁵.

Soft Tissue Biopsy and Culture

Invasive diagnostic procedures are indicated with suspected abdominal tuberculosis. In addition to specimens of involved sites (lymph node, intestine, peritoneum, liver biopsy), bone marrow aspiration for culture may be useful and have a good diagnostic yield in disseminated (military) tuberculosis particularly in HIV infected patients.

Diagnostic Algorithm²⁶

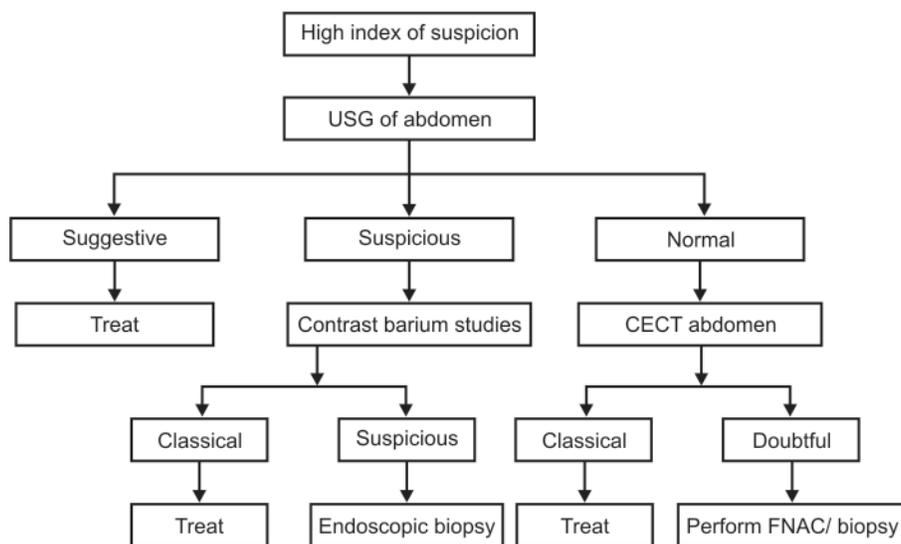
Neither clinical signs, laboratory, radiological and endoscopic methods nor bacteriological and histopathological findings provide a gold standard by themselves for the diagnosis of abdominal TB, hence, an algorithmic approach is useful. An algorithmic approach to the radiological diagnosis of abdominal tuberculosis is presented in Flow Chart 1. Other supportive investigations are done based on the clinical features and imaging.

Treatment

The treatment of abdominal tuberculosis is on the same lines as for pulmonary tuberculosis. Conventional antitubercular therapy for at least 6 months including initial 2 months of HREZ (e.g. isoniazid, rifampicin, ethambutol and pyrazinamide) followed by 4 month HR is recommended in all patients with abdominal tuberculosis. However, previously, the antitubercular therapy was extended upto 8 to 12 months, but recently, a 6 month short course chemotherapy regimen has been found as effective as standard 12 months regimen. However, many physicians still extend the duration of treatment to 12 to 18 months⁴. Corticosteroids have been employed to decrease fibrosis during healing so as to prevent development of obstruction but now-a-days, not preferred as they may delay healing and predispose to perforation or further obstruction. Studies have now shown that even obstructing intestinal lesions can be successfully treated with antitubercular drugs without the need for surgery and complete resolution of radiological abnormalities may occur.

Surgical treatment is done to manage the complications such as obstruction, perforation (free or with access or fistula) and massive hemorrhage not responding to conservative therapy. Strictures are managed by stricturoplasty or resection of the involved segment of the bowel. The perforation is managed by resection and anastomosis rather than by simple closure so as to avoid fistula formation. Bypass surgery such as entero-enterostomy, ileotransverse colostomy is not recommended for obstructive lesions as they may cause formation of blind loops leading to obstruction, fistulation, malabsorption etc.

Flow chart 1: Algorithmic approach to diagnosis of abdominal tuberculosis



Now it has been proved by few studies, that antitubercular drugs alone can relieve obstructing intestinal lesions.

Treatment of HIV Co-existent Tuberculosis²

The key therapeutic principles underlying the treatment of HIV-TB are:

1. The treatment of TB should precede the treatment of HIV infection, i.e. HAART.
2. Patients already on HAART, should continue the same treatment with appropriate modifications in HAART and ATT.
3. Patients who are not receiving HAART, the need and time of initiation of HAART have to be decided on individual basis after assessing the CD₄ count and type of TB.

Principles of ATT in the setting of HIV positive TB are identical to those for HIV-negative cases with two exceptions. In HIV-infected patients with TB, DOTS should be initiated with isoniazid, rifampicin, ethambutol and pyrazinamide (HREZ) for first two months followed by isoniazid and rifampicin (HR) for subsequent 7 months. Since rifampicin resistance is common in HIV patients if CD₄ count is <100/mm³, therefore, first exception, is that treatment regimen should be daily or thrice a week instead of twice a week DOTS during the continuation phase. Second exception is that the continuation phase should be extended to

7 months, so as to make it a regimen of 9 month duration for HIV-TB patients.

Adverse reactions to both ATT and antiretroviral therapy are common, need to be carefully monitored. *Immune restoration syndrome or immune reconstitution inflammatory syndromes (IRIS)* have been reported in 32-36% of patients with HIV-TB infection, within days to weeks after start of antiretroviral therapy.

Fever, lymphadenopathy, worsening pulmonary infiltrates, serositis, skin lesions, new or expanding CNS mass lesion, ARF and ARDS can occur as manifestations of IRIS.

CONCLUSION

Abdominal tuberculosis, a frequently recognized form of extrapulmonary TB is increasing with increasing incidence of HIV infection. The peritoneum and ileocaecal region are commonly involved in majority of the cases by hematogenous spread or through swallowing of infected sputum from primary tubercular infection. The pulmonary tuberculosis may be apparent in about half of these cases. Considerable advances in the diagnosis and management of various forms of abdominal tuberculosis have led to early diagnosis of this disease. Barium studies, CT scan, invasive procedures and serological tests now can help in timely diagnosis and early institution of treatment of such cases so as to reduce morbidity and mortality from this curable but potentially lethal disease.

REFERENCES

1. Sharma SK, Mohan A. Extrapulmonary tuberculosis. *Ind J Med Res* 2004;124:316-53.
2. Sharma SK, Mohan A, Kadiravan T. HIV-TB co-infection: Epidemiology diagnosis and management. *Ind J Med Res* 2005;121:550-67.
3. WHO. In: Global tuberculosis control: surveillance, planning, financing, Geneva: World Health Org 2006, 242.
4. Sharma MP, Bhatia V. Abdominal tuberculosis. *Ind J Med Res* 2004;120(4):305.
5. Marks IN. Abdominal tuberculosis baillierers. *Clin Trop Med Common Dis* 1998;3:329.
6. Marshall JB. Tuberculosis of gastrointestinal tract and peritoneum. *Am J Gastroenterol* 1993;88: 989-99.
7. Sood R, Baba CS. Diagnosis of abdominal tuberculosis. Role of imaging. *J Ind Acad Clin Med* 2001;2:169.
8. Kapoor VK. Abdominal tuberculosis. *Postgrad Med J* 1998;74:459.
9. Kapoor VK. Abdominal tuberculosis; the Indian Contribution. In *J Gastroenterology* 1998;17:141.
10. Kumar N, Aggarwal R. Abdominal tuberculosis. In: *API Textbook of Medicine*, 7th ed. National Book Depot (Distributor): Mumbai. 2003, 562.
11. Lingenfelter T, Zak J, Marks IN, et al. Abdominal tuberculosis; still a potentially lethal disease. *Am J Gastroenterol* 1993;88:744.
12. Kapoor VK, Chattopadhyay TK, Sharma LK. Radiology of abdominal tuberculosis. *Austral Radiol* 1988;32:365.
13. Kedar RP, Shoh PP, Shivde RS, Malde HM. Sonographic findings in gastrointestinal and peritoneal tuberculosis. *Clin Radiol* 1994; 49:24-9.
14. Jain R, Sawhney S, Bhargava DK, Berry M. Diagnosis of abdominal tuberculosis: sonographic findings in patients with early disease. *AJR* 1995;165:1391-5.
15. Suri R, Gupta S, Gupta SK, Singh K, Suri S. Ultrasound guided FNAC in abdominal tuberculosis. *Br J Radiol* 1998;71:723-27.
16. Suri R, Gupta S, Suri R. Computed tomography in abdominal tuberculosis: pictorial review. *Br J Radiol* 1999;72:92-8.
17. Zirinsky K, Auh YH, Kneel JB. Computed tomography, sonography and MR imaging of abdominal tuberculosis. *JCAT* 1985;9:961-3.
18. Bhargava DK, Tandon HD, Chawla TC. Diagnosis of ileocaecal and colonic tuberculosis by colonoscopy. *Gastrointest Endosc* 1985;31:68-70.
19. Singh V, Kumar P, Kamal J. Clinico-colonoscopy profile of colonic tuberculosis. *Am J Gastroenterol* 1996;91:565-8.
20. Rai S, Thomas WM. Diagnosis of abdominal tuberculosis; the importance of laparoscopy. *J Roy Soc Med* 2003;96:586-88.
21. Bhargava DK, Shrinivas, Chopra P. Peritoneal tuberculosis; laparoscopic pattern and its diagnostic accuracy. *Am J Gastroenterol* 1992;87:109-12.
22. Dwivedi M, Misra SP, Misra V, Kumar R. Value of ADA estimation in the diagnosis of tubercular ascites. *Am J Gastroenterol* 1990;85:1123-5.
23. Bhargava DK, Gupta M, Nijhawal S. Adenosine deaminase (ADA) in peritoneal tuberculosis; diagnostic value in ascitic fluid and serum. *Tubercle* 1999;71:121-6.
24. Sharma SK. Diagnostic accuracy of ascitic fluid IFN- γ and ADA assays in the diagnosis of tubercular ascites. *J Interferon Cytokine Res* 2006;26:484-88.
25. Kesarwani RC, Pandey A, Misra A, Singh AK. Polymerase chain reaction (PCR): its comparison with convention techniques for diagnosis of extrapulmonary tubercular disease. *Ind J Surg* 2004;66:84-88.
26. Oya UB, GUI D, Resat D. A clinical dilemma: abdominal tuberculosis. *World J Gastroenterol* 2003;9:1098-1101.

intra-abdominal tuberculosis. 4. diagnosis management. 4. tuberculosis. 1. intra-abdominal. 1. diagnosis. 1. References. (Supplied by CrossRef). Article in Chang Gung Med J Hu ML et al. Chang Gung Med J 2009. Article in Cochrane Database Syst Rev Jullien S et al. Cochrane Database Syst Rev 2016. Member Articles. Racial and Geographic Disparities in Interhospital ICU Transfers. Currently, you can use PubFacts Points to promote and increase readership of your articles. Abdominal tuberculosis (TB) is a collective term that includes TB of the gastrointestinal tract, peritoneum, omentum, mesentery and its lymph nodes and other abdominal organs such as liver, spleen and pancreas. Its presentation has dramatically changed with increasing incidence of HIV coexistence, making the diagnosis in HIV infected persons quite challenging. Also, abdominal tuberculosis can mimic a variety of other abdominal conditions/diseases; hence clinical acumen and timely use of appropriate diagnostic modalities play a very important role in preventing morbidity and mortality. Types of... Chugh S , Jain V. Medicine update-Chapter 102: Abdominal Tuberculosis " Current Concepts in Diagnosis and Management.600-8. Tweet. Pin It.