Abdominal tuberculosis: a diagnostic challenge

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Abstract

Tuberculosis (TB), a multisystemic disease with myriad presentations and manifestations, is the most common cause of infectious disease-related mortality worldwide. Although TB rates are decreasing in the United States, the disease is becoming more common in many parts of the world. In addition, the prevalence of drug resistant TB is increasing worldwide. Abdominal tuberculosis is an increasingly common disease that poses diagnostic challenge, as the nonspecific features of the disease which may lead to diagnostic delays and development of complications. This condition is regarded as a great mimicker of other abdominal pathology. A high index of suspicion is an important factor in early diagnosis. Abdominal involvement may occur in the gastrointestinal tract, peritoneum, lymphnodes or solid viscera. Various investigative methods have been used to aid in the diagnosis of abdominal tuberculosis. Early diagnosis and initiation of antituberculous therapy and surgical treatment are essential to prevent morbidity and mortality. Most of the patients respond very well to standard antitubercular therapy and surgery is required only in a minority of cases. Imaging plays an important role in diagnosis of abdominal tuberculosis because early recognition of this condition is important. In this review we emphasize on the various presentations and diagnostic tools specially radiological features of the gastrointestinal TB as well as other types of abdominal TB.

Key words: Abdominal tuberculosis, diagnosis, therapy etc. ***J Cox Med coll 2017;3(2): 33-41***

Introduction

Tuberculosis is a significant health problem of developing countries. There has also been a resurgence of tuberculosis in developed countries due to migration of people from developing countries, worsening social conditions, shortcomings of the public health services and immuno-compromised status due to diseases and various medications[1].

Tuberculosis (TB) is a life threatening disease which can virtually affect any organ system. Global burden of tuberculosis is nearly 12 million. India subcontinent including Bangladesh has the world's largest tuberculosis cases which is around 26% of the world TB cases, followed by China and South Africa[2]. The Sustainable Development Goals (SDGs) for 2030 were adopted by the United Nations in 2015. One of the targets is to end the global TB epidemic. The WHO End TB Strategy, approved by the World Health Assembly in 2014, calls for a 90% reduction in TB deaths and an 80% reduction in the TB incidence rate by 2030, compared with 2015. In 2015, there were an estimated 10.4 million new (incident) TB cases worldwide, of which 5.9 million (56%) were among men, 3.5 million (34%) among women and 1.0 million (10%) among children. People living with HIV accounted for 1.2 million (11%) of all new TB cases[3]. The primary site of TB is usually lung, from which it can get disseminated into other parts of the body. The other routes of spread can be contiguous involvement from adjacent tuberculous lymphadenopathy or primary involvement of extra pulmonary organ. The diagnosis of extra pulmonary TB can be difficult as it presents with nonspecific clinical and radiological features, and requires high degree of suspicion for diagnosis. The abdominal TB, which is not so commonly seen as pulmonary TB, can be a source of significant morbidity and mortality and is usually diagnosed late due to its nonspecific clinical presentation[4]. Approximately 15%-25% of cases with abdominal TB have concomitant pulmonary TB (PTB)[2].

Hence, it is quite important in identifying these lesions with high index of suspicion especially in endemic areas. The abdominal TB usually occurs in

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four forms: tuberculous lymphadenopathy, peritoneal tuberculosis, gastrointestinal (GI) tuberculosis and visceral tuberculosis involving the solid organs. Usually a combination of these findings occurs in any individual patient[2].

Abdominal tuberculosis is presumed to be highly prevalent in Bangladesh. There is no extensive study done in our country regarding abdominal tuberculosis. One retrospective study was done by Rouf HMA in general Hospital, Sirajgonj[5] intestinal obstruction (25%), and 27% was with chronic symptoms. Faiz M.A. did another retrospective study in 1989 in IPGM&R on extra- pulmonary tuberculosis and found intestinal tuberculosis in 5 cases out of 47 patients having extra-pulmonary tuberculosis. Sheldon CD et al. conducted a retrospective study in east London among Bangladeshi immigrants and showed the crude incidence in Bangladeshi community was 7.7cases/100000/year which was significantly higher than that of European (0.3 cases/100000/year) [6, 7].

In this review we emphasize on the various presentations and diagnostic tools specially radiological features of the gastrointestinal TB as well as other types of abdominal TB.

Materials And Methods

Search strategy and selection criteria In addition to the review of key papers, we undertook searches of electronic databases. For PubMed, the search items were "abdominal tuberculosis" restricted to the past 25 years. "Gastrointestinal tuberculosis, tubercular ascites, serositis, iliocaecal tuberculosis terms were used for search items.", abdominal tuberculosis and pathophysiology", "tuberculosis of alimentary system and diagnosis", "antitubercular chemotherapy" were search items without a time restriction. Articles were selected on the basis of their effect on abdominal tuberculosis treatment or control. When more than one paper illustrated a specific point, the most representative paper was chosen.

Aetiopathogenesis

The disease may develop secondary to primary focus elsewhere in the body; usually the lungs or it may originate within intestinal tract from swallowed sputum or rarely ingestion of cow's milk [8]. The postulated mechanisms by which the tubercle bacilli reach the gastrointestinal tract are: (i) hematogenous spread from the primary lung focus in childhood, with later reactivation; (ii) ingestion of bacilli in sputum from active pulmonary focus; (iii) direct spread from adjacent organs; and (iv) through lymph channels from infected nodes (Table-I). The earlier belief that most cases are due to reactivation of quiescent foci is being challenged with a recent study using DNA fingerprinting showing that 40 per cent cases are due to reinfection. In India, the organism isolated from all intestinal lesions has been Mycobacterium tuberculosis and not M.bovis[7,9].

The most common site of involvement is the ileocaecal region, possibly because of the increased physiological stasis, increased rate of fluid and electrolyte absorption, minimal digestive activity and an abundance of lymphoid tissue at this site. It has been shown that the M cells associated with Peyer's patches can phagocytose BCG bacillis[9]. In Bhansali's series, including 196 patients with gastrointestinal tuberculosis, ileum was involved in 102 and caecum in 100 patients[10]. Of the 300 patients in a study[11] ileocaecal involvement was present in 162. The frequency of bowel involvement declines with distance both proximally and distally from the ileocaecal region. Peritoneal involvement may occur due to spread from lymph nodes, intestinal lesions or from tubercular salpingitis in women. Abdominal lymph nodal and peritoneal tuberculosis may occur without gastrointestinal involvement in about one third of the cases[9].

Table I: Modes of involvement in abdominal tuberculosis

- By ingestion
  - Infected food or milk - Primary intestinal tuberculosis
  - Infected sputum - Secondary intestinal tuberculosis
- Hematogenous spread from distant tubercular focus
- Contagious spread from infected adjacent foci
- Through lymphatic channel

Pathology

Tuberculous granulomas are initially formed in the mucosa or the Peyer's patches. These granulomas are of variable sizes and characteristically tend to be confluent, in contrast to those in Crohn's disease. Granulomas are often seen just beneath the ulcer bed, mainly in the submucosal layer. Submucosal oedema or widening is inconspicuous. Tubercular ulcers are relatively superficial and usually do not penetrate beyond the muscularis[12]. They may be single or multiple, and the intervening mucosa is usually uninvolved. These ulcers are usually

transversely oriented in contrast to Crohn's disease where the ulcers are longitudinal or serpiginous. Cicatricle healing of these circumferential 'girdle ulcers' results in strictures. Occlusive arterial changes may produce ischaemia and contribute to the development of strictures. Endarteritis also accounts for the rarity of massive bleeding in cases of intestinal tuberculosis. Patients with strictures had occlusion of the vasa recta, while ulcerated lesions had hypervascularity. In long-standing lesions there may be variable degree of fibrosis of the bowel wall which extends from submucosa into the muscularis. Many sections may show only non-specific chronic inflammation and no granulomas[9].

Hyperplastic tuberculosis usually occurs in the ileocecal region, although solitary or multiple lesions in the distal ileum are sometimes seen. There is early involvement of regional lymph nodes which may caseate. Patients of this variety present with sub-acute intestinal obstruction, mass in right iliac fossa, perforation and sometimes complete intestinal obstruction. Some times ileocecal tuberculosis presents as acute abdomen without any lump in right lower abdomen or any previous history pointing towards tuberculosis[7].

Table II: Classification of abdominal tuberculosis.[2]

Tubercular lymphadenopathy	
Peritoneal tuberculosis	Acute Chronic Wet ascitic type Fixed fibrotic type Dry plastic type Encysted/loculated type
Visceral tuberculosis	Liver, pancreas, spleen etc.
Gastrointestinal tuberculosis	Esophageal tuberculosis Gastric tuberculosis Duodenal tuberculosis Jejunal and ileocecal tuberculosis Colorectal tuberculosis

In tuberculous peritonitis, the peritoneum is studded with multiple yellow-white tubercles. It is thick and hyperaemic with a loss of its shiny luster. The omentum is also thickened. Peritoneal tuberculosis occurs in 3 forms: (i) Wet type with ascites; (ii) Encysted (loculated) type with a localized abdominal swelling; and (iii) Fibrotic type with abdominal masses composed of mesenteric and omental thickening, with matted bowel loops felt as lump(s) in the abdomen. Combinations of these types are also common[9].

The lymph nodes in the small bowel mesentery and the retro peritoneum are commonly involved, and these may caseate and calcify. Disseminated abdominal tuberculosis involving the gastrointestinal tract, peritoneum, lymph nodes and solid viscera has also been described[13].

Clinical Presentation

Abdominal tuberculosis is predominantly a disease of young adults. Two-thirds of the patients are 21-40 yr old and the sex incidence is equal, although some Indian studies have suggested a slight female predominance[12]. The spectrum of disease in children is different from adults, in whom adhesive peritoneal and lymph nodal involvement is more common than gastrointestinal disease[7,9,14]. The clinical presentation of abdominal tuberculosis can be acute, chronic or acute on chronic. Most patients have constitutional symptoms of fever (40-70%), pain (80-95%), diarrhoea (11-20%), constipation, alternating constipation and diarrhoea, weight loss (40-90%), anorexia and malaise. Pain can be either colicky due to luminal compromise or dull and continuous when the mesenteric lymph nodes are involved[9]. The classic doughy abdomen is associated with the fibro adhesive form of tuberculous peritonitis and is rarely seen. Other clinical features depend upon the site, nature and extent of involvement and are detailed below:

Table III: Clinical features

Site	Type	Clinical features
Small intestine	Ulcerative	Diarrhoea, Malabsorption
	Stricturous	Obstruction
Large intestine	Ulcerative	Rectal bleeding
	Hypertrophic	Lump, obstruction
Peritoneal	Ascitic	Pain, distension
	Adhesive	Obstruction
Lymph nodes		Lump, obstruction

Chronic abdominal pain, sub acute or acute intestinal obstruction, doughy abdomen and visible peristalsis, diarrhea alternating with constipation, abdominal mass may be palpable especially in right iliac fossa and may present as acute peritonitis when mesenteric lymph node caseate and secrete its secretion in peritoneal cavity causing tuberculous peritonitis. These can mimic with any disease involving abdominal organs such as inflammatory bowel disease,

colonic cancer, or infectious disease and ascites of any causes[7,15]. The diagnosis of abdominal tuberculosis is often delayed, increasing the morbidity associated with this treatable condition[7,16]. However, its presentation can be vague, non-specific and can masquerade as other conditions[7,17].

Sometimes it may present as pneumoperitonium especially in ulcerative type, unexplained anemia, chronically ill looking, malnutrition, hemoptysis in case of associated pulmonary tuberculosis and sometimes clubbing. Patients are mostly unvaccinated and contact positive[7].

Oesophageal tuberculosis is a rare entity, constituting only 0.2 per cent of cases of abdominal tuberculosis[9]. Oesophageal involvement occurs mainly by extension of disease from adjacent lymph nodes. The patient usually presents with low grade fever, dysphagia, odynophagia and an ulcer, most commonly midoesophageal. The disease usually mimics oesophageal carcinoma and extraoesophageal focus of tuberculosis may not be evident[7,9].

Stomach and duodenal tuberculosis each constitute around 1 per cent of cases of abdominal tuberculosis. Gastroduodenal tuberculosis may mimic peptic ulcer disease with a shorter duration of history and non response to anti-secretary therapy[18]. It may also simulate gastric carcinoma[9].

The largest published series of duodenal tuberculosis reported 30 cases from India[18]. Most patients (73%) had symptoms of duodenal obstruction. In a majority of these cases obstruction was due to extrinsic compression by tuberculous lymph nodes, rather than by intrinsic duodenal lesion. The remainders (27%) had a history of dyspepsia and were suspected of having duodenal ulcers. Two of these patients presented with hematemesis[18].

Patients with ileocaecal tuberculosis complain of colicky abdominal pain, borborygmi and vomitings. Abdominal examination may reveal no abnormality or a doughy feel. A well defined, firm, usually mobile mass is often palpable in the right lower quadrant of the abdomen. Associated lymphadenitis is responsible for the presence of one or more lumps which are mobile if mesenteric nodes are involved and fixed if para-aortic or illiac group of nodes are enlarged. The most common complication of small bowel or ileocaecal tuberculosis is obstruction due to narrowing of the lumen by hyperplastic caecal tuberculosis, by strictures of the small intestine, which are commonly multiple, or by adhesions. Adjacent lymph nodal involvement can lead to traction, narrowing and fixity of bowel loops. In India, around 3 to 20 per cent of all cases of bowel obstruction are due to tuberculosis[7,10,19,20]. Tuberculosis accounts for 5-9 percent of all small intestinal perforations in India, and is the second commonest cause after typhoid fever[21,22]. Tubercular perforations are usually single and proximal to a stricture[23]. Acute tubercular peritonitis without intestinal perforation is usually an acute presentation of peritoneal disease but may be due to ruptured caseating lymph nodes[7,10,22]. Mal-absorption is a common complication. Next to tropical sprue, it is the most important cause of malabsorption syndrome in India. In patients with malabsorption, history of abdominal pain suggests the diagnosis of tuberculosis[23].

Segmental or isolated colonic tuberculosis refers to involvement of the colon without ileo-caecal region, and constitutes 9.2 per cent of all cases of abdominal tuberculosis. It commonly involves the sigmoid, ascending and transverse colon[24]. Multifocal involvement is seen in one third (28 to 44%) of patients with colonic tuberculosis[25,26]. Pain is the predominant symptom in 78-90 percent of patients and Haematochezia occurs in less than one third[25,27]. The bleeding is frequently minor and massive bleeding is less common.

Clinical presentation of rectal tuberculosis is different from more proximal disease. Haematochezia is the most common symptom (88%) followed by constitutional symptoms (75%) and constipation (37%)[27]. Overall rectal tuberculosis is rare and may occur in the absence of other lesions in the chest or small and large bowel[28,29]. Anal tuberculosis is less uncommon and has a distinct clinical presentation. Tubercular fistulae are usually multiple.

Investigations

Investigations in abdominal TB commonly includes routine blood counts with raised ESR. Sputum analysis can reveal associate PTB in less than 20 percent cases. Mantoux test, Ascitic fluid examination, X-rays and barium studies, CT scan of Abdomen with contrast, PCR for tubercular peritonitis, Diagnostic Laparoscopy for abdominal tuberculosis in peritoneal seedling and extra luminal intestinal TB, Endoscopy for upper

Gastrointestinal tuberculosis can be done according to site and specific area. Chest radiographs may show hilar lymphadenopathy or tuberculous lesion in case of concurrent active pulmonary T.B. However a normal chest radiograph does not rule out the possibility of abdominal tuberculosis[7,30-32].

X-rays abdomen radiograph can show air fluid levels and dilated gut loops indicating intestinal obstruction or air under diaphragm in case of gut perforation. It can also show calcifications in mesenteric lymph nodes[7,30-32].

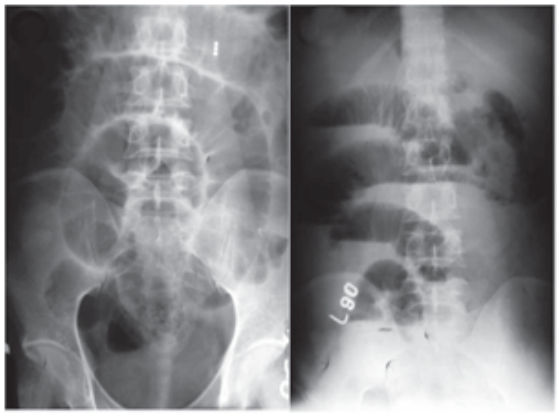

Figure 1 : Plain X ray images. Plain abdominal radiographs in supine (A) and erect (B) position showing dilated small bowel loops with air fluid levels in a patient who presented with subacute intestinal obstruction secondary to tubercular ileal stricture.

USG : abdomen can show mesenteric thickening, enlarged mesenteric lymph nodes, thickened omentum, peritoneal ascites with septations, dilated small bowel loops and bowel wall thickening. It is also an important tool for USG aspiration of ascetic fluid[7,32]. The following features may be seen, usually in combination[7].

(i) Intra-abdominal fluid which may be free or loculated; and clear or complex (with debris and septae).
(ii) "Club sandwich" or "sliced bread" sign is due to localized fluid between radially oriented bowel loop.
(iii) Lymphadenopathy may be discrete or conglomerated (matted). The echo texture is mixed heterogenous. Both caseation and calcification are highly suggestive of a tubercular etiology, neither being common in malignancy related lymphadenopathy.
(iv) Bowel wall thickening is best appreciated in the ileo-cecal region. The thickening is uniform and concentric as opposed to the eccentric thickening in Crohn's disease and variegated appearance of malignancy.
(v) Pseudo-kidney sign - involvement of the ileocecal region which is pulled up to a sub-hepatic position.

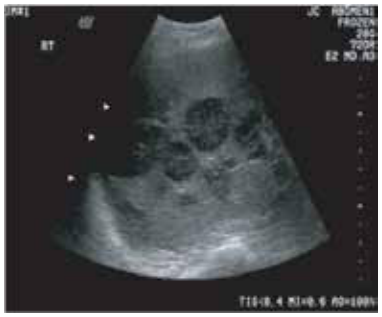

Figure 2 Ultrasound image. Multiple enlarged conglomerate lymphnodes with hypoechoic centers due to caseation.

Barium studies

In various studies conducted all over world, the barium contrast radiography was helpful in about 75% of patients suspected to have intestinal tuberculosis. Findings included dilated bowel loops, strictures, deformed and pulled-up caecum, ulceration of ileum, bowel wall thickening, and extrinsic compression by lymph nodes. Thus, contrast barium studies seem to have a good diagnostic yield, when performed in patients with suspected intestinal involvement [30,31,32].

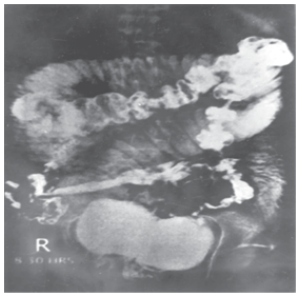

Figure 3 Barium meal follow through image. Barium meal follow through showing stricture of terminal ileum and deformed ileocecal junction with pulled up cecum (an inverted umbrella sign)

Barium enema

The following features may be seen[7]:

(i) Early involvement of the ileocaecal region manifesting as spasm and oedema of the ileocaecal valve. Thickening of the lips of the

ileocaecal valve and/or wide gaping of the valve with narrowing of the terminal ileum ("Fleischner" or "inverted umbrella sign") are characteristic.

(ii) "Conical caecum", shrunken in size and pulled out of the iliac fossa due to contraction and fibrosis of the mesocolon. The hepatic flexure may also be pulled down.

(iii) Loss of normal ileo-cecal angle and dilated terminal ileum, appearing suspended from a retracted, fibrosed caecum ("goose neck deformity").

(iv) "Purse string stenosis"- localized stenosis opposite the ileocaecal valve with a rounded off smooth caecum and a dilated terminal ileum.

(v) "Sterling's sign" is characterized by lack of barium retention in the inflamed segments of the ileum, caecum and variable length of the ascending colon, with a normal configured column of barium on either side.

(vi) "String sign" - persistent narrow stream of barium indicating stenosis. Both Sterling and String signs can also be seen in Crohn's disease and hence not specific for tuberculosis.

CT scan

The most common findings on CT scan highly suggestive of abdominal tuberculosis are high density ascites, lymphadenopathy, bowel wall thickening, and irregular soft tissue densities in the omental area. Abdominal lymphadenopathy is the commonest manifestation of tuberculosis on CT.[7,30,31].

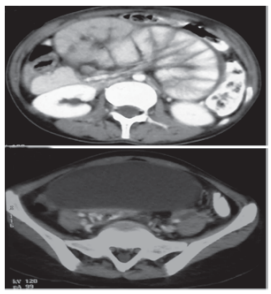

Figure 4 Computed tomography images. A: Axial images of the same patient showing small bowel loops congregated in the centre of the abdomen encased by a soft tissue density membrane. Enlarged discrete lymph nodes are also seen in the mesentery (arrow head); B: Loculated ascites is seen just below the level of conglomerate bowel loops.

Mantoux test

It is very important adjuvant test in diagnosis of extra pulmonary T.B. Immunization with BCG vaccine can cause a positive test, but the reactions are usually only 5-10 mm and tend to decrease with time. People with PPD reactions of 15 mm or more are assumed to be infected with mycobacterium TB even if they are vaccinated with BCG. False positive result may occur in persons previously vaccinated with BCG and in those infected with non tuberculous or atypical mycobacterium. False negative result may occur in improper testing technique, concurrent infections, malnutrition, advanced age, immunologic disorders, lymphoreticular malignancies, Corticosteroid therapy, chronic renal failure, HIV infection and fulminant tuberculosis[7].

Ascitic fluid examination

The ascitic fluid in tuberculosis is straw colored with protein >3g/dl, and total cell count of 150-4000/ml, consisting predominantly of lymphocytes (>70%). The ascites to blood glucose ratio is less than 0.9650 and serum ascites albumin gradient is less than 1.1 g/ dl. The yield of organisms on smear and culture is low. Staining for acid fast bacilli is positive in less than 3 per cent of cases. A positive culture is obtained in less than 20 per cent of cases, and it takes 6-8 weeks for the mycobacterial colonies to appear[7].

Adenosine deaminase (ADA) is an aminohydrolase that converts adenosine to inosine and is thus involved in the catabolism of purine bases. The enzyme activity is more in T than in B lymphocytes, and is proportional to the degree of T cell differentiation. ADA is increased in tuberculous ascetic fluid due to the stimulation of T-cells by mycobacterial antigens. ADA levels were determined in the ascetic fluid of 49 patients by Dwivedi et al[33]. The levels in tuberculous ascites were significantly higher than those in cirrhotic or malignant ascites. Taking a cut off level of 33 U/l, the sensitivity, specificity and diagnostic accuracy were 100, 97 and 98 per cent in these condition respectively[33]. In coinfection with HIV the ADA values can be normal or low. Falsely high values can occur in malignant ascites[7].

PCR (Polymerase chain reaction)

PCR testing of ascitic fluid is a good tool to

detect DNA of mycobacterium tuberculosis[34].

Colonoscopy

It is a very useful tool for identifying colonic and ileocecal tuberculosis. Mucosal nodules (2-6mm) and ulcers (4-8cm) are pathognomonic of tuberculosis. The nodules have a pink surface with no friability and are most often found in the caecum especially near the ileo-cecal valve. Areas of strictures with nodular and ulcerated mucosa may be seen. Other findings are pseudopolypoid edematous folds, and a deformed and edematous ileocecal valve. Diffuse involvement of the entire colon is rare (4%), but endoscopically can look very similar to ulcerative colitis. Biopsies should be taken from the edge of the ulcers. A combination of histology and culture of the biopsy material can be expected to establish the diagnosis in over 60 per cent of cases[7].

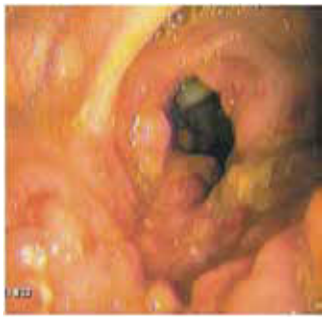

Figure 5 : Colonoscopic image which showing mocosal thickening of the caecum and patulous thickened ileocaecal valve.

Diagnostic laparoscopy

Laparoscopy provides a good deal of visual confirmation of findings, taking biopsy and collecting ascitic fluid for further investigations[30]. Caseating Granulomas may be found in 85-90 per cent of the biopsies. The laparoscopic findings in peritoneal tuberculosis can be grouped into 3 categories:

(i) Thickened peritoneum with tubercles: Multiple, yellowish white, uniform sized (about 4-5 mm) tubercles diffusely distributed on the parietal peritoneum. The peritoneum is thickened, hyperemic and lacks its usual shiny luster.

(ii) The omentum, liver and spleen can also be studded with tubercles.

(iii) Thickened peritoneum without tubercles.

(iv) Fibro-adhesive peritonitis with markedly thickened peritoneum and multiple thick adhesions fixing the viscera.

Management

All patients with abdominal tuberculosis should be given standard full course of Anti tubercular therapy (ATT). Duration of treatment is different in different centers. Treatment with three drug regimen for nine months was also used as anti tubercular treatment. Previously 18-24 months regime was popular, then 1 year regime and now 6 months of total duration is considered sufficient for chemotherapy[30,32].

In Bangladesh, there is a national guide line for treatment of all kinds of TB patients. It recommends for total 6 months ATT for the treatment of abdominal tuberculosis patients[35]. All patients of abdominal tuberculosis should receive conventional anti-tubercular therapy for at least 6 months including initial 2 months of rifampicin, isoniazid, pyrazinamide, ethambutol and next 4 months of rifampicin and isoniazide[35]. Some authors have recommended the addition of corticosteroids in patients with peritoneal disease in order to reduce subsequent complications of adhesions[7].

Surgical management is usually indicated for complications of abdominal tuberculosis in the form of obstruction, perforation, uncontrolled bleeding etc. Various surgical procedures are usually performed like: segmental resection of the diseased segment including right hemi-colectomy, release of band and adhesions, repair of the perforation, stricturoplasty, exteriorization of the perforation loop and cholecystectomy

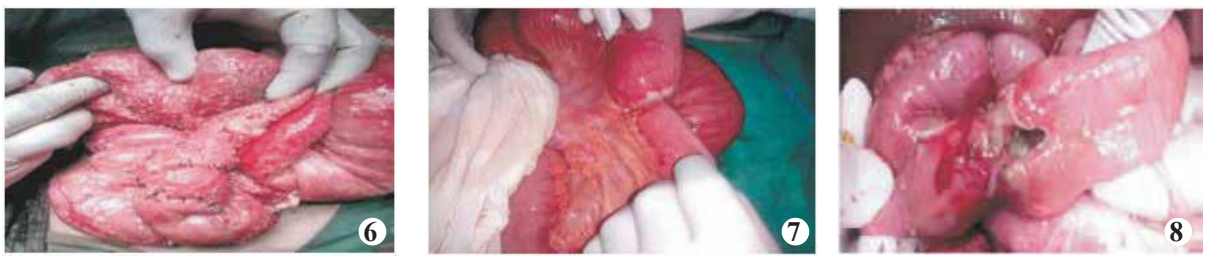

Figure 6, 7, 8 : Extensive peritoneal tubercles (6), Concentric tubercular stricture in the ileum (7), Perforated tubercular ulcer with distal stricture in the ileum causing purulent peritonitis (8).

Conclusion

A high clinical index of suspicion and judicious use of diagnostic procedure can certainly help in timely diagnosis and treatment and thus reduce the mortality of this curable but potentially lethal disease. One or more of the following four criteria along with high clinical index of suspicion may be helpful to diagnose abdominal tuberculosis-

(1) Histological evidence of tubercle with caseation necrosis. (2) Histological demonstration of acid fast bacilli in a lesion. (3) Culture of suspected tissue resulting in growth of M. tuberculosis. (4) Increased ADA (Adenosine deaminase) in ascetic fluid (>37 IU/L).

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