REVIEW ARTICLE

ABDOMINAL TUBERCULOSIS -A REVIEW

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Introduction:

Tuberculosis is an infectious disease that has plagued mankind since Neolithic times (8000 BC) [1]. It was recognized as a contagious disease by the time of Hippocrates (400 BC), when it was termed as 'phthisis' (Greek Phthinien, meaning to waste away)[2] Abdominal TB which may involved the gastrointestinal tract, peritoneum, lymph nodes or solid viscera, constitutes up to 12% of extra pulmonary TB and 1-3% of the total[3,4]. Abdominal tuberculosis includes tuberculosis of gastrointestinal tract, peritoneum, mesenteric lymph node, Liver, Spleen, excluding genito-urinary system[5]. Tuberculosis (TB) can involve any part of the gastrointestinal tract from mouth to anus, the peritoneum and the pancreatobiliary system. It can have a varied presentation, frequently mimicking other common and rare diseases[6]. TB of the gastrointestinal tract is the sixth most frequent form of extra-pulmonary site, after lymphatic, genitourinary, bone and joint, miliary and meningeal tuberculosis[7]. The term Extrapulmonary Tuberculosis (EPTB) has been used to describe isolated occurrence of tuberculosis at body sites other than the lung. However, when an extra-pulmonary focus is evident in a patient with pulmonary tuberculosis, such patients have been categorized under pulmonary tuberculosis as per the guidelines of the World Health Organization (WHO) [8].

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", "antuitubercular chemotherapy" were search items without a time restriction. The Cochrane database of systematic reviews was searched by use of the term "abdominal tuberculosis". 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.

Epidemiology:

Tuberculosis causes some 3 million deaths per year worldwide and is increasing in developed and developing countries[9]. Despite the accelerated efforts to control the disease for decades, it remains the seventh leading cause of death globally[10]. Abdominal tuberculosis is still highly prevalent in developing country like India, South Africa, and Saudi Arabia[11,12,13,14,15]. In Western countries, with the development of effective anti-tubercular chemo-therapy and preventive measure, incidence of pulmonary tuberculosis was declined but extra-pulmonary tuberculosis remained constant1[16,17]. There is considerable variation in the incidence of abdominal tuberculosis in different ethnic groups. The disease is endemic in South East Asian and Latin American countries. In the United Kingdom, immigrants from Asia are significantly more prone to this disease compared to the native population[18]. In the U.S.A. all forms of tuberculosis are seen amongst the immigrant population, people residing in the north American Indian reservations and in patients suffering from HIV-AIDS.[19,20]In the era before the human immunodeficiency virus (HIV) pandemic, and in studies involving immune-competent adults, it has been observed that EPTB constituted about 15 to 20 per cent of all cases of TB[21-31]. In HIV-positive patients, EPTB accounts for more than 50 per cent of all cases of TB[32-40]. The diagnosis of EPTB, especially involving deeply located in accessible areas is very difficult..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 [41]with intestinal obstruction (25%), and 27% chronic

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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[42]. 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) [43].

Pathogenesis and pathology:

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[44].

The postulated mechanisms by which the tubercle bacilli reach the gastrointestinal tract are: (i) haematogenous 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) and through lymph channels from infected nodes. 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 re-infection. In India, the organism isolated from all intestinal lesions has been Mycobacterium tuberculosis and not M.bovis [45,46]. Tuberculosis (TB) can involve any part of the gastrointestinal tract from mouth to anus, the peritoneum and the pancreatobiliary system. The most common site of involvement is the ileo-caecal 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[47]. In Bhansali's series, including 196 patients with gastrointestinal tuberculosis, ileum was involved in 102 and caecum in 100 patients[48]. Of the 300 patients in a study ileocaecal involvement was present in 162[49]. The frequency of bowel involvement declines as one proceeds both proximally and distally from the ileocaecal region. Peritoneal involvement may occur from 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[50].

Tuberculous granulomas are initially formed in the mucosa or the Peyer's patches. These granulomas are of variable size 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[51]. 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[52]. Cicatrical healing of these circumferential 'girdle ulcers' results in strictures. Occlusive arterial changes may produce ischaemia and contribute to the development of strictures[53]. Endarteritis also accounts for the rarity of massive bleeding in cases of intestinal tuberculosis. Shah et al[54] correlated findings on barium studies and superior mesenteric angiography in 20 patients. Angiograms were abnormal in all and showed arterial encasement, stretching and crowding of vessels, and 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. Mesenteric lymph nodes may be enlarged, matted and may caseate. Characteristic granulomas may be seen only in the mesenteric lymph nodes**.** The reverse, i.e., the presence of granulomas in the intestine and no granulomas in the draining lymph nodes is rare[50]. Hoon et al[51] originally classified the gross morphological appearance of the involved bowel into ulcerative, ulcerohyperplastic and hyperplastic varieties. Tandon and Prakash[50] described the bowel lesions as ulcerative and ulcerohypertrophic types. Ulcerative form has been found more often in malnourished adults, while hypertrophic form is classically found in relatively well nourished adults. These ulcerative and stricturous lesions are usually seen in the small intestine. Colonic and ileocaecal lesions are ulcerohypertrophic. The patient often presents with a right iliac fossa lump constituted by the ileocaecal region, mesenteric fat and lymph nodes. The ileocaecal angle is distorted and often obtuse. Both sides of the ileocaecal valve are usually involved leading to incompetence of the valve, another point of distinction from Crohn's disease. 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[58]

Intestinal tuberculosis: Intestinal tuberculosis is usually seen in underdeveloped countries. Organisms involved are Mycobacterium tuberculosis and

Mycobacterium Bovis. Primary focus is in intestine and mesenteric lymph nodal involvement causes Ghon complex formation. Tuberculous granulomas are initially formed in the mucosa or the peyer's patches. These granulomas are seen just beneath the ulcer bed mainly in the submucosa. Tuberculous ulcers are superficial and usually transversely oriented. Cicatrical healing of these ulcers may produce strictures. More over occlusive arterial changes associated with tuberculosis may produce ischemia and perforation of ulcers as well as aid in development of strictures.[60,61]

Ulcerative tuberculosis:It is mostly secondary to pulmonary tuberculosis and arises as a result of swallowing tubercle bacilli. There are multiple ulcers in the terminal ileum, lying transversely, and the overlying serosa is thickened, reddened, and covered in tubercles. [62]

Hyperplastic tuberculosis:

This variety 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 presented with sub-acute intestinal obstruction, mass in right iliac fossa, perforation and some times complete intestinal obstruction. [62]. Some times ileocecal tuberculosis presents as acute abdomen without any lump in right lower abdomen or any previous history pointing towards tuberculosis

Tuberculous peritonitis: It is of two varieties according to mode of presentation. One is acute and other is chronic. Acute variety presented as acute peritonitis and on opening peritoneum straw colored fluid come with tubercles scattered over peritoneum and greater omentum. Chronic variety presented as pain abdomen, fever, weight loss, ascites and sometime as abdominal mass. Source of infection are, mesenteric lymphadenitis, intestinal tuberculosis, blood borne from pulmonary tuberculosis[62] Peritoneal tuberculosis occurs in 3 forms: 1) Wet type with ascites; 2) Encysted (loculated) type with a localized abdominal swelling; 3) Fibrotic type with abdominal masses composed of mesenteric and omental thickening, with matted bowel loops felt as lump(s) in the abdomen. A combination of these types are also common.

Tuberculous mesenteric lymphadenitis: These lymph nodes caseate and can drain their secretion in peritoneal cavity causing tuberculous peritonitis.The involved lymph node calcifies in about a year.

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 predominanc[53]. The clinical presentation of abdominal tuberculosis can be acute, chronic or acute on chronic. Symptoms and signs of tuberculosis are non-specific and protean. Weight loss, Fever (low grade), chronic cough, malaise, anorexia,night sweets are features developed due to cytokines released by activated macrophages. i.e. IL-1, TNFa and not by the organism itself.

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. Other clinical features depend upon the site, nature and extent of involvement and are detailed below:

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[56]. The diagnosis of abdominal tuberculosis is often delayed, increasing the morbidity associated with this treatable condition[57,63-70]. However, its presentation can be vague, non-specific and can masquerade as other conditions[58,71-76]. Some times present as pneumoperitonium especially in ulcerative type, anemia, chronically ill looking, malnutrition, hemoptysis in case of associated pulmonary Tuberculosis and sometimes clubbing. Patients are mostly unvaccinated and having contact positive. [77,78-82]

Tuberculosis of the oesophagus:

Oesophageal tuberculosis is a rare entity, constituting only 0.2 per cent of cases of abdominal tuberculosis[47]. 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[60].

Gastroduodenal tuberculosis

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[61]. Most patients (73%) had symptoms of duodenal obstruction. The remainder (27%) had a history of dyspepsia and were suspected of having duodenal ulcers. Duodenal tuberculosis is often isolated with no associated pulmonary lesions in more than 80 per cent cases[62].

Ileocaecal tuberculosis

Patients 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[48,63,64]. Tuberculosis accounts for 5-9 per cent of all small intestinal perforations in India, and is the second commonest cause after typhoid fever[65,66]. Tubercular perforations are usually single and proximal to a stricture[53]. Acute tubercular peritonitis without intestinal perforation is usually an acute presentation of peritoneal disease but may be due to ruptured caseating lymph nodes[48, 66].

Mal-absorption is a common complication. Next to tropical sprue, it is the most important cause of mal-absorption syndrome in India. In a patient with mal-absorption, a history of abdominal pain suggests the diagnosis of tuberculosis[67].

Segmental colonic tuberculosis

Segmental or isolated colonic tuberculosis refers to involvement of the colon without ileo-cecal region, and constitutes 9.2 per cent of all cases of abdominal tuberculosis. It commonly involves the sigmoid, ascending and transverse colon[68]. Multifocal involvement is seen in one third (28 to 44%) of patients with colonic tuberculosis[69,70]. The median duration of symptoms at presentation is less than 1 yr[71]. Pain is the predominant symptom in 78-90 per cent of patients and Haematochezia occurs in less than one third[69,72]. The bleeding is frequently minor and massive bleeding is less common. Overall, tuberculosis accounts for about 4 per cent of patients with lower gastrointestinal bleeding[66].

Rectal and anal tuberculosis

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%) [72]. Overall rectal tuberculosis is rare and may occur in the absence of other lesions in the chest and small and large bowel[73,74]. Anal tuberculosis is less uncommon and has a distinct clinical presentation. Tubercular fistulae are usually multiple. Constitutional symptoms were not present in any patient[75]. Anal tuberculosis is also seen in pediatric patients[76].

Investigations:

Commonly includes- Routine CBC 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 siddling and extraluminal 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. [77,78,83-91].

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.[77,78,91].

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 ascitic fluid. [77,91-97].The following features may be seen, usually in combination [98].

(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 ileo-cecal region which is pulled up to a sub-hepatic position.

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 [77,78,91].

Barium enema:

The following features [97] may be seen:

(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 are 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. [77,78,91].

Mantoux test:

It is very important adjuvant test in diagnosis of extrapulmonary 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 mycobacteria. 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.[79,80,81].

Ascitic fluid examination:

The ascitic fluid in tuberculosis is straw colored with protein >3g/dl, and total cell count of 150-4000/ il, 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 wk for the mycobacterial colonies to appear. [82].

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 ascitic fluid due to the stimulation of T-cells by mycobacterial antigens. ADA levels were determined in the ascitic fluid of 49 patients by Dwivedi *et al*[94]. 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 respectively[94]. In the study by Bhargava *et al*[95], serum

ADA level above 54U/l, ascitic fluid ADA level above 36 U/l and a ascitic fluid to serum ADA ratio >0.985 were found suggestive of tuberculosis[96]. In co-infection with HIV the ADA values can be normal or low. Falsely high values can occur in malignant ascites. High interferon-levels in tubercular ascites have been reported to be useful diagnostically[83]. Combining both ADA and interferon estimations may further increase sensitivity and specificity.

PCR (Polymerase chain reaction):

PCR testing of ascitic fluids is a good tool to detect DNA of mycobacterium tuberculosis[82].

Colonoscopy:

It is a very useful tool for identifying colonic and ileo-cecal tuberculosis. Mucosal nodules (2-6mm) and ulcers (4-8cm) are pathognomic of tuberculosis. [78].Colonoscopy is an excellent tool to diagnose colonic and terminal ileal involvement but is still often underutilized. Mucosal nodules of variable sizes (2 to 6 mm) and ulcers in a discrete segment of colon, 4 to 8 cm in length are pathognomic. The nodules have a pink surface with no friability and are most often found in the caecum especially near the ileo-cecal valve. Large (10 to 20 mm) or small (3 to 5 mm) ulcers are commonly located between the nodules. The intervening mucosa may be hyperemic or normal[70]. Areas of strictures with nodular and ulcerated mucosa may be seen. Other findings are pseudopolypoid edematous folds, and a deformed and edematous ileo-cecal valve. Diffuse involvement of the entire colon is rare (4%), but endoscopically can look very similar to ulcerative colitis. Lesions mimicking carcinoma have also been described[70-72]. Most workers take up to 8-10 colonoscopic biopsies for histopathology and culture. Biopsies should be taken from the edge of the ulcers. However, there is a low yield on histopathology because of predominant sub-mucosal involvement. Granulomas have been reported in 8-48 per cent of patients and caseation in a third (33-38%) of positive cases[71]. A combination of histology and culture of the biopsy material can be expected to establish the diagnosis in over 60 per cent of cases.

Diagnostic laparoscopy:

Laparoscopy provides a good deal of visual confirmation of findings, taking biopsy and collecting ascitic fluid for further investigations.[77]

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

(ii) Thickened peritoneum without tubercles.

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

Diagnosis:

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[9]. Many authors in endemic countries also recommend clinical trial[85, 86.]

One or more of the following four criteria along with high clinical index of suspicion must be fulfilled 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 ascitic fluid (>37 IU/L)

Management:

Management of all patients with abdominal tuberculosis should be given standard full course of ATT. Duration of treatment is different in different centers. Treatment with three drug regimen for nine months was also used as anti tuberculous treatment . Successful treatment of abdominal tuberculosis for one to one and a half year[87] is also the recommendation in some authors[87,88,89].

Previously 18-24 months regime was popular, then 1 year regime and now 6 months of total duration is considered sufficient for chemotherapy[90,91,92].

In Bangladesh, there is a national guide line for treatment of all kinds of TB patients. It is recommended total 6 months ATT for the treatment of abdominal tuberculosis patients[93].

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[93].

Some authors have recommended the addition of corticosteroids in patients with peritoneal disease in order to reduce subsequent complications of adhesions[99]

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