Review Article
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Abdominal tuberculosis

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Tuberculosis can involve any part of the gastrointestinal tract and is the sixth most frequent site of extrapulmonary involvement. Both the incidence and severity of abdominal tuberculosis are expected to increase with increasing incidence of HIV infection. Tuberculosis bacteria reach the gastrointestinal tract via haematogenous spread, ingestion of infected sputum, or direct spread from infected contiguous lymph nodes and fallopian tubes. The gross pathology is characterized by transverse ulcers, fibrosis, thickening and stricturing of the bowel wall, enlarged and matted mesenteric lymph nodes, omental thickening, and peritoneal tubercles.

Peritoneal tuberculosis occurs in three forms : wet type with ascitis, dry type with adhesions, and fibrotic type with omental thickening and loculated ascites. The most common site of involvement of the gastrointestinal tuberculosis is the ileocaecal region. Ileocaecal and small bowel tuberculosis presents with a palpable mass in the right lower quadrant and/or complications of obstruction, perforation or malabsorption especially in the presence of stricture. Rare clinical presentations include dysphagia, odynophagia and a mid oesophageal ulcer due to oesophageal tuberculosis, dyspepsia and gastric outlet obstruction due to gastroduodenal tuberculosis, lower abdominal pain and haematochezia due to colonic tuberculosis, and annular rectal stricture and multiple perianal fistulae due to rectal and anal involvement.

Chest X-rays show evidence of concomitant pulmonary lesions in less than 25 per cent of cases. Useful modalities for investigating a suspected case include small bowel barium meal, barium enema, ultrasonography, computed tomographic scan and colonoscopy. Ascitic fluid examination reveals straw coloured fluid with high protein, serum ascitis albumin gradient less than 1.1 g/dl, predominantly lymphocytic cells, and adenosine deaminase levels above 36 U/l. Laparoscopy is a very useful investigation in doubtful cases. Management is with conventional antitubercular therapy for at least 6 months. The recommended surgical procedures today are conservative and a period of preoperative drug therapy is controversial.

Key words Abdominal tuberculosis - extrapulmonary - gastrointestinal - peritoneal - ultrasonography

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[1]. The clinician must look for tuberculosis, and confirm or exclude this treatable malady in any patient who presents with gastrointestinal disease.

Autopsies conducted on patients with pulmonary tuberculosis before the era of effective antitubercular drugs revealed intestinal involvement in 55-90 per cent cases, with the frequency related to the extent of pulmonary involvement. Pimparkar found evidence of abdominal tuberculosis (bowel, peritoneum and liver) in 3.72 per cent of 11,746 autopsies carried out in K.E.M. Hospital, Mumbai between 1964 to

1970[2]. Both the incidence and the severity of abdominal tuberculosis are expected to increase with increasing incidence of HIV infection in India. About 0.4 million people in India are coinfected with HIV and tuberculosis. In a study from Mumbai, HIV seroprevalence was found in 16.6 per cent in patients with abdominal tuberculosis as compared to 1.4 per cent in voluntary blood donors[3]. Extra-pulmonary forms of TB which account for 10-15 per cent of all cases may represent up to 50 per cent of patients with AIDS. 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[4].

Pathogenesis

The postulated mechanisms by which the tubercule 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*) 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 reinfection. In India, the organism isolated from all intestinal lesions has been *Mycobacterium tuberculosis* and not *M.bovis* [5,6].

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[4]. In Bhansali's series, including 196 patients with gastrointestinal tuberculosis, ileum was involved in 102 and caecum in 100 patients[7]. Of the 300 patients in a study ileocaecal involvement was present in 162[8]. 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[9].

Pathology

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[10]. 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[11]. Cicatrical healing of these circumferential 'girdle ulcers' results in strictures. Occlusive arterial changes may produce ischaemia and contribute to the development of strictures[12]. Endarteritis also accounts for the rarity of massive bleeding in cases of intestinal tuberculosis. Shah *et al*[13] 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. 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.

Mesenteric lymph nodes may be enlarged, matted and may caseate. Characteristic granulomas may be seen only in the mesenteric lymph nodes. This is especially common in patients who have taken antitubercular therapy for some time. The reverse, *i.e.*, the presence of granulomas in the intestine and no granulomas in the draining lymph nodes is rare[9].

Hoon *et al*[10] originally classified the gross morphological appearance of the involved bowel into ulcerative, ulcerohyperplastic and hyperplastic varieties. Tandon and Prakash[9] 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. The bowel wall is thickened and the serosal surface is studded with nodules of variable size. 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[14].

Peritoneal tuberculosis occurs in 3 forms: (*i*) Wet type with ascitis; (*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. A combination of these types are also common.

Clinical features

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

Tuberculosis of the oesophagus

Oesophageal tuberculosis is a rare entity, constituting only 0.2 per cent of cases of abdominal tuberculosis[4]. Till 1997 only 58 cases had been reported in the English literature[16]. 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[17].

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[18]. It may also simulate gastric carcinoma. Chowdhary *et al*[19] reported the rare concurrence of carcinoma and tuberculosis of stomach in the same patient. The largest published series of duodenal tuberculosis reported 30 cases from India[20]. 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 remainder (27%) had a history of dyspepsia and were suspected of having duodenal ulcers. Two of these patients presented with hematemesis[20]. Other reported complications by various authors are perforation[21], fistulae (pyeloduodenal, duodenocutaneous, blind)[21], excavating ulcers extending into pancreas[22] and obstructive jaundice by compression of the common bile duct[23].

Duodenal tuberculosis is often isolated with no associated pulmonary lesions in more than 80 per cent cases[21]. Barium studies reveal evidence of segmental narrowing. Duodenal strictures are usually short but can involve long segments of the duodenum. CT may reveal wall thickening and/or lymphadenopathy. There is no specific picture of duodenal tuberculosis on endoscopy, and demonstration of granulomas or acid fact bacilli on endoscopic biopsy material is

unusual. Surgical bypass has been required in the majority of cases to relieve obstruction but successful endoscopic balloon dilatation (TTS balloon, Microvasive) of duodenal strictures has been reported by Vij *et al*[24] in two cases.

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[7].

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[7,25,26]. In a large series of 348 cases of intestinal obstruction, Bhansali and Sethna[25] found tuberculosis to be responsible for 54 (15.5%) cases; 33 cases were small bowel and 21 large bowel obstruction. Tandon *et al*[27] studied 186 patients over 5 yr and observed an increase in patients with more protracted course and subacute intestinal obstruction in recent years.

Tuberculosis accounts for 5-9 per cent of all small intestinal perforations in India, and is the second commonest cause after typhoid fever[28,29]. Evidence of tuberculosis on chest X-ray and a history of subacute intestinal obstruction are important clues. Pneumoperitoneum may be detected on radiographs in only half of the cases[30]. Tubercular perforations are usually single and proximal to a stricture[12]. Acute tubercular peritonitis without intestinal perforation is usually an acute presentation of peritoneal disease but may be due to ruptured caseating lymph nodes[7, 29].

Malabsorption is a common complication. Next to tropical sprue, it is the most important cause of malabsorption syndrome in India[7]. In a patient with malabsorption, a history of abdominal pain suggests the diagnosis of tuberculosis[30]. Pimparkar and Donde[31] studied 40 patients with malabsorption and divided then into those with and without bowel stricture. They performed glucose and lactose tolerance tests, d-xylose test, faecal fat and schillings test for B_{12} malabsorption and found them to be abnormal in 28, 22, 57, 60 and 63 per cent respectively in patients with stricture compared to 0, 0, 8, 25 and 30 per cent respectively without strictures. Tandon *et al*[32] also reported biochemical evidence of malabsorption in 75 per cent of patients with intestinal obstruction and in 40 per cent of those without it. The cause of malabsorption in intestinal tuberculosis is postulated to be bacterial overgrowth in a stagnant loop, bile salt deconjugation, diminished absorptive surface due to ulceration, and involvement of lymphatics and lymph nodes.

Segmental colonic tuberculosis

Segmental or isolated colonic tuberculosis refers to involvement of the colon without ileocaecal region, and constitutes 9.2 per cent of all cases of abdominal tuberculosis. It commonly involves the sigmoid, ascending and transverse colon[33]. Multifocal involvement is seen in one-third (28 to 44%) of patients with colonic tuberculosis[34,35]. The median duration of symptoms at presentation is less than 1 yr[36]. Pain is the predominant symptom in 78-90 per cent of patients and hematochezia occurs in less than one third[34,37]. The bleeding is frequently minor and massive bleeding is less common. Singh *et al*[36] reported rectal bleeding in 31 per cent of patients with colonic tuberculosis, and it was massive in 13 per cent. Bhargava *et al*[38], reported bleeding in 70 per cent cases. Overall, tuberculosis accounts for about 4 per cent of patients with lower gastrointestinal bleeding[29]. Other manifestations of colonic tuberculosis include fever, anorexia, weight loss and change in bowel habits. The diagnosis is suggested by barium enema or colonoscopy.

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%)[37]. The high frequency of rectal bleeding may be because of mucosal trauma caused by scybalous stool traversing the strictured segment. Digital examination reveals an annular stricture. The stricture is usually tight and of variable length with focal areas of deep ulceration. It is usually within 10 cm of the anal verge[36]. Associated perianal disease is very rare. Excessive fibrosis associated with the rectal inflammation results in an increase in presacral space. Overall rectal tuberculosis is rare and may occur in the absence of other lesions in the chest and small and large bowel[39,40].

Anal tuberculosis is less uncommon and has a distinct clinical presentation. Tubercular fistulae are usually multiple. Dandapat *et al*[41] reported that 12 out of 15 multiple fistulae were of tubercular origin, as compared to only 4 out of 61 solitary perianal fistulae. Shukla *et al*[42] reported that in India, tuberculosis accounted for up to 14 per cent of cases of fistula *in ano*. Anal discharge was present in all cases and perianal swelling in one third. Constitutional symptoms were not present in any patient[42]. Anal tuberculosis is also seen in paediatric patients[43].

Diagnosis and investigations

Paustian in 1964[4] stated that one or more of the following four criteria must be fulfilled to diagnose abdominal tuberculosis: (*i*) Histological evidence of tubercles with caseation necrosis; (*ii*) a good typical gross description of operative findings with biopsy of mesenteric nodes showing histologic evidence of tuberculosis; (*iii*) animal inoculation or culture of suspected tissue resulting in growth of *M. tuberculosis*; and (*iv*) histological demonstration of acid fast bacilli in a lesion.

These criteria must be kept in mind, and the diagnosis substantiated by adequate radiological and histopathological studies. Non specific findings include raised ESR, anaemia, and hypoalbuminaemia.

Radiological studies

Chest X-ray: Evidence of tuberculosis in a chest X-ray supports the diagnosis but a normal chest X-ray does not rule it out. Sharma *et al*[44] studied 70 cases of abdominal tuberculosis and found evidence of active or healed lesions on chest X-ray in 22 (46%). X-rays were more likely to be positive in patients with acute complications (80%)[44]. In Prakash's series of 300 patients, none had active pulmonary tuberculosis but 39 per cent had evidence of healed tuberculosis[8]. Tandon *et al*[27] found chest X-ray to be positive in only 25 per cent of their patients. Hence, about 75 per cent cases do not have evidence of concomitant pulmonary disease.

Plain X-ray abdomen: Plain X-ray abdomen may show enteroliths, features of obstruction *i.e.*, dilated bowel loops with multiple air fluid levels, evidence of ascitis, perforation or intussusception. In addition, there may be calcified lymph nodes, calcified granulomas and hepatosplenomegaly.

Small bowel barium meal: The features which may be seen: Accelerated intestinal transit; hypersegmentation of the barium column ("chicken intestine"), precipitation, flocculation and dilution of the barium; stiffened and thickened folds; luminal stenosis with smooth but stiff contours ("hour glass stenosis"), multiple strictures with segmental dilatation of bowel loops, may also be found; and fixity and matting of bowel loops.

Barium enema: The following features[44] 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*) Fold thickening and contour irregularity of the terminal ileum, better appreciated on double contrast study.

(*iii*)"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.

(*iv*) Loss of normal ileocaecal angle and dilated terminal ileum, appearing suspended from a retracted, fibrosed caecum ("goose neck deformity").

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

(*vi*) "Stierlin's sign" is a manifestation of acute inflammation superimposed on a chronically involved segment and is characterized by lack of barium retention in the inflammed segments of the ileum, caecum and variable length of the ascending colon, with a normal configured column of barium on either side. It appears as a narrowing of the terminal ileum with rapid empyting into a shortened, rigid or obliterated caecum.

(vii) "String sign" – persistant narrow stream of barium indicating stenosis.

Both Stierlin and String signs can also be seen in Crohn's disease and hence are not specific for tuberculosis.

Enteroclysis followed by a barium enema may be the best protocol for evaluation of intestinal tuberculosis.

Ultrasonography

Barium studies though accurate for intrinsic bowel abnormalities, do not detect lesions in the peritoneum. Ultrasound is very useful for imaging peritoneal tuberculosis.

The following features may be seen, usually in combination[45].

(*i*) Intra-abdominal fluid which may be free or loculated; and clear or complex (with debri and septae). Fluid collections in the pelvis may have thick septa and can mimic ovarian cyst.

(*ii*) "Club sandwich" or "sliced bread" sign is due to localized fluid between radially oriented bowel loops, due to local exudation from the inflammed bowel (interloop ascitis).

(*iii*) Lymphadenopathy may be discrete or conglomerated (matted). The echotexture is mixed heterogenous, in contrast to the homogenously hypoechoic nodes of lymphoma. Small discrete anechoic areas representing zones of caseation may be seen within the nodes. With treatment the nodes show a transient increase in size for 3-4 wk and then gradually reduce in size. Calcification in healing lesions is seen as discrete reflective lines. 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 ileocaecal region. The thickening is uniform and concentric as opposed to the eccentric thickening at the mesenteric border found in Crohn's disease and the variegated appearance of malignancy.

(*v*) Pseudokidney sign – involvement of the ileocaecal region which is pulled up to a subhepatic position.

Computed tomographic (CT) scan

Ileocaecal tuberculosis is usually hyperplastic and well evaluated on CT scan. In early disease there is slight symmetric circumferential thickening of caecum and terminal ileum. Later the ileocaecal valve and adjacent medial wall of the caecum is asymmetrically thickened. In more advanced disease gross wall thickening, adherent loops, large regional nodes and mesenteric thickening can together form a soft tissue mass centered around the ileocaecal junction[46]. CT scan can also pick up ulceration or nodularity within the terminal ileum, along with narrowing and proximal dilatation. Other areas of small and large bowel involvement manifest as circumferential wall thickening, narrowing of the lumen and ulceration. In the colon, involvement around the hepatic flexure is common. Complications of perforation, abscess, and obstruction are also seen.

Tubercular ascitic fluid is of high attenuation value (25-45 HU) due to its high protein content. Strands, fine septae and debris within the fluid are characteristic, but are better appreciated on ultrasonography[46]. Thickened peritoneum and enhancing peritoneal nodules may be seen.

Mesenteric disease on CT scan is seen as a patchy or diffuse increase in density, strands within the mesentery, and a stellate appearance. Lymph nodes may be interspersed. Omental thickening is well seen often as an omental cake appearance. A fibrous wall can cover the omentum, developing from long standing inflammation and is called omental line. An omental line is less common in malignant infiltration[47].

Caseating lymph nodes are seen as having hypodense centers and peripheral rim enhancement. Along with calcification, these findings are highly suggestive of tuberculosis. In tuberculosis the mesenteric, mesenteric root, celiac, porta hepatis and peripancreatic nodes are characteristically involved, reflecting the lymphatic drainage of the small bowel. The retroperitoneal nodes (*i.e.*, the periaortic and pericaval) are relatively spared, and are almost never seen in isolation, unlike lymphoma[46].

Colonoscopy

Colonoscopy is an excellent tool to diagnose colonic and terminal ileal involment but is still often underutilised. 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 ileocaecal 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[35]. Areas of strictures with nodular and ulcerated mucosa may be seen. Other findings are pseudopolypoid edematous folds, and a deformed and edematous ileocaecal 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[35-37].

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 submucosal involvement. Granulomas have been reported in 8-48 per cent of patients and caseation in a third (33-38%) of positive cases[36]. The yield of acid fast bacilli stains has been variable in studies. Culture positivity is not related to the presence of granulomas. Bhargava *et al*[35] reported positive cultures in 40 per cent of patients and concluded that routine culture of biopsy tissue increases the diagnostic yield. A combination of histology and culture of the biopsy material can be expected to establish the diagnosis in over 60 per cent of cases.

Immunological tests

Chawla *et al*[48] reported that an optical density (OD) of 0.81 on ELISA and fluoroscent coefficient of 2.56 on soluble antigen fluorescent antibody (SAFA) as cut-off gave positivity of 92 and 83 per cent, respectively, with 12 and 8 per cent false positives respectively. Bhargava *et al*[49] used competitive ELISA with monoclonal antibody against 38 Kd protein and found a sensitivity of 81 per cent, specificity of 88 per cent and diagnostic accuracy of 84 per cent. However, ELISA remains positive even after therapy, the response to mycobacteria is variable and its reproducibility is poor. Hence the value of immunological tests remains undefined in clinical practice[29].

Ascitic fluid examination

The ascitic fluid in tuberculosis is straw coloured with protein >3g/dl, and total cell count of 150-4000/µl, consisting predominantly of lymphocytes (>70%). The ascites to blood glucose ratio is less than 0.96[50] and serum ascitis 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. However Singh *et al*[46] in an earlier study cultured 1 litre of ascitic fluid after centrifugation and obtained 83 per cent culture positivity.

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*[51]. The levels in tuberculous ascitis were significantly higher than those in cirrhotic or malignant ascitis. Taking a cut off level of 33 U/l, the sensitivity, specificity and diagnostic accuracy were 100, 97 and 98 per cent respectively[51]. In the study by Bhargava *et al*[52], 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[53]. In coinfection with HIV the ADA values can be normal or low. Falsely high values can occur in malignant ascitis. High interferon-γ levels in tubercular ascitis have been reported to be useful diagnostically[54]. Combining both ADA and interferon estimations may further increase sensitivity and specificity.

Laparoscopic findings

Bhargava *et al*[14] studied 87 patients with high protein ascites, of which 38 were diagnosed as having tuberculosis. They found visual appearances to be more helpful (95% accurate) than either histology, culture or guinea pig innoculation (82, 3 and 37.5% sensitivity respectively). 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*) Fibroadhesive peritonitis with markedly thickened peritoneum and multiple thick adhesions fixing the viscera.

Management

All patients should receive conventional antitubercular therapy for at least 6 months including initial 2 months of rifampicin, isoniazid, pyrazinamide and ethambutol. A randomized comparison of a 6 month short course chemotherapy with a 12 month course of ethambutol and isoniazid (supplemented with streptomycin for the initial 2 wk) was conducted by Balasubramanium *et al*[55] at Tuberculosis Research Centre, Chennai, in 193 adult patients. Cure rate was 99 and 94 per cent in patients given short-course and the 12 month regimen respectively. However many physicians extend the treatment duration to 12 to 18 months.

The surgical treatment of intestinal tuberculosis has gone through three phases[56]. Bypassing the stenosed segment by enteroenterostomy or by ileotransverse colostomy was practiced when effective antitubercular drugs were unavailable, as any resectional surgery was considered hazardous in the presence of active disease. This practice however, produced blind loop syndrome, and fistulae and recurrent obstruction often occurred in the remaining segments. With the advent of antituberculous drugs, more radical procedures became popular in an attempt to eradicate the disease locally. These included right hemicolectomy with or without extensive removal of the draining lymph nodes and wide bowel resections. These procedures were often not tolerated well by the malnourished patient. Moreover the lesions are often widely spaced and not suitable for resection.

The recommended surgical procedures today are conservative. A period of pre operative drug therapy is controversial. Strictures which reduce the lumen by half or more and which cause proximal hypertrophy or dilation are treated by strictureplasty[56]. This involves a 5-6 cm long incision along the anti-mesenteric side which is closed transversely in two layers. A segment of bowel bearing multiple strictures or a single long tubular stricture may merit resection. Resection is segmental with a 5 cm margin.

Tubercular perforations are usually ileal and are associated with distal strictures. Resection and

anastomosis is preferred as simple closure of the lesions is associated with a high incidence of leak and fistula formation.

Two reports suggest that obstructing intestinal lesions may relieve with antitubercular drugs alone without surgery. Anand *et al*[57] reported clinical and radiological resolution of tuberculous strictures with drug therapy even in patients with subacute intestinal obstruction. They treated 39 patients with obstructive symptoms using medical therapy. At the end of one year 91 per cent showed clinical improvement, 70 per cent had complete radiological resolution and surgery was needed in only 3 cases (8%). Predictors of need for surgery were long strictures (>12 cm) and multiple areas of involvement[57]. Similar observations were made by Balasubramaniam *et al*[53]. The mean time required for the relief of obstructive symptoms was 6 months.

Summary

Abdominal tuberculosis is defined as infection of the peritoneum, hollow or solid abdominal organs with *Mycobacterium tuberculi*. The peritoneum and the ileocaecal region are the most likely sites of infection and are involved in the majority of the cases by hematogenous spread or through swallowing of infected sputum from primary pulmonary tuberculosis. Pulmonary tuberculosis is apparent in less than half of the patients. Patients usually present with abdominal pain, is usually made through a combination of radiologic, endoscopic, microbiologic, histologic and molecular techniques. Antimicrobial treatment is the same as for pulmonary tuberculosis Surgery is occasionally required.

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