GASTROENTEROLOGY IN INDIA

Abdominal tuberculosis: the Indian contribution

V K KAPOOR

Department of Surgical Gastroenterology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow 226 014

Tuberculosis can quite rightly be termed India's 'national disease'. The abdomen is the most common site of extra-pulmonary involvement. This article reviews the Indian data on abdominal tuberculosis. The data were obtained from articles retrieved through Medline by using abdominal/gastrointestinal/peritoneal tuberculosis in the title/abstract and India in the address.

Epidemiology

About a decade back, abdominal tuberculosis accounted for 0.8% of hospital admissions\(^1\) and 0.7% of surgical admissions,\(^2\) with similar prevalence in children admitted to a hospital.\(^3\) Both the incidence and severity of abdominal tuberculosis are expected to increase with the increasing incidence of HIV infection in India; HIV seroprevalence was significantly higher in patients with abdominal tuberculosis than in voluntary blood donors (16.6% vs 1.4%).\(^4\)

Clinical series

One of the earliest Indian reports of the condition was that by Anand, in 1956, of 50 patients undergoing right hemicolectomy.\(^5\) The largest Indian series, of 300 surgically managed patients over 12 years, was reported by Bhanwali\(^6\) he classified abdominal tuberculosis into gastrointestinal, peritoneal and lymph nodal types, depending on the predominant site of involvement. Acute and chronic presentations of the disease were observed in 19% and 28% of the patients, respectively. Another large surgical series, of 300 patients with intestinal lesions seen over 18 years (1959-1977), was reported by Prakash;\(^7\) 191 of these had subacute intestinal obstruction.

Tandon et al\(^8\) studied 186 patients over 5 years, starting 1977, and observed a change from an active disease with peritoneal and lymph nodal involvement, constitutional symptoms and associated pulmonary lesions in earlier cases, to a more protracted course with subacute intestinal obstruction in later cases. The authors hypothesized that this was due to generous but inappropriate use of anti-tubercular therapy. Singh et al\(^9\) recently reported a series of 145 patients seen in a medical unit - 29 of these had peritoneal involvement and the rest had gastrointestinal lesions.

Sharma et al\(^10\) reported 110 children managed over a 10-year period (1981-90) and highlighted the different spectrum of the disease in children - adenohypophysal and lymph nodal tuberculosis being more common than gastrointestinal disease. Narasimharao et al\(^5\) had earlier reported a higher frequency of gastrointestinal lesions in a series of 56 children. Singh et al\(^11\) reported the pathoge-
spleenism have also been reported. Derangement of liver function tests and liver biopsy changes in abdominal tuberculosis have been described.

**Complications**

Tuberculosis is usually regarded as a chronic disease; however, about one-fourth to one-third of patients with abdominal tuberculosis may present with an acute abdomen due to various complications. It is a common cause of intestinal obstruction and perforation; it may uncommonly cause peritonitis and lower gastrointestinal bleeding.

Malabsorption is common, more so in the presence of subacute intestinal obstruction. It is caused by bacterial overgrowth and bile salt deconjugation in the upper small intestine. Yachha et al. found that intestinal tuberculosis accounted for 9% of cases of malabsorption in children above the age of 2 years. Malabsorption, if associated with abdominal pain, should lead to a suspicion of tuberculosis. Desai et al. demonstrated disturbances of bile salt metabolism in intestinal tuberculosis; whether this is in any way responsible for the higher frequency of gallstones in these patients is not known.

Small intestinal perforation has been reported in 8%-15% of patients with abdominal tuberculosis. Tuberculosis is the second common cause of small intestinal perforation, after typhoid. History of subacute intestinal obstruction or evidence of tuberculosis (active or healed) on chest X-ray should suggest the diagnosis in a patient presenting with peritonitis. An increased incidence of perforation on anti-tubercular therapy has also been observed. Acute tubercular peritonitis without intestinal perforation is usually an acute manifestation of peritoneal disease but may be due to ruptured caseating lymph nodes.

Small amount of bleeding per rectum is common in intestinal tuberculosis especially of the ulcerative type, but massive bleeding is rare. Tuberculosis accounts for about 4% of patients with lower gastrointestinal bleeding. Some large series did not report any case of massive lower gastrointestinal bleeding. Vimala et al. reported that only 2 of 300 patients with abdominal tuberculosis seen over a 16-year period had massive lower gastrointestinal bleeding caused by ulcerated lesions in the ileocecal region; both these patients had previous recurrent attacks of melena. Singh et al. reported rectal bleeding in 31% of patients and it was massive in 13%, but all of them had colonic tuberculosis. Massive lower gastrointestinal bleeding due to tuberculosis usually requires surgery, but control by transarterial embolization has been reported.

Misra et al. reported coexisting tuberculosis and carcinoma of the colon and Jain et al. observed a higher frequency of carcinoma in patients with colonic tuberculosis, suggesting a need for epidemiological and histological studies into the etiological relationship between the two.

**Abdominal tuberculosis and Crohn’s disease**

Das and Shukla did not see a single patient with Crohn’s disease during the period over which they saw 182 patients with abdominal tuberculosis; Prakash, on the other hand, reported that 13 of 281 patients with intestinal strictures had Crohn’s disease. Around this period (1959-77), he managed 300 patients with abdominal tuberculosis. The differentiation between abdominal tuberculosis and Crohn’s disease has been elucidated in detail.

Crohn’s disease is rare in India but many cases are probably missed or misdiagnosed as tuberculosis.

**Investigations**

The clinical diagnosis of abdominal tuberculosis remains elusive and its accuracy has remained at around 50% from 1976 through 1985 to 1992. Definitive diagnosis of tuberculosis needs microbiological proof. Methods like demonstration of tubercle bacilli in the thoracic duct lymph are, however, of historical interest only. Since abdominal tuberculosis is paucibacillary, the yield of organisms is low and characteristic histological changes are taken as diagnostic. It is important to subject the tissues to culture examination also, as mycobacteria may be grown even if histology does not reveal granulomas. All human infections reported from India have been due to *Mycobacterium tuberculosis*. 56-57

One of the most significant Indian contributions to abdominal tuberculosis is the study by Tandon and Prakash on the pathology of intestinal tuberculosis. Though the hallmark of diagnosis is demonstration of caseating granulomas on histological examination, non-caseating granulomas may be seen in some cases due to low virulence of the organism or high resistance of the host, or due to previous
Abdominal tuberculosis: the Indian contribution

Kapoor

anti-tubercular therapy. This article described in detail the effect of anti-tubercular therapy on histological changes, an aspect studied earlier by Wig et al.\(^7\) Granulomas have been reported in only 40% of patients with abdominal tuberculosis,\(^24,72\) and may be present only in lymph nodes and absent in intestinal lesions.

Operative findings suggestive of tuberculosis, but without histological or microbiological evidence, were observed in one-fourth of patients.\(^6\) The entity of nonspecific chronic inflammatory lesion observed by Anguli and Menon\(^73\) is common. Prakash\(^9\) reported that 56 of 281 ulcero-constrictive lesions of the bowel were neither tubercular nor Crohn's in etiology. It remains unexplained whether this nonspecific enteritis is a healed tuberculous lesion or a result of an ischemic insult due to tuberculosis.\(^74\) Prabh\(\) et al\(^75\) recently reported gastric metaplasia in one-third of patients with intestinal tuberculosis; this epithelium is not frequently infected by Helicobacter pylori.

Most reports mention that the yield of organisms in the ascitic fluid in peritoneal tuberculosis is low, but Singh et al\(^71\) were able to culture acid-fast bacilli in 83% of patients using one liter of fluid. Adenosine deaminase (ADA) levels were estimated in the ascitic fluid by Dwivedi et al.\(^76\) The levels in tuberculous ascites were significantly higher than those in cirrhotic or malignant ascites. At a cut-off level of 33 U/L, the sensitivity, specificity and overall diagnostic accuracy were 100%, 97% and 98%, respectively. Bhargava et al\(^77\) also found the serum and ascitic fluid ADA activity to be significantly higher in patients with peritoneal tuberculosis as compared to those with cirrhosis and peritoneal malignancy; serum and ascitic fluid levels above 54 U/L and 36 U/L, respectively and ascitic fluid/serum ADA ratio of > 0.985 were considered suggestive of tuberculosis.

Laparoscopy as a diagnostic aid in tuberculosis was first reported by Udwalla.\(^78\) Bhargava et al\(^79\) later described the laparoscopic findings in detail. Shukla et al\(^80\) reported a small-incision peritoneal biopsy under local anesthesia.

Fine-needle aspiration cytology (FNAC) of abdominal lump has been shown to be accurate for diagnosis;\(^81\) culture of the aspirate adds to the sensitivity.\(^82\) Radhika et al\(^83\) demonstrated acid-fast bacilli on Ziehl-Neelsen staining in 47 of 105 FNAC smears.

Bhargava et al\(^84,85\) first reported colonoscopic findings in ileocecal and colonic tuberculosis — edematous and deformed ileocecal value, narrowed cecum, mucosal nodules and ulceration. Similar colonoscopic findings in large intestinal lesions have been described in various series.\(^23,24,66\) Bhargava et al\(^72\) found that combined histological and bacteriological examination of the endoscopic biopsy specimen gave the diagnosis in 60% of patients. At colonoscopy, performing multiple (4-8) biopsies, or FNAC in addition to biopsies, increases the diagnostic yield because the lesions are submucosal.\(^86\) Anand et al\(^87\) recently reported the use of polymerase chain reaction on endoscopic biopsy specimens.

Kapoor et al\(^88\) reported evidence of tuberculosis (active or healed) on chest X-ray in 46% of patients; chest X-ray was positive in 80% of patients with acute complications. Enteroliths form proximal to stricture\(^89\) and may be seen on plain abdominal X-ray.\(^90\) Radiological studies (barium meal follow-through and barium enema) are the mainstay of diagnosis. They, however, have limitations, e.g., false-positive and false-negative studies, inability to differentiate between intestinal lesions and peritoneal adhesions, and inaccuracy in detecting multiple lesions and in localizing the site of lesion.\(^1,83,91\) Kedar et al\(^92\) have reported ultrasonicographic findings in abdominal tuberculosis.

The diagnostic value of immunological tests like soluble antigen fluoroscent antibody (SAFA)\(^93,94\) and enzyme-linked immunosorbent assay (ELISA)\(^95,96,97\) has been evaluated by many workers.\(^98\) Chawla et al\(^99\) reported that an optical density (OD) of 0.81 on ELISA and fluorescent coefficient of 2.56 on SAFA as cut-off gave positivity of 92% and 83%, respectively, with 12% and 8% false positives, respectively. Bhargava et al\(^100\) reported ELISA sensitivity of 81%, specificity of 88% and diagnostic accuracy of 84% using a cut-off level of 0.7 OD. Both these groups concluded that none of the serological tests are perfect and it is unlikely that these alone will provide the diagnosis in all cases. In areas where tuberculosis is common, these tests may, however, support a clinical and radiological diagnosis in the absence of histological or microbiological confirmation.

Various authors\(^24,96\) advocate a therapeutic trial with antitubercular therapy (ATT) on clinical or radiological suspicion alone, but this may not be advisable especially because diseases like adenocarcinoma, lymphoma and immuno-proliferative small intestinal disease (IPSID)\(^100\) can be misdiagnosed as intestinal tuberculosis. Bhanvani\(^9\) observed that when diagnostic studies are equivocal, it is prudent to resort to laparotomy, which also gives an opportunity for surgical management of intestinal lesions.

Management

Most surgical reports advocate surgical treatment for intestinal lesions.\(^9\) Anand,\(^99\) for the first time, reported resolution (clinical and radiological) of tuberculous strictures with ATT even in patients with subacute intestinal obstruction. Similar observations have been made by Balasubramanian et al.\(^101\) Singh et al\(^104\) reported resolution of colonic lesions with ATT. Bhanvani\(^9\) recommended initial conservative management of acute tubercular abdomen; half of the patients, however, did not improve and required delayed emergency surgery, which was associated with a high (23%) mortality. Better results have been
subsequently obtained with emergency surgical management of acute tubercular abdomen.38

The initial surgical approach to abdominal tuberculosis was to perform bypass procedures to relieve intestinal obstruction as resectional surgery was considered hazardous. With the availability of specific anti-tubercular drugs, the approach shifted towards radical surgical procedures like right hemicolectomy.102 This was performed in a significant number of patients reported by Bhanalsi and Prakash.3 but was performed in only 2 of 42 reported recently by Dandapat et al.103 Surgical procedures presently recommended are conservative in nature104 — limited (segmental) resection with 5 cm margin on either side for ileocecal lesions21, 105,106 and stricureplasty107 or ileoplasty108 for ileal strictures.

Stricureplasty can be performed for active strictureing tubercular enteritis also.107 Strictures with near-total obstruction of the lumen and multiple strictures in a short segment need resection but multiple resections should be avoided to preserve the length of the small bowel. Early strictures with adequate lumen may be left alone or dilated.107,108 Joshi22 described a U anastomosis (like Finney’s pyloroplasty) for intestinal strictures. Parikh104 described enteroplasty even in the presence of a proximal perforation also, the incision dividing the stricture encircling the perforation. Bypass procedures should be avoided as far as possible. Bhanalsi6 and Prakash3 reported reoperation and resection in patients who had undergone a bypass initially.

Bhanalsi6 recommended that surgery should be preceded by ATT whenever possible, but Prakash7 advocated preoperative ATT only in patients with tubercular toxemia and those with active tuberculosis elsewhere. Balasubramanian et al101 have recently shown that even a 6-month (short course) regimen of ATT is effective in the treatment of abdominal tuberculosis.

In an early prospective randomized study, Singh et al11 had shown that addition of corticosteroids to ATT prevented late fibrotic complications in patients with tuberculous peritonitis. None of the 23 patients who received prednisone 30 mg daily for three months had adhesive intestinal obstruction; in comparison 3 of 16 patients receiving ATT alone required surgery for adhesive intestinal obstruction during a 3-year follow up. The role of corticosteroids in patients with tuberculous peritonitis has not been studied subsequently.

Prognosis

Despite advances in diagnostic methods, newer anti-tubercular drugs and better surgical techniques, mortality continues to be high. Bhanalsi6 had reported overall mortality rate of 7%; one of the reasons was a large number of bypass procedures performed in this series. Dandapat et al.7 had reported 12% mortality; emergency surgery which was associated with 25% mortality accounted for most of the deaths. Emergency surgery in Bhanalsi’s6 experience carried 23% mortality. In a recent experience, Sircar et al109 reported 11% mortality in 298 patients. Jain et al10 reported 2 deaths among 17 patients with acute tubercular abdomen. Mortality in children is also high and was reported to be 9%.27

Research

Much is not known about the etiopathogenesis of abdominal tuberculosis. Since a majority of patients with intestinal tuberculosis do not have associated or antecedent pulmonary tuberculosis, Wig et al10 and Tandon et al6 felt that intestinal tuberculosis is usually primary. Rao et al111 suggested a hypersensitivity phenomenon as the cause for ileocecal disease. Das and Shukla112 produced hypertrophic tuberculosis by injecting infected ileocecal tissue intraperitoneally into guinea pigs. Prakash85 described changes ranging from irregularity to complete occlusion of the vasa recta in ulcero-constrictive lesions. Based on angiographic findings, Shah et al113 hypothesized a role for ischemia in tuberculosis — ulcerative lesions have hypervascularity, which may explain associated bleeding, whereas occlusive arterial changes may produce strictures. Kuvajerwala et al114 studied histologically the mesenteric vessels in resected intestinal specimens from patients with obstruction and perforation due to tuberculosis; they found changes including granulomas, thrombi and fibrosis in 45 of 68 patients, and postulated that these changes lead to ischemia of the bowel contributing to the development of ulcer, fibrosis, strictures and perforation. Sarode et al115 reported hyperaggregation of platelets in 88% of patients with intestinal tuberculosis and hypothesized this to be the cause of chronic inflammation.

The role of ATT in resolving gastrointestinal lesions (especially obstructing ones) needs prospective evaluation. Many myths about the disease, e.g., why some patients have involvement of the gastrointestinal tract whereas others have parietal or lymph nodal lesions; why some patients have ulcero-stricture lesions and others ulcero-hypertrophic; why some patients with parietal disease have ascites but others have adhesions116 still need to be resolved.

References

5. Anand SS. Hypertrophic ileocecal tuberculosis in India.
Abdominal tuberculosis: the Indian contribution

49. Tandon RK, Sarin SK, Berry M, Malik RB, Kapur BML. Gall stone disease and lithogenicity of bile in intestinal...
Kapoor

Abdominal tuberculosis: the Indian contribution

74. Tandon HD. The pathology of intestinal tuberculosis and distinction from other diseases causing stricture. Trop Gastroenterol 1981; 2: 77-93.
90. Gupta NM, Pinjla RK, Talwar BL. Celiac enterocolitis.
Abdominal tuberculosis: the Indian contribution


100. Wig KL, Gupta SP, Chitkara NL. A study of hyperplastic intestinal tuberculosis and allied abdominal conditions. Indian J Tuberc 1956; 3: 105-10.


Correspondence to: Dr Kapoor. Fax (622) 44 0017, 44 0973. E-mail: vikkepoor@sppgl.ren.nic.in