Abdominal tuberculosis: still a problem today

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Summary

Thirty cases of abdominal tuberculosis were diagnosed over an 18-year period, 26 being in immigrants; the mean age at presentation was 33 years. There were 25 cases of tuberculous peritonitis, two of tuberculous mesenteric lymphadenitis, and one each of gastric tuberculosis, colonic tuberculosis and tuberculous ischiorectal abscess. The presenting symptoms were of vague ill-health and not diagnostic, and the most common findings were of pyrexia, abdominal tenderness and ascites. The most common haematological abnormalities were raised ESR (22 patients) and mild anaemia (15 patients). Most commonly, diagnosis was made by formal laparotomy (19 cases) with biopsy (18 cases); histological examination gave a positive diagnosis in all 18 cases, but bacteriological examination yielded the tubercle bacillus in only 10 (56%). There were 2 deaths in the series, not directly related to the abdominal tuberculosis.

In view of its infrequent and vague presentation, care is required to avoid overlooking the diagnosis of abdominal tuberculosis, particularly in the immigrant population.

Introduction

The incidence of gastrointestinal tuberculosis in Britain dropped dramatically in the 1950s following the discovery of antituberculous chemotherapy, the introduction of effective pasteurization of milk, the tuberculin testing of dairy herds and the recognition of Crohn's disease (which had previously often been erroneously diagnosed as tuberculosis). Since then there has been a slight increase in its incidence, and this probably reflects the influx of immigrants from third world countries, particularly as recent studies have demonstrated that the highest incidences occur in areas with a large immigrant population. Nevertheless, intra-abdominal tuberculosis is not frequently seen in the UK and, as its presenting symptoms are often vague, there may be undue delay before the diagnosis is finally made. The present survey of cases presenting to a London hospital over an 18-year period emphasizes certain aspects of this condition.

Methods

During the period 1966–1984, 30 cases of gastrointestinal tuberculosis presented to King's College Hospital, London. The age range of the patients was 11 to 61 years (mean 33). The male:female ratio was 23:7. Of the 30 patients, only 4 were born in the UK; the origins of the remainder were India (8), Middle East (7), West Indies (3), Africa (2) and one each from Pakistan, China, Iran, Spain, Sierra Leone and Sri Lanka. Five patients arrived in the UK with the disease, and of the remaining 21 immigrants, 12 had lived in Britain for periods varying between 18

months and 17 years. It was not possible to determine the period of domicile in Britain for the other 9 immigrants.

The majority of patients (25 of 30, i.e. 83%) presented with tuberculous peritonitis, and of the 5 remaining patients, 2 had tuberculous mesenteric lymphadenitis, one had colonic tuberculosis, one had gastric tuberculosis and one presented with a tuberculous ischiorectal abscess. A wide variety of presenting symptoms were seen, although the most common were fever, abdominal pain, loss of weight and malaise. On examination the most common findings were pyrexia, abdominal tenderness and ascites. Table 1 shows the presenting symptoms and signs. The duration of symptoms prior to admission varied considerably between four weeks and one year, although only two of the 30 patients gave a history longer than six months.

Laboratory investigations were not diagnostic. The most consistent abnormality was a raised ESR; this was greater than 20 mm/hour in 22/26 (85%) and greater than 40 mm/hour in 16 (62%). In 2 of the 7 females the haemoglobin was less than 11 g/100 ml, and in 13 of the 23 males the haemoglobin was less than 12 g/100 ml. In only 3 patients from the whole series was the white count greater than 11×10^9/ml. A tuberculin test (Mantoux or Tine) was performed in 17 cases and found to be positive in only 11 (65%). Chest X-rays were performed on all patients and in 50% there were changes consistent with active or previous pulmonary tuberculous infection. A small bowel meal was carried out in 12 cases: in 7 no abnormality was demonstrated, but in 5 radiological features suggestive of gastrointestinal tuberculosis were present, i.e. mucosal oedema of the small bowel, ileal strictures, and in one case a contracted caecum and ascending colon in the presence of a colonic stricture.

Results

Seventeen out of 25 patients with tuberculous peritonitis underwent laparotomy (Figure 1), and the classical finding of multiple white nodules scattered over the peritoneal surface and greater omentum was seen in 16 cases. Peritoneal biopsies from these 16 cases all showed histological changes of tuberculosis, but only 8 yielded positive cultures for the tubercle bacillus. In one case the plastic variety of tuberculous peritonitis was found, and with the whole of the bowel matted together by dense adhesions no biopsies were taken. This patient was therefore treated on clinical suspicion alone (which included a positive Tine test and X-ray evidence of pulmonary tuberculosis), and showed a good response to antituberculous therapy.

Of the 8 cases who did not undergo laparotomy, 2 were diagnosed by laparoscopy with positive biopsy; 4 had clinically detectable ascites tapped,

Table 1. Presenting symptoms and signs in diagnostic categories

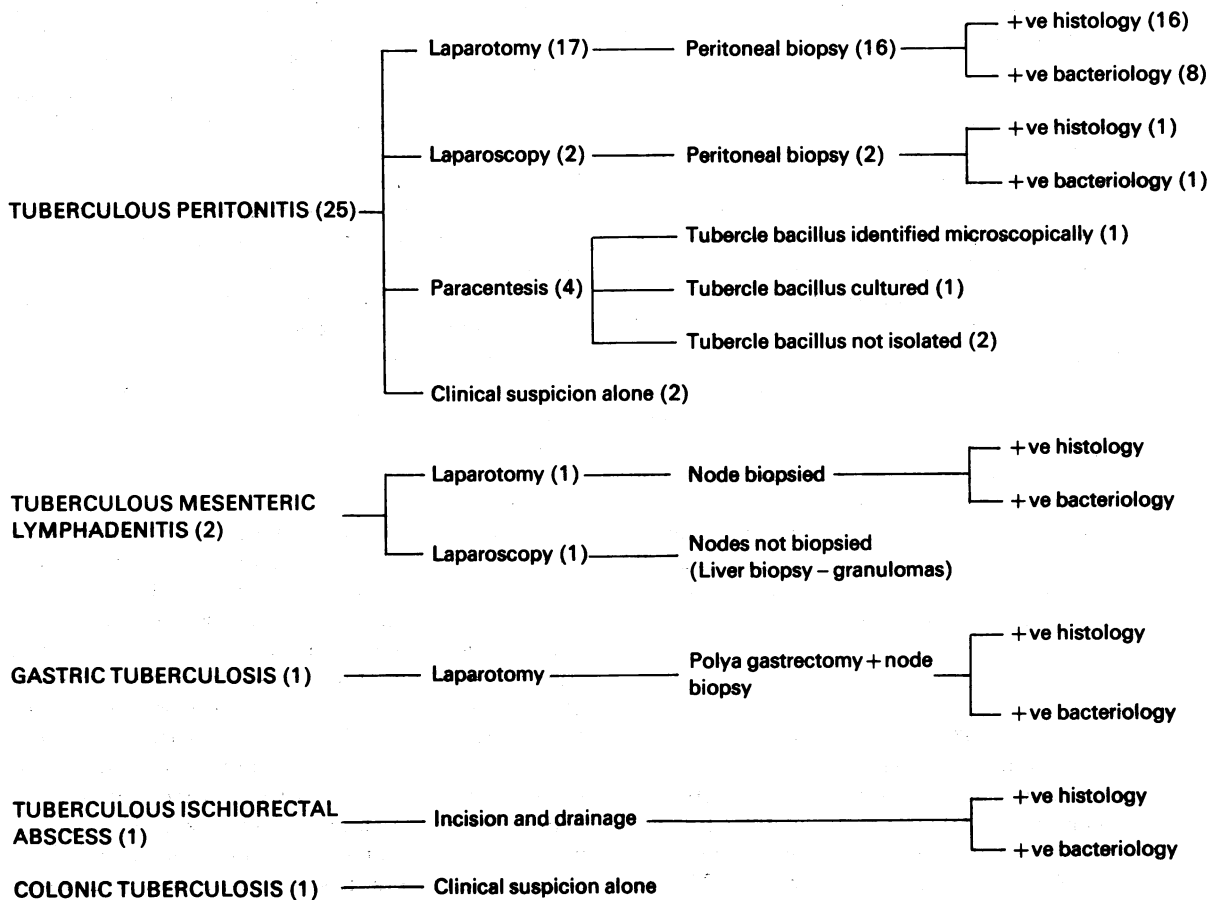|	Ischiorectal abscess (n = 1)	Colonic tuberculosis (n = 1)	Gastric tuberculosis (n = 1)	Tuberculous mesenteric lymphadenitis (n = 2)	Tuberculous peritonitis (n = 25)
Symptoms					
Fever				2	17
Pain		1	1	2	18
Weight loss		1	1	2	20
Swelling				1	9
Malaise		1		1	19
Cough	1	1	1	1	7
Diarrhoea		1			7
Vomiting		1			2
Others	Discharging perianal sinus				Infertility
Signs					
Fever			1	2	22
Tenderness		1	1	1	13
Ascites					14
Mass				2	4 (1 = Ca liver)
Duration of symptoms	1/12	5/12	5/52	6/12 6/52	4/52 to 1 yr

Figure 1. Diagnostic procedures in 30 cases of abdominal tuberculosis

but in only 2 was there subsequent identification of the tubercle bacillus; and the remaining 2 were treated on suspicion alone, both making a full recovery. One of the patients undergoing laparoscopy was completely asymptomatic and was subjected to examination during investigations for primary infertility. Both peritoneal biopsies and endometrial curettings demonstrated histological characteristics of tuberculosis.

Of the patients with tuberculous mesenteric lymphadenitis, one underwent a laparotomy and node biopsy which yielded positive histological and bacteriological proof of the diagnosis; the other underwent a laparoscopy which showed a mass of mesenteric glands, and a liver biopsy demonstrated granulomata.

The patient with gastric tuberculosis was diagnosed following a laparotomy for possible carcinoma of the stomach. At operation the antrum of the stomach and the duodenum were found to be thickened. A Polya gastrectomy was performed and enlarged omental lymph nodes biopsied. Subsequent histological examination demonstrated caseous granulomata in both the resected stomach and omental lymph nodes, and tubercle bacilli were cultured from the lymph nodes. The tuberculous ischiorectal abscess was diagnosed histologically and bacteriologically, following the incision and drainage of the abscess in an otherwise asymptomatic patient. The diagnosis of colonic tuberculosis was made on the basis of the clinical presentation and the radiological changes previously described.

In all cases treatment was with standard antituberculous chemotherapy, in accordance with sensitivity results whenever these were available. Twenty-eight of the 30 patients showed a good response to treatment, thus validating the diagnosis in those without histological or bacteriological proof. Two patients died. One was a 42-year-old Arab man with a coincidental carcinoma of the liver, and the other a 27-year-old Indian woman who developed rifampicin-induced acute liver failure.

Discussion

Tuberculosis has affected the human race ever since the neolithic period, as evidenced by skeletal remains[1]. In the 4th century BC Hippocrates labelled pulmonary tuberculosis 'phthisis' and also pointed out intestinal complications associated with this condition[2]. Since then a review of the historical literature has revealed several instances in which autopsy reports have described changes which in modern interpretation would suggest gastrointestinal tuberculosis[1,2]. It was not until after 1882, however, when Koch identified the tubercle bacillus, that the true nature of these changes could be clearly identified.

Abdominal tuberculosis is regarded as a rarity these days, and consequently may not be thought of immediately when a patient first presents with vague abdominal symptoms. Thirty cases have been admitted in 18 years to King's College Hospital, which serves a resident population of 218 000[3] and a catchment population 239 000[4], of whom 21.6% live in immigrant-headed households[5]. Twenty-six (87%) of our cases were immigrants, 9 coming from the Indian subcontinent. Previous reports have pointed out that the majority of the cases in the UK are immigrants[6-10]. The wide range of ethnic groups represented in this series demonstrates that no race is immune from this disease. Four cases were classed as British, although 2 of these were born to immigrant parents (West Indian and Mauritian), leaving only 2 representatives of the indigenous population. Although the immigrant community usually provides the majority of patients with the disease, the occurrence of gastrointestinal tuberculosis must not be overlooked in British patients, as Wales et al.[11] and Shukla and Hughes[12] have reported series in which the indigenous population yields most cases.

Twenty-one of the 26 immigrants in the present series developed symptoms whilst living in the UK, implying that they either contracted the disease here or reactivated a quiescent focus from a previous infective episode. Only one patient gave a history of contact with active disease (a brother) and, as the incidence of tuberculosis is so much higher in many of these other countries of origin, there is good reason for believing that in many instances the immigrant arrives with quiescent disease which reactivates at a later date, perhaps due to some stress-induced alteration in the patient's immune response[2].

Gastrointestinal tuberculosis most commonly occurs in the fourth decade[13], and the mean age of 33 in our series reflects this. Our male to female ratio of 23:7 is higher than in other series, but confirms the opinion that in Britain the disease is more common in males[2,6,7,9,11]. This observation is the reverse of the female-dominated series from Third World countries[14,15], and may be due to the larger number of male immigrants living in overcrowded conditions in this country. In addition, in many Third World countries, the women assume a nursing role among infected members of the family[16].

Gastrointestinal tuberculosis often induces vague symptoms, and this may partly explain the delay in presentation of some patients (four weeks to one year in this series). Our findings of loss of weight, abdominal pain, malaise and fever as the most common presenting symptoms are in agreement with other published series, as are our findings of a raised temperature, abdominal tenderness and ascites as the most common physical findings. Unfortunately, there is no test that is pathognomonic for tuberculosis. Several blood tests are often abnormal, but they are not invariably present and all are non-specific. A raised ESR and a mild anaemia are believed to be the most common haematological abnormalities[13], and this was certainly the finding in our cases. Leukocytosis was present in only 3 of our patients. As most gastrointestinal tuberculosis seen today is secondary, evidence of a primary site must be sought elsewhere. The chest is the most common site, but in our series sputum samples were particularly unrewarding and an abnormal chest X-ray was only seen in 50% of cases. This figure is actually higher than in several other series, in which evidence of pulmonary disease was found in less than 50%[2,7,9,10,12], although Wales et al.[11] found evidence of pulmonary disease in 12 of their 14 patients. We did not find the small bowel meal examination particularly helpful, as changes were demonstrated in only 5 of 12 cases. The X-ray appearances were difficult to differentiate from Crohn's disease and diagnosis tended to be influenced by the overall clinical picture. Unfortunately, tuberculin

testing is not always positive in patients with active gastrointestinal tuberculosis. Our finding of a positive result in 11 (65%) of 17 patients tested confirms this. The varying percentages of positive tests in other series have been commented upon by Findlay[16].

Abdominal tuberculosis usually presents in one of three forms: tuberculous peritonitis, involvement of the gastrointestinal tract per se, or a combination of both. Gastric tuberculosis is rare; Mukerjee and Singal[17] reported an incidence of 2.8% in their series of 500 patients. Barium meal findings may mimic carcinoma of the stomach and, if endoscopic biopsies are not diagnostic, then the true nature of the pathology may not become apparent until the gastrectomy specimen is examined histologically. This was the sequence of events in our case.

Tuberculous colitis in the absence of ileocaecal involvement is very uncommon[18-20]. When it presents as a stricture, differentiation from a colonic neoplasm is often difficult; stool cultures, colonoscopy, and biopsy may be helpful. In our single case stool cultures were negative, and the diagnosis was based upon the clinical picture, the radiological appearances, and a strongly positive Mantoux test. Therapy was instituted and a good response seen.

The case of tuberculous ischiorectal abscess emphasizes the importance of taking biopsies from all perianal abscesses. The diagnosis, not entertained preoperatively, was made only when positive histology and bacteriology were obtained.

The patient in this series with tuberculous histology in both endometrial curettings and laparoscopic peritoneal biopsies represents a case of tuberculous peritonitis where the organism has gained entry via the female genital tract. This patient, a 26-year-old Egyptian girl, had been investigated elsewhere for primary infertility for six years. The diagnosis was only established at laparoscopy when scattered tubercles were seen over the pelvic peritoneum.

The absolute diagnosis of abdominal tuberculosis requires either positive histological or bacteriological proof, the former usually being the more successful[10]. In the present series all peritoneal biopsies taken in 16 cases of tuberculous peritonitis yielded positive histology, but positive bacteriology was reported in only 50%. The success rate of bacterial identification in other series has been variously reported as 4%[10], 58%[2] and 75%[12]. The best means of obtaining biopsy material from cases of tuberculous peritonitis is debatable. Abdominal paracentesis is the least invasive method of all, but is not reliable. If the organisms are not seen on direct microscopic examination of the fluid, then subsequent culture takes six weeks and may, even then, not be positive[7,13,21].

Levine[22] claimed 100% diagnostic success with blind peritoneal needle biopsy, although there was one death from the technique[23]. However, other series have shown lower success rates[21,24,25] and the procedure can only be performed safely when ascites is present. A laparotomy is probably the most reliable as a full examination of the peritoneal cavity can be carried out and biopsies taken with ease. Such a procedure yielded a definite diagnosis of tuberculous peritonitis in 16 (94%) of 17 cases in our series. In order to avoid a major incision and general anaesthesia, Das and Shukla[26] described a

less invasive form of peritoneal biopsy via a right iliac fossa grid-iron incision performed under local anaesthesia. Access through this incision, however, is limited, and problems may arise if there is no suitably placed lesion for biopsy. Laparoscopy and target biopsy has been advocated by Wolfe et al.[27], a successful diagnosis being obtained in 8 (73%) of 11 cases in their series. In experienced hands, laparoscopy may be the procedure of choice as it is minimally invasive, but it is probably unsuitable for cases of plastic tuberculous peritonitis.

Following the introduction of successful antituberculous chemotherapy, the mortality from abdominal tuberculosis has decreased significantly[1] and, in view of this, it is important that the diagnosis be made quickly so that effective treatment can be instituted. In this series neither of the 2 deaths was attributable directly to tuberculosis. Acute liver failure secondary to rifampicin, the cause of one death in our series, is well recognized, and treatment should be stopped immediately there is any evidence of impaired liver function.

References
1 Dineed P, Homan WP, Grafe WR. Tuberculous peritonitis. Ann Surg 1976;184:717-22
2 Addison NV. Abdominal tuberculosis – a disease revived. Ann R Coll Surg Engl 1983;65:105-11
3 OPCS-based mid-year (1981) resident population estimate, corrected for split wards by Statistics and Operational Research Department, SETRHA. London: OPCS, 1981
4 OPCS-based mid-year (1981) resident population estimate, corrected for split wards and adjusted for in-patient flows by Statistics and Operational Research Department, SETRHA. London: OPCS 1981
5 OPCS Census. Small area statistics. London: OPCS, 1981
6 Findlay JM, Addison NV, Stevenson DK, Mirza ZA. Tuberculosis of the gastrointestinal tract in Bradford, 1967-1977. J R Soc Med 1979;72:587-90
7 Khoury GA, Payne CR, Harvey DR. Tuberculosis of the peritoneal cavity. Br J Surg 1978;65:808-11
8 Kaufman HD, Donovan I. Tuberculous disease of the abdomen. J R Coll Surg Edinb 1974;19:377-80
9 Mandal BK, Schofield PF. Abdominal tuberculosis in Britain. Practitioner 1976;216:683-9
10 Lambrianides AL, Ackroyd N, Shorey BA. Abdominal tuberculosis. Br J Surg 1980;67:887-9
11 Wales JM, Mumtaz H, MacLeod WM. Gastrointestinal tuberculosis. Br J Dis Chest 1976;70:39-57
12 Shukla HS, Hughes LE. Abdominal tuberculosis in the 1970s: a continuing problem. Br J Surg 1978;65:403-5
13 Findlay JM. Tuberculosis of the gastrointestinal tract. In: Allan RN, Keighley MRB, Alexander-Williams J, Hawkins C, eds. Inflammatory bowel diseases. Edinburgh: Churchill Livingstone, 1983:562-71
14 Pimparkar BD. Abdominal tuberculosis. J Assoc Physicians India 1977;25:801-11
15 Wig KL, Chitkara NL, Gupta SP, Kishore K, Manchanda RL. Ileocecal tuberculosis with particular reference to isolation of Mycobacterium tuberculosis. Am Rev Respir Dis 1961;84:169-78

16 Findlay JM. Gastrointestinal tuberculosis. In: Taylor S, ed. *Recent advances in surgery, vol. 10*. Edinburgh: Churchill Livingstone, 1980:225-40

17 Mukerjee P, Singal AK. Intestinal tuberculosis, 500 operated cases. *Proc Assoc Surg East Africa* 1979;2: 70-5

18 Ahuja SK, Gaiha M, Sachdev S, Mahesh Wari HB. Tuberculous colitis simulating ulcerative colitis. *J Assoc Physicians India* 1976;24:617-9

19 Abrams JS, Holden WD. Tuberculosis of the gastro-intestinal tract. *Arch Surg* 1964;89:282-3

20 Brenner SM, Annes G, Parker JG. Tuberculous colitis simulating non specific granulomatous disease of the colon. *Am J Dig Dis* 1970;15:85-92

21 Mehrotra MP, Mathur KS, Agarwal AN. Value of peritoneal biopsy in clinically diagnosed cases of abdominal tuberculosis. *J Assoc Physicians India* 1966;14:625-8

22 Levine H. Needle biopsy diagnosis of tuberculous peritonitis. *Am Rev Respir Dis* 1968;97:889-94

23 Levine H. Needle biopsy in diagnosis of tuberculous peritonitis. *Am Rev Respir Dis* 1968;98:519

24 Singh MM, Bhargava AN, Jain KP. Tuberculous peritonitis: an evaluation of pathogenic mechanisms, diagnostic procedures and therapeutic measures. *N Engl J Med* 1969;281:1091-4

25 Jain SC, Misra SM, Misra NP, Tandon PL. Diagnostic value of ascitic fluid examination. *J Assoc Physicians India* 1966;14:59-69

26 Das P, Shukla HS. Clinical diagnosis of abdominal tuberculosis. *Br J Surg* 1976;63:941-6

27 Wolfe JHN, Behn AR, Jackson BT. Tuberculous peritonitis and role of diagnostic laparoscopy. *Lancet* 1979;i:852-3