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AIM AND SCOPE

World Journal of Gastrointestinal Surgery (*World J Gastrointest Surg*, *WJGS*, online ISSN 1948-9366, DOI: 10.4240) is a peer-reviewed open access academic journal that aims to guide clinical practice and improve diagnostic and therapeutic skills of clinicians.

WJGS covers topics concerning micro-invasive surgery; laparoscopy; hepatic, biliary, pancreatic and splenic surgery; surgical nutrition; portal hypertension, as well as associated subjects. The current columns of *WJGS* include editorial, frontier, diagnostic advances, therapeutics advances, field of vision, mini-reviews, review, topic highlight, medical ethics, original articles, case report, clinical case conference (Clinicopathological conference), and autobiography. Priority publication will be given to articles concerning diagnosis and treatment of gastrointestinal surgery diseases. The following aspects are covered: Clinical diagnosis, laboratory diagnosis, differential diagnosis, imaging tests, pathological diagnosis, molecular biological diagnosis, immunological diagnosis, genetic diagnosis, functional diagnostics, and physical diagnosis; and comprehensive therapy, drug therapy, surgical therapy, interventional treatment, minimally invasive therapy, and robot-assisted therapy.

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MINIREVIEWS

Abdominal tuberculosis: Is there a role for surgery?

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Abstract

It is important that surgeons are familiar with the various manifestations of tuberculosis (TB). Although TB has been declining in incidence in the developed world, it remains an important problem in endemic areas of the developing world. The aim of the review was to elucidate the natural history and characteristics of abdominal TB and ascertain the indications for surgery. TB can affect the intestine as well as the peritoneum and the most important aspect of abdominal TB is to bear in mind the diagnosis and obtain histological evidence. Abdominal TB is generally responsive to medical treatment, and early diagnosis and management can prevent unnecessary surgical intervention. Due to the challenges of early diagnosis, patients should be managed in collaboration with a physician familiar with anti-tuberculous therapy. An international expert consensus should determine an algorithm for the diagnosis and multidisciplinary management of abdominal TB.

Key words: Tuberculosis; Peritoneal; Intestinal; Surgery; Anti-tuberculous therapy

Core tip: It is important to bear in mind the non-specific manifestations of abdominal tuberculosis. There is no gold standard for the diagnosis and high clinical suspicion is required. Diagnostic laparoscopy is increasingly useful but joint decision making with physician familiar with anti-tuberculous therapy is important. Surgery is reserved for abdominal complications.

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INTRODUCTION

Abdominal tuberculosis (TB) continues to represent a diagnostic challenge to clinicians[1]. The abdomen is involved in 10%-30% of patients with pulmonary TB and

accounts for between 5% and 10% of TB notifications in the United Kingdom. Greater than 75% of cases occur in immigrants, with most coming from the Indian sub-continent[2,3]. There is a slight male predominance in abdominal TB with a peak incidence in the 4th and 5th decades in the immigrant population, and in the elderly in the United Kingdom[3,4]. In the United States, among native-born white Americans, abdominal TB is primarily a disseminated disease of elderly, debilitated patients with chronic illnesses. Among foreign-born individuals, abdominal TB occurs in the young, immunocompetent patients from endemic areas[5]. The diagnosis is thus difficult and often delayed[6]. Surgeons must be aware of the wide clinical spectrum of abdominal TB and have a high index of suspicion when confronted with patients from an endemic area presenting with unclear abdominal symptoms[2,6]. The aim of the review is to offer an opinion on the role of surgery in abdominal TB and stimulate debate in an area of ongoing interest.

PATHOGENESIS AND PATHOLOGY

The principal forms of abdominal TB are intestinal and peritoneal but a third form - nodal - is also recognized. In practice, the various forms may coexist[6,7]. In the past, many cases of abdominal TB occurred as a direct result of the ingestion of *Mycobacterium bovis* in unpasteurized milk. In most cases today intestinal TB is due to reactivation of primary disease caused by *Mycobacterium tuberculosis*. The reactivation of *Mycobacterium tuberculosis* and the atypical opportunistic *Mycobacterium avium* intracellulare infection in the acquired immune deficiency syndrome have a poor prognosis because of immunosuppression[8-11]. TB bacteria reach the gastrointestinal tract *via* haematogenous spread (from a pulmonary focus acquired during primary infection in childhood), ingestion of infected sputum, or direct spread from infected contiguous lymph nodes and fallopian tubes. Swallowed bacilli pass through the Peyer's patches of the intestinal mucosa and are transported by macrophages through the lymphatics to the mesenteric lymph nodes where they remain dormant. Reactivation of disease in these nodes especially in the immunocompromised including diabetes, renal failure and malignancy may lead to abdominal TB, with the spread of the bacteria to the peritoneum or intestine[4]. Intestinal TB can involve any part of the alimentary tract (from oesophagus to the anus)[2,11]. Gastro-duodenal TB is uncommon (1%) due to the bactericidal properties of gastric acid, scarcity of lymphoid tissue in the mucosa and rapid emptying of gastric contents[12]. The ileocaecal region is the most common site of involvement (75%) because of increased physiological stasis, fluid and electrolyte absorption, minimal digestive activity and abundance of lymphoid tissue (Peyer's patches)[12]. TB of the ileocaecum presents usually with a palpable mass in the right iliac fossa. Perianal disease with abscesses and fistulas can occur, but is uncommon[3,7]. The naked eye appearance of intestinal TB may resemble Crohn's disease, with skip lesions. 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 tubercle (Figure 1). The histology shows numerous granulomas which are not always caseating, and, often, acid-fast bacilli cannot be found if there is low mycobacterial load (Figure 2)[7,13,14]. Tuberculous peritonitis is usually due to reactivation of a tuberculous focus in the peritoneum with concurrent pulmonary, intestinal or genital TB (especially from the fallopian tubes). This is usually seen in debilitated patients and alcoholics[6,7]. Peritoneal TB occur in three forms: Wet type with ascites, dry type with adhesions, and fibrotic type with omental thickening and loculated ascites[14]. Peritoneal TB is characterized by tubercles that appear as white "seedlings" on the parietal and visceral surfaces of the peritoneum. Inflammation and exudation leads to the formation of straw-coloured ascites (Figure 3). When there is associated infiltration and thickening of the omentum, intestinal walls, and formation of caseous masses it is referred to as "plastic" peritonitis[15-17]. *Mycobacterium tuberculosis* can spread to the genital tract *via* the blood or lymphatics. Granulomata develop in the tubes and subsequently the other genital organs. The endometrium is involved

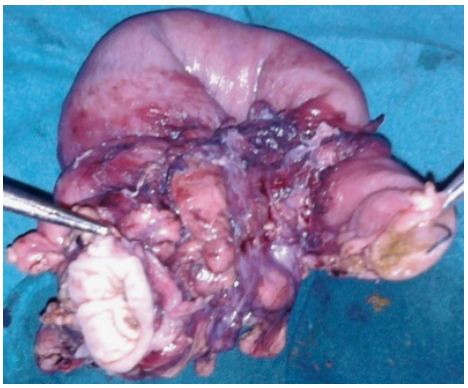

Figure 1 Intestinal tuberculosis (ileocaecal) (with permission from Chumber *et al*[51], 2001).

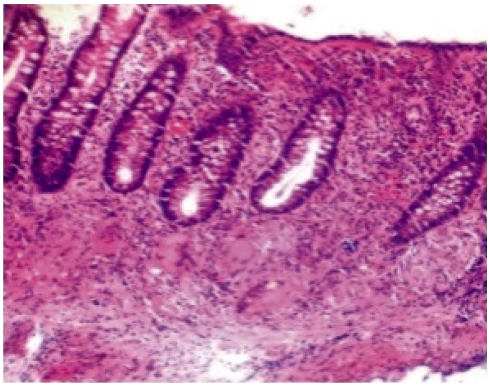

Figure 2 Histopathology (H/E stain): Showing multiple mucosal and submucosal epitheloid cell granulomas with Langhan's giant cells in a case of colonic tuberculosis (with permission from Tandon *et al*[35], 1972).

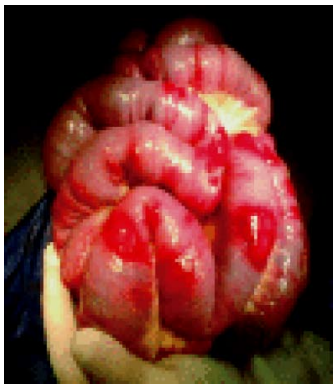

Figure 3 Peritoneal tuberculosis (with permission from Bolognesi *et al*[43], 2013).

in up to 80% of cases and the ovaries in 20%-30%[15]. Fillion *et al*[11] reported in a low incidence country that the main organs involved were the peritoneum (66%), the mesenteric lymph nodes (62%), and the bowel (33%). Atypical presentation of peritoneal TB such as portal vein thrombosis from encasement, with splenomegaly and ascites can delay diagnosis or result in misdiagnosis[14]. Half (50%) of HIV patients with TB have extrapulmonary involvement, compared with only 10%-15% of TB patients who are not infected with HIV[7]. In HIV-infected patients abdominal TB is of a rapidly progressive nature, often fatal though usually treatable.

CLINICAL FEATURES

The clinical symptoms and signs of abdominal TB are non-specific and the diagnosis may be overlooked or mistaken for other disease processes[2]. The clinical picture is different in children to that in adults. About 90% of the features of abdominal TB in children are due to involvement of the peritoneum and lymph nodes and 10% related to intestinal lesions[4-6]. Abdominal pain is common, accompanied by ascites (75%) or an abdominal mass caused by an inflamed mesentery (30%)[2,3]. The most common signs are abdominal tenderness and hepatosplenomegaly. Patients with the "plastic" type of peritoneal TB may have a characteristic "doughy" abdomen but this form is, however, uncommon today[16,17]. Usually the onset of tuberculous peritonitis is insidious with fever, anorexia and weight loss. In a high prevalent area in sub-Saharan Africa, the common presenting symptoms and signs were abdominal pain 76.6%; ascites 59.6%; weight loss 53.2% and fever 29.8%. The average duration of symptoms before presentation was 3 mo and 13% of patients had earlier been treated for pulmonary TB[10].

Intestinal TB presents in a variety of ways. Up to 30% of cases may present as an acute abdomen, either with acute intestinal obstruction or with symptoms and signs suggestive of an acute appendicitis from an obstructing TB lymphadenopathy[11]. Hypertrophic ileocaecal TB is particularly common in the Indian subcontinent as a cause of intestinal obstruction. It must be distinguished from Crohn's disease which is rare in most tropical countries[14]. Tuberculous enteritis may, if the patient recovers, lead to stenotic lesions causing small bowel obstruction[18]. Intestinal perforation and acute bleeds do occur, but are unusual. Classically malabsorption from strictures and sometimes with steatorrhoea, can result from TB of the small intestine, and occasionally when the terminal ileum is involved, patients present with anaemia due to vitamin B_{12} deficiency[14]. Some cases present with disturbance of bowel habit, usually diarrhoea. The remainder of patients with intestinal TB have vaguer symptoms and signs, such as weight loss, malaise and abdominal tenderness. A few patients with only nodal disease present with an abdominal mass consisting of enlarged mesenteric lymph nodes[2,6]. As many as 60% of patients with abdominal TB have evidence of TB elsewhere. Chest X-ray, however, show evidence of concomitant pulmonary lesions in less than 25% of cases[10,11,19,20]. Genitourinary TB may present in a similar manner to pelvic inflammatory disease (PID), with chronic low-grade pelvic pain and ultimately with amenorrhoea and infertility. Abnormal uterine bleeding is a presenting symptom in 10%-40% of patients[15]. Examination is normal in many women but an adnexal mass or fixing of the pelvic organs may be detected[21]. It should be noted that the human immunodeficiency virus (HIV) may alter the manifestations of, and host susceptibility to, other infections[9,15,21]. Other rare clinical presentations include dysphagia, odynophagia and a mid oesophageal ulcer due to oesophageal TB; dyspepsia and gastric outlet obstruction due to gastroduodenal TB; lower abdominal pain and rectal bleeding due to colonic TB; and annular rectal stricture and multiple perianal fistulae due to rectal and anal involvement[2,6].

INVESTIGATIONS

Neither clinical signs, laboratory, radiological and endoscopic methods nor bacteriological and histopathological findings provide a gold standard by themselves in the diagnosis of abdominal TB[10]. The clinical awareness is thus primary[10,22]. Most laboratory tests are unhelpful. The erythrocyte sedimentation rate (ESR) is often moderately raised in 79% of patients, and although there may be a mild normochromic, normocytic anaemia, a leucocytosis is uncommon. Hypoalbuminaemia is not uncommon but liver function tests are usually normal[11,23]. Abdominal TB has a multitude of possible presentations and requires a diagnostic approach adjusted to the individual presentation. This approach should be as little invasive as possible and be based on the best available imaging. Ultrasound scans of abdomen were abnormal in 68%, showing ascites, hepatomegaly and or enlarged nodes. Computed tomography (CT) was the most frequent imaging modality (88%) in the United States. The

findings suggestive of abdominal TB were mesenteric/omental stranding (50%), ascites (37%), and retroperitoneal lymphadenopathy (31%). Seventeen of 18 patients required operative intervention, and one patient underwent CT-guided drainage of a psoas abscess[20]. Mantoux test was positive in 33% and ascitic fluid was diagnostic for TB in 29%. Thus, a positive tuberculin skin test (*e.g.*, Mantoux) may be helpful, though some series have found less than 50% of the cases of proven abdominal TB to be tuberculin positive[23]. Chest X-ray showed abnormal findings in 25% of the patients suggesting past or present pulmonary TB and sputum was positive for acid-fast bacilli (AFB) in 14.3%[10]. A high index of suspicion is, required for the diagnosis of peritoneal TB as the analysis of peritoneal fluid for tuberculous bacilli is often ineffective and may cause mortality due to delayed diagnosis. Examination of the ascitic fluid usually reveals an exudate (protein > 25 g/L) and a raised white blood cell count (WBC) > 0.1×10^9/L consisting principally of lymphocytes. A direct stain for acid-fast bacilli is positive in less than 5% of cases, though up to 40% will be positive if the ascitic fluid is cultured. By centrifuging large volumes of ascitic fluid and culturing the sediment, the diagnostic yield may be increased to up to 80%[23,24]. However, tuberculous peritonitis-associated mortality is high among patients waiting for the results of mycobacterial culture of ascitic fluid samples[24]. Direct stains and culture of stool specimens may sometimes be positive, but the yield is generally low[23]. Barium studies may show some abnormality in about 50% of patients with intestinal TB but are not diagnostic[25]. To confirm the diagnosis, it is important to try to obtain material for culture and histology. As culture may take up to 6 wk, the histological evidence is important. There are a variety of ways of obtaining tissue for histology. Colonoscopy may be useful. Biopsy specimens obtained during colonoscopy of the terminal ileum and ileocaecal valve may show active chronic ileocolitis with ulceration and granuloma formation[25]. Invasive procedures are frequently necessary to obtain samples but also for the treatment of digestive involvement[11]. In light of new evidence, peritoneal biopsy through laparoscopy has emerged as the gold standard for diagnosis and both lymphoma and carcinomatosis can be excluded by this means[26]. Laparoscopy is most reliable as it is minimally-invasive effective modality for diagnosis of peritoneal TB, and can be performed under local anaesthesia. It is rapid, safe, greater than 75% accuracy in diagnosis and spares the patient the discomfort of a laparotomy[11,27]. It allows the biopsy of the typical studded tubercles of the peritoneum and other organs which are sent for culture and histology. However, laparoscopy is costly and is not available in many of the poorer areas of the world. Blind percutaneous peritoneal biopsy with an Abrams or Cope needle biopsy usually in the left lower quadrant just lateral to the rectus muscle is diagnostic in up to 75% of cases of peritoneal TB[3]. The complications of the procedure albeit uncommon include intestinal perforation, bleeding and infection. Thus, for this to be safe, the patient must have clinically detectable ascites. The diagnostic yield can be increased if the peritoneum is exposed by dissection under local anaesthesia[11]. Some patients with abdominal TB without ascites have the diagnosis confirmed indirectly by culture and histology of percutaneously biopsied liver tissue with hepatic TB[3,16]. Diagnostic laparotomy may be resorted to where endoscopic procedures are not available or when they fail to give a definite histopathological diagnosis or for an undiagnosed abdominal mass[16]. While laparotomy will reveal the diagnosis in patients with abdominal TB who present with an acute abdomen, the procedure may be hazardous in sick, emaciated patients with malabsorptive syndrome. It is also not always accurate for the "cold" cases[28] and laparotomy should, thus, essentially be performed only when complications of abdominal TB develop[29]. The suspicion of genitourinary TB in a woman from an endemic area with bilateral tubal calcification from chronic infection seen on abdominal X-ray or radiographic evidence of pulmonary TB should be confirmed if possible, by positive culture of the organisms in endometrial tissue obtained from biopsy or dilatation and curettage[13-15]. Endometrial biopsy does not have 100% sensitivity but the detection rate is greatest towards the end of the menstrual cycle. A Mantoux or Heaf test should be reactive in a woman with active TB unless she is immunosuppressed. The enzyme-linked immunoabsorbent assay (ELISA) using mycobacteria saline-extracted antigen for the serodiagnosis of abdominal TB gives a diagnostic accuracy of 84%[16,23]. Another test for early diagnosis of tuberculous peritonitis is the determination of adenosine deaminase activity (ADA) in the peritoneal fluid[1,13]. New diagnostic procedures, and especially molecular biology-polymerase chain reaction (PCR), may help diagnose unusual clinical presentations of TB[11,23]. As abdominal TB should be considered in all cases with ascites. PCR of ascitic fluid obtained by ultrasound-guided fine needle aspiration is a reliable method for its diagnosis and should at least be attempted before more invasive interventions[13,30].

DIFFERENTIAL DIAGNOSIS

Abdominal TB, with its vague symptoms and signs and non-specific laboratory investigations, can mimic many other diseases (Table 1). The main differential diagnosis to consider with intestinal TB is Crohn's disease. Crohn's disease is uncommon in the immigrant population at risk for TB, and in Caucasians its peak incidence occurs in the 20-40 age group, while that of intestinal TB is in the older age group (50-70 years)[5]. Although perianal disease and enteric fistulas can be due to TB, this is uncommon in comparison with Crohn's disease. Distinguishing between these two entities is a challenge because there is marked overlap in the clinical presentation and the radiographic, laboratory, and endoscopic findings, as well as in the

Table 1 Differential diagnosis of abdominal tuberculosis

Intestinal TB	Peritoneal TB
Crohn's disease	Carcinomatosis
Intestinal lymphoma	Bacterial peritonitis
Carcinoma	Talc peritonitis
Yersinia infections	Chronic liver diseases
Amoeboma	

TB: Tuberculosis.

presence of granulomas on histological examination[31-33]. Misdiagnosis of Crohn's disease in a patient with intestinal TB would result in treatment with steroids and biologic agents, which then has the potential to cause disease progression that leads to increased morbidity and mortality[34]. Misdiagnosis of intestinal TB in a patient with Crohn's disease would lead to prolonged anti-tuberculous therapy and delay the necessary immunosuppression required to induce disease remission[35]. Both diseases have an insidious onset but diarrhoea, rectal bleeding and extraintestinal manifestations are more common in patients with Crohn's disease. Intestinal TB can target extrapulmonary sites in a manner that resembles the classic extraintestinal manifestations of Crohn's disease, such as reactive arthritis, erythema nodosum, and uveitis[36]. Ascites and fever are more commonly seen in patients with intestinal TB. Both diseases involve the ileum and colonic segments of the bowel. Isolated involvement of the terminal ileum is commonly seen in patients with Crohn's disease (terminal ileitis), whereas involvement of the ileocaecal area and a patulous ileocecal valve is seen in patients with intestinal TB (ileo-caecal TB). In patients with Crohn's disease, mucosal injury has a cobblestone appearance with aphthous and longitudinal rake ulcers, whereas in patients with intestinal TB, the ulcers are transverse in orientation[37-39]. Furthermore, the granulomas associated with intestinal TB are more frequent and confluent and larger than those associated with Crohn's disease. As tissue samples are positive for acid-fast bacilli in only 25% to 30% of cases of intestinal TB, the use of molecular techniques such as PCR assays of fresh biopsy specimens, can improve the diagnostic yield[28,40]. Makharia *et al*[38] interestingly developed a scoring system for differentiation of CD and intestinal TB based on clinical endoscopy and histology using the findings of sigmoid colon involvement, blood in stools, weight loss and focally enhanced colitis. Other differential diagnoses are carcinoma, lymphoma, *Yersinia* infections and, in some parts of the world, amoeboma[3]. Peritoneal TB must be differentiated from carcinomatosis, talc peritonitis, bacterial peritonitis, and from ascites due to heart failure or liver disease (Table 1). Although ascites due to cardiac failure is usually easy to distinguish, it is important to realize that there is an increased incidence of abdominal TB in alcoholics, and that liver disease with ascites may coexist with peritoneal TB and the ascites may not have the characteristics of an exudate[5,19]. Some patients may therefore warrant a laparoscopy or diagnostic laparotomy for atypical diagnostic problems especially as diseases such as CD, lymphoma and malignancy can mimic TB in every way[26-28].

MANAGEMENT

Most patients with abdominal TB respond to medical treatment with standard anti-tuberculous chemotherapy and carries good prognosis if promptly diagnosed and treated[8,26]. The drug treatment is identical to pulmonary TB with conventional chemotherapy for at least 6 mo. Rifampicin and isoniazid are given for 6 mo, with two additional drugs-pyrazinamide and streptomycin (given at a dose of 0.75-1.0 g daily depending on body weight, age and renal function) for the first 2 mo[16]. The main cause of failure in medical treatment in the endemic and developing countries is patient defection or poor compliance[10]. Shorter and more effective regimes, based on rifampicin that can be completed in 6 mo have increased patient compliance[16]. For patients in whom the diagnosis is strongly suspected, but for whom the histological proof is unobtainable or inconclusive, it is justifiable to undertake a trial of anti-tuberculous therapy[10,41]. Akinkuolie *et al*[10], reported that 85.1% of patients with clinically diagnosed abdominal TB in a high prevalent area recovered after receiving anti-tuberculous therapy for a period of 9-12 mo. However, all those with HIV infection and not on antiretroviral treatment died from immunosupression[8,10]. A few patients who developed adhesions, obstruction or perforation at some time following chemotherapy required surgery[7]. Intravenous anti-TB therapy in combination with surgery may be needed for severe forms of TB with extensive gastrointestinal involvement[42].

ROLE OF SURGERY

Surgery is essentially reserved for those with acute surgical complications including free perforation, confined perforation with abscess or fistula, massive bleeding, complete obstruction, or obstruction not responding to medical management[6,11,26,27]. Obstruction is the most common complication with multiple and/or long strictures less likely to respond to medical therapy[40,43]. The obstruction may also be exacerbated during anti-tuberculous therapy due to healing by cicatrisation[40]. About 20%-40% of patients with abdominal TB present with an acute abdomen and need surgical management[44]. Chronic patients with subacute obstruction are managed conservatively and surgery is planned after suitable work-up[45]. Being a systemic disease surgical resection should be conservative. Multiple small bowel strictures may be treated by strictureplasty to avoid major resection[46-48]. An alternative is colonoscopic balloon dilatation of readily accessible, short and fibrous tuberculous ileal strictures causing subacute obstructive symptoms. Although the experience is very limited, this technique appears safe and may obviate the need for surgery in this setting[49]. Acute tubercular peritonitis

and mesenteric lymphadenitis need to be managed with caution. If a laparotomy is carried out only a biopsy needs to be performed with peritoneal toilet and the abdomen closed without a drain[50].

The surgery performed in gastrointestinal TB are essentially of three types[14]. The first type is the surgery which is done to bypass the involved segments of bowel such as an enteroenterostomy or an ileotransverse colostomy. As in Crohn's disease, these surgeries are usually complicated by blind loop syndrome, fistula formation and recurrent disease in the remaining segments and hence usually not performed routinely. The second type are segmental resections such as the limited ileo-caecal resection for obstructing ileo-caecal TB with adjuvant anti-tuberculous therapy to eradicate the disease completely[47,51]. However, these surgeries are hindered by the malnourished status of most of the patients which make them poor surgical candidates. Also the lesions can be widely placed and extensive resection may not be possible in all the cases. Postoperative complications include anastomotic leak, faecal fistula, peritonitis, intraabdominal sepsis, persistent obstruction, wound infection and dehiscence[47,48,52]. Re-operation may be required for recurrent obstruction. The third type of surgery is bowel conserving strictureplasty of those stenotic lesions with obstructive symptoms[18,47,48]. Strictureplasty for cases with multiple strictures was introduced as a better technique than multiple resections and enteroanastomoses, as it does not sacrifice any part of the small bowel and avoids the risk of short-bowel syndrome or blind loops[53]. Long strictures with active inflammation or multiple strictures in a segment may require resection unless there is concern about bowel length[18]. With adjuvant anti- tuberculous therapy, microscopic disease at the resection margin should not influence recurrence of disease[14,18-20,54]. The Heineke-Mikulicz pyloroplasty technique is usually used. In a small number of cases with longer strictures where bowel conservation is required, a Finney or a Jaboulay strictureplasty may be used[17-19]. Strictures of recent onset that are not very tight may be left alone, or dilated *via* an enterotomy[53,54]. Tubercular perforations are mostly ileal and proximal to a stricture. If they are close to one another, resection of the segment is performed. If the stricture is not close, the perforation can be closed in layers and the stricture dealt with by strictureplasty or resection, depending on the length of the narrowed segment. Delayed diagnosis and injudicious treatment are responsible for the mortality rate of 4%-12%[50]. The high mortality was partly associated with malnutrition, anaemia and hypoalbuminaemia, the mortality being higher (12%-25%) in the presence of acute complication[47].

Fillion *et al*[11]'s study in a low prevalence country reported that out of 86% presenting with abdominal symptoms, 76% underwent surgery, with 10% in an emergency setting. 81% of patients received six months or more of anti-TB treatment. Seventy-six percent had a positive outcome. Wani *et al*[30] reported a study on surgical emergencies of tubercular abdomen in developing countries. Abdominal pain, vomiting, and constipation were commonest presenting symptoms. About 20% patients had history of pulmonary TB and 16% patients presented with ascites. PCR for blood and ascitic fluid was positive in 72% and 87.5% patients, respectively. As in the low prevalence developed country, the indications and principles of management were the same. About 24% of patients were managed non-operatively and responded to anti-tuberculous therapy. Seventy-six percent needed surgery among which 20% were operated as emergency. Adhesiolysis of gut (47.3%), strictureplasty (10.5%), resection anastomosis (5.2%), right hemicolectomy (5.2%), and ileotransverse anastomosis (7.8%) were performed and peritoneal biopsy and lymph node biopsy in 21% of patients. The tuberculous bowel perforations were usually treated with resection of involved segments with primary anastomosis[17,18]. Generally, emergency surgery in those severely ill patients presenting late carried high mortality from toxaemia, hypoproteinaemia, anaemia and immunosuppression. The mortality rate ranged between 14%-50% in developing countries[10,30], and 6%-37% in developed countries[11,16]. Morbidity included delayed wound healing with occurrence of incisional hernia, recurrent obstruction and faecal fistula[14,47]. Both medically and surgically managed patients responded dramatically to anti-tuberculous therapy with increase in haemoglobin level and fall in ESR[28-30].

CONCLUSION

Abdominal TB is generally responsive to medical treatment, and early diagnosis and management can prevent unnecessary surgical intervention. However, abdominal TB should be considered a surgical problem in the acute and chronic abdomen. Laparoscopy is emerging as the gold standard for diagnosis since diseases such as Crohn's disease, lymphoma and malignancy can mimic TB. Due to the challenges of early diagnosis, patients should be managed in collaboration with a physician familiar with anti-tuberculous therapy. An international expert consensus should recommend an algorithm for the diagnosis and multidisciplinary management of abdominal TB.

REFERENCES

1 **Khan R**, Abid S, Jafri W, Abbas Z, Hameed K, Ahmad Z. Diagnostic dilemma of abdominal tuberculosis in non-HIV patients: an ongoing challenge for physicians. *World J Gastroenterol* 2006; **12**: 6371-6375 [PMID: 17072964 DOI: 10.3748/wjg.v12.i39.6371]

2 **Teh LB**, Ng HS, Ho MS, Ong YY. The varied manifestations of abdominal tuberculosis. *Ann Acad Med Singapore* 1987; **16**: 488-494 [PMID: 3435016]

3 **Wells AD**, Northover JM, Howard ER. Abdominal tuberculosis: still a problem today. *J R Soc Med* 1986; **79**: 149-153 [PMID: 3701750 DOI: 10.1177/014107688607900307]

4 **Mehta JB**, Dutt A, Harvill L, Mathews KM. Epidemiology of extrapulmonary tuberculosis. A comparative analysis with pre-AIDS era. *Chest* 1991; **99**: 1134-1138 [PMID: 2019168 DOI: 10.1378/

chest.99.5.1134]
5 **Tan KK**, Chen K, Sim R. The spectrum of abdominal tuberculosis in a developed country: a single institution's experience over 7 years. *J Gastrointest Surg* 2009; **13**: 142-147 [PMID: 18769984 DOI: 10.1007/s11605-008-0669-6]
6 **Badaoui E**, Berney T, Kaiser L, Mentha G, Morel P. Surgical presentation of abdominal tuberculosis: a protean disease. *Hepato-gastroenterology* 2000; **47**: 751-755 [PMID: 10919025]
7 **Menzies RI**, Alsen H, Fitzgerald JM, Mohapeloa RG. Tuberculous peritonitis in Lesotho. *Tubercle* 1986; **67**: 47-54 [PMID: 3715983 DOI: 10.1016/0041-3879(86)90031-0]
8 **Harries AD**. Tuberculosis and human immunodeficiency virus infection in developing countries. *Lancet* 1990; **335**: 387-390 [PMID: 1968123 DOI: 10.1016/0140-6736(90)90216-R]
9 **Weledji EP**, Nsagha D, Chichom A, Enoworock G. Gastrointestinal surgery and the acquired immune deficiency syndrome. *Ann Med Surg* (Lond) 2015; **4**: 36-40 [PMID: 25685343 DOI: 10.1016/j.amsu.2014.12.001]
10 **Akinkuolie AA**, Adisa AO, Agbakwuru EA, Egharevba PA, Adesunkanmi AR. Abdominal tuberculosis in a Nigerian teaching hospital. *Afr J Med Med Sci* 2008; **37**: 225-229 [PMID: 18982814]
11 **Fillion A**, Ortega-Deballon P, Al-Samman S, Briault A, Brigand C, Deguelte S, Germain A, Hansmann Y, Pelascini E, Rabaud C, Chavanet P, Piroth L. Abdominal tuberculosis in a low prevalence country. *Med Mal Infect* 2016; **46**: 140-145 [PMID: 26995289 DOI: 10.1016/j.medmal.2016.02.003]
12 **Sharma MP**, Bhatia V. Abdominal tuberculosis. *Indian J Med Res* 2004; **120**: 305-315 [PMID: 15520484]
13 **Uzunkoy A**, Harma M, Harma M. Diagnosis of abdominal tuberculosis: experience from 11 cases and review of the literature. *World J Gastroenterol* 2004; **10**: 3647-3649 [PMID: 15534923 DOI: 10.3748/wjg.v10.i24.3647]
14 **Wariyapperuma UM**, Jayasundera CI. Peritoneal tuberculosis presenting with portal vein thrombosis and transudative Ascites - a diagnostic dilemma: case report. *BMC Infect Dis* 2015; **15**: 394 [PMID: 26423615 DOI: 10.1186/s12879-015-1122-6]
15 **Tang LC**, Cho HK, Wong Taam VC. Atypical presentation of female genital tract tuberculosis. *Eur J Obstet Gynecol Reprod Biol* 1984; **17**: 355-363 [PMID: 6479428 DOI: 10.1016/0028-2243(84)90115-1]
16 **Ahmed ME**, Hassan MA. Abdominal tuberculosis. *Ann R Coll Surg Engl* 1994; **76**: 75-79 [PMID: 8154817]
17 **Târcoveanu E**, Filip V, Moldovanu R, Dimofte G, Lupaşcu C, Vlad N, Vasilescu A, Epure O. [Abdominal tuberculosis--a surgical reality]. *Chirurgia* (Bucur) 2007; **102**: 303-308 [PMID: 17687859]
18 **Pujari BD**. Modified surgical procedures in intestinal tuberculosis. *Br J Surg* 1979; **66**: 180-181 [PMID: 427385 DOI: 10.1002/bjs.1800660312]
19 **Chen HL**, Wu MS, Chang WH, Shih SC, Chi H, Bair MJ. Abdominal tuberculosis in southeastern Taiwan: 20 years of experience. *J Formos Med Assoc* 2009; **108**: 195-201 [PMID: 19293034 DOI: 10.1016/S0929-6646(09)60052-8]
20 **Hassan I**, Brilakis ES, Thompson RL, Que FG. Surgical management of abdominal tuberculosis. *J Gastrointest Surg* 2002; **6**: 862-867 [PMID: 12504225 DOI: 10.1016/S1091-255X(02)00063-X]
21 **Campbell S**, Monga A. Infections in gynaecology 2000. In Gynaecology by Ten teachers 17th edition. Stuart Campbell and Ash Monga, Publishers, 2000
22 **Uygur-Bayramicli O**, Dabak G, Dabak R. A clinical dilemma: abdominal tuberculosis. *World J Gastroenterol* 2003; **9**: 1098-1101 [PMID: 12717865 DOI: 10.3748/wjg.v9.i5.1098]
23 **Rana S**, Farooqui MR, Rana S, Anees A, Ahmad Z, Jairajpuri ZS. The role of laboratory investigations in evaluating abdominal tuberculosis. *J Family Community Med* 2015; **22**: 152-157 [PMID: 26392795 DOI: 10.4103/2230-8229.163029]
24 **Chow KM**, Chow VC, Hung LC, Wong SM, Szeto CC. Tuberculous peritonitis-associated mortality is high among patients waiting for the results of mycobacterial cultures of ascitic fluid samples. *Clin Infect Dis* 2002; **35**: 409-413 [PMID: 12145724 DOI: 10.1086/341898]
25 **Muneef MA**, Memish Z, Mahmoud SA, Sadoon SA, Bannatyne R, Khan Y. Tuberculosis in the belly: a review of forty-six cases involving the gastrointestinal tract and peritoneum. *Scand J Gastroenterol* 2001; **36**: 528-532 [PMID: 11346208 DOI: 10.1080/003655201750153412]
26 **Rai S**, Thomas WM. Diagnosis of abdominal tuberculosis: the importance of laparoscopy. *J R Soc Med* 2003; **96**: 586-588 [PMID: 14645607 DOI: 10.1258/jrsm.96.12.586]
27 **Târcoveanu E**, Dimofte G, Bradea C, Lupaşcu C, Moldovanu R, Vasilescu A. Peritoneal tuberculosis in laparoscopic era. *Acta Chir Belg* 2009; **109**: 65-70 [PMID: 19341199 DOI: 10.1080/00015458.2009.11680374]
28 **Akgun Y**. Intestinal and peritoneal tuberculosis: changing trends over 10 years and a review of 80 patients. *Can J Surg* 2005; **48**: 131-136 [PMID: 15887793]
29 **Yajnik V**, McDermott S, Khalili H, Everett JM. CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL. Case 7-2016. An 80-Year-Old Man with Weight Loss, Abdominal Pain, Diarrhea, and an Ileocecal Mass. *N Engl J Med* 2016; **374**: 970-979 [PMID: 26962732 DOI: 10.1056/NEJMcpc1509455]
30 **Wani MU**, Parvez M, Kumar SH, Naikoo GM, Jan M, Wani HA. Study of Surgical Emergencies of Tubercular Abdomen in Developing Countries. *Indian J Surg* 2015; **77**: 182-185 [PMID: 26246698 DOI: 10.1007/s12262-012-0755-6]
31 **Tandon HD**, Prakash A. Pathology of intestinal tuberculosis and its distinction from Crohn's disease. *Gut* 1972; **13**: 260-269 [PMID: 5033841 DOI: 10.1136/gut.13.4.260]
32 **Almadi MA**, Ghosh S, Aljebreen AM. Differentiating intestinal tuberculosis from Crohn's disease: a diagnostic challenge. *Am J Gastroenterol* 2009; **104**: 1003-1012 [PMID: 19240705 DOI: 10.1038/ajg.2008.162]
33 **Huang X**, Liao WD, Yu C, Tu Y, Pan XL, Chen YX, Lv NH, Zhu X. Differences in clinical features of Crohn's disease and intestinal tuberculosis. *World J Gastroenterol* 2015; **21**: 3650-3656 [PMID: 25834333 DOI: 10.3748/wjg.v21.i12.3650]
34 **Wagner TE**, Huseby ES, Huseby JS. Exacerbation of Mycobacterium tuberculosis enteritis masquerading as Crohn's disease after treatment with a tumor necrosis factor-alpha inhibitor. *Am J Med* 2002; **112**: 67-69 [PMID: 11812409 DOI: 10.1016/S0002-9343(01)01035-X]
35 **Tandon R**, Ahuja V. Differentiating intestinal tuberculosis from Crohn's disease. In: Jewell DP, editor. Inflammatory bowel disease. Tokyo: Macmillan Medical Communications, 2014: 41-60
36 **Singh B**, Kedia S, Konijeti G, Mouli VP, Dhingra R, Kurrey L, Srivastava S, Pradhan R, Makharia G, Ahuja V. Extraintestinal manifestations of inflammatory bowel disease and intestinal tuberculosis: Frequency and relation with disease phenotype. *Indian J Gastroenterol* 2015; **34**: 43-50 [PMID: 25663290 DOI: 10.1007/s12664-015-0538-7]
37 **Lee YJ**, Yang SK, Byeon JS, Myung SJ, Chang HS, Hong SS, Kim KJ, Lee GH, Jung HY, Hong WS, Kim JH, Min YI, Chang SJ, Yu CS. Analysis of colonoscopic findings in the differential diagnosis between intestinal tuberculosis and Crohn's disease. *Endoscopy* 2006; **38**: 592-597 [PMID: 16673312 DOI: 10.1055/s-2006-924996]
38 **Makharia GK**, Srivastava S, Das P, Goswami P, Singh U, Tripathi M, Deo V, Aggarwal A, Tiwari RP, Sreenivas V, Gupta SD. Clinical, endoscopic, and histological differentiations between Crohn's disease and intestinal tuberculosis. *Am J Gastroenterol* 2010; **105**: 642-651 [PMID: 20087333 DOI: 10.1038/ajg.2009.585]
39 **Zhao XS**, Wang ZT, Wu ZY, Yin QH, Zhong J, Miao F, Yan FH. Differentiation of Crohn's disease from intestinal tuberculosis by clinical and CT enterographic models. *Inflamm Bowel Dis* 2014; **20**: 916-925 [PMID: 24694791 DOI: 10.1097/MIB.0000000000000025]
40 **Rathi P**, Gambhire P. Abdominal Tuberculosis. *J Assoc Physicians India* 2016; **64**: 38-47 [PMID: 27730779]
41 **Afzal S**, Qayum I, Ahmad I, Kundi S. Clinical diagnostic criteria for suspected ileocaecal tuberculosis. *J Ayub Med Coll Abbottabad* 2006; **18**: 42-46 [PMID: 17591009]
42 **Goldani LZ**, Spessatto CO, Nunes DL, Oliveira JG, Takamatu E, Cerski CT, Goldani HA. Management of Severe Gastrointestinal Tuberculosis with Injectable Antituberculous Drugs. *Trop Med Health* 2015; **43**: 191-194 [PMID: 26543395 DOI: 10.2149/tmh.2015-09]
43 **Bolognesi M**, Bolognesi D. Complicated and delayed diagnosis of tuberculous peritonitis. *Am J Case Rep* 2013; **14**: 109-112 [PMID: 23826447 DOI: 10.12659/AJCR.883886]

44 **Saxena P**, Saxena S. The role of laparoscopy in diagnosis of abdominal tuberculosis. *Int Surg J* 2016; **3**: 1557-1563 [DOI: 10.18203/2349-2902.isj20162747]

45 **Ha HK**, Ko GY, Yu ES, Yoon K, Hong WS, Kim HR, Jung HY, Yang SK, Jee KN, Min YI, Auh YH. Intestinal tuberculosis with abdominal complications: radiologic and pathologic features. *Abdom Imaging* 1999; **24**: 32-38 [PMID: 9933670 DOI: 10.1007/s002619900436]

46 **Dandapat MC**, Mohan Rao V. Management of abdominal tuberculosis. *Indian J Tuberc* 1985; **32**: 126-129

47 **Bhansali SK**. Abdominal tuberculosis. Experiences with 300 cases. *Am J Gastroenterol* 1977; **67**: 324-337 [PMID: 879148]

48 **Pattanayak S**, Behuna S. Is abdominal tuberculosis a surgical problem? *Ann R Coll Surg Engl* 2015; **97**: 414-419 [DOI: 10.1308/rcsann.2015.0010]

49 **Bhasin DK**, Sharma BC, Dhavan S, Sethi A, Sinha SK, Singh K. Endoscopic balloon dilation of ileal stricture due to tuberculosis. *Endoscopy* 1998; **30**: S44 [PMID: 9615897 DOI: 10.1055s-2007-1001274]

50 **Kapoor VK**. Abdominal tuberculosis. *Postgrad Med J* 1998; **74**: 459-467 [PMID: 9926119 DOI: 10.1136/pgmj.74.874.459]

51 **Chumber S**, Samaiya A, Subramaniam R, Dehran M, Vashisht S, Karak AK, Srivastava A. Laparoscopy assisted hemi-colectomy for ileo-caecal tuberculosis. *Trop Gastroenterol* 2001; **22**: 107-112 [PMID: 11552482]

52 **Palmer KR**, Patil DH, Basran GS, Riordan JF, Silk DB. Abdominal tuberculosis in urban Britain--a common disease. *Gut* 1985; **26**: 1296-1305 [PMID: 4085907]

53 **Katariya RN**, Sood S, Rao PG, Rao PL. Stricture-plasty for tubercular strictures of the gastro-intestinal tract. *Br J Surg* 1977; **64**: 496-498 [PMID: 922310 DOI: 10.1002/bjs.1800640713]

54 **Fazio VW**, Marchetti F, Church M, Goldblum JR, Lavery C, Hull TL, Milsom JW, Strong SA, Oakley JR, Secic M. Effect of resection margins on the recurrence of Crohn's disease in the small bowel. A randomized controlled trial. *Ann Surg* 1996; **224**: 563-571; discussion 571-573 [PMID: 8857860 DOI: 10.1097/00000658-199610000-00014]

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