

# ABDOMINAL TUBERCULOSIS : AN ANALYSIS OF 50 CASES

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## **CERTIFICATE**

This is to certify that the dissertation titled “**ABDOMINAL TUBERCULOSIS : AN ANALYSIS OF 50 CASES**” is the bonafide original work of DR. C.KAVERI in partial fulfillment of the requirements for M.S. Branch – I (General Surgery) Examination of the Tamilnadu DR. M.G.R Medical University to be held in September 2006. The Period of study was from August 2003 to March 2006.

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## **DECLARATION**

I, **DR. C.KAVERI** , solemnly declare that dissertation titled **“ABDOMINAL TUBERCULOSIS : AN ANALYSIS OF 50 CASES”** is a bonafide work done by me at Govt. Stanley Medical College and Hospital during August 2003 to March 2006 under guidance and supervision of my unit chief **Prof. . K. NITHIYANATHAN, M.S**, Addl. Professor of Surgery.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.S. Degree (Branch – I ) in General Surgery.**

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## INTRODUCTION

The term abdominal tuberculosis refers to tuberculous infection of the gastrointestinal tract, mesenteric lymph nodes, peritoneum and omentum, and of solid organs related to GIT such as liver, spleen and pancreas<sup>[1]</sup>.

Tuberculosis is still common disease in the developing world<sup>[2]</sup>. However, the diagnosis of abdominal tuberculosis is obscure. Joseph Walsh in 1909, remarked that “It is impossible to diagnose Abdominal tuberculosis with any degree of certainty.” Unfortunately this remains even today in situations where it is relatively common. The sign and symptoms are often vague and laboratory investigation and radiological findings are non-specific.

Many cases go unnoticed until a surgically removed specimen is examined histopathologically. A majority of stenotic lesions of the bowel are of tuberculous in nature and has been found on the operating table has one of the common findings in cases of acute intestinal obstruction.

With tuberculosis being a common disease in our country accounting for both extrapulmonary and pulmonary infections, we studied the various presentations of Abdominal tuberculosis and analyzed the pathology in the present scenario.

The study was conducted in the department of the General Surgery, Govt.Stanley Medical College and Hospital, Chennai between August 2003 to March 2006.

## AIMS OF STUDY

- ❖ To analyze the various clinical presentations of abdominal tuberculosis.
- ❖ To analyze the frequency of affection of GIT in abdominal tuberculosis.
- ❖ To analyze the accuracy of diagnosis of abdominal tuberculosis clinically.
- ❖ To review the role of common investigations in the diagnosis of abdominal tuberculosis.

## REVIEW OF LITERATURE

### *HISTORY*

Tuberculosis was first recognized in the fourth century BC. Hippocrates, who called tuberculosis “Phthisis” (from Greek, meaning to decay), appreciated the severity of tuberculous enteritis as a complication of pulmonary tuberculosis.

In the nineteenth century and the early part of twentieth century, tuberculosis was widely prevalent in most parts of the world, and was the major cause of intestinal stricture and bowel obstruction.

Rokit Ansky : In 1845 described, tuberculosis of Gut, mesenteric nodal involvement and perforation of gut. Francis Sylvins of Leyden(1614-1672) first employed the term “tubercle” to describe the pathology. Virchow (1821-1902) described development of caseation in tuberculous tissue.

The modern era in tuberculosis began in 1882 with the identification of the causative organism, Mycobacterium tuberculosis, by Robert Koch; this facilitated not only a definitive diagnosis of illness but also the development of the effective anti-mycobacterial agents.

The use of X-rays in Enteric tuberculosis was first described by Edward Sterilin in 1911. Brown and Samson reviewed the result of Barium meal methods for early diagnosis in 1932. Crohn & Yarns in 1940 viewed the problems of intestinal tuberculosis, and found existence of hyperplastic tuberculosis.

The Nobel prize winning discovery of drug streptomycin in 1914 was followed by PAS by Lehman (1946), inh by Grunberg et al(1951), and Pyrazinamide by Mctune (1955). It was realized that organisms develop resistance

to these antibiotics especially streptomycin, and quest to new antituberculosis drugs started and second line of drugs were discovered.

Today multidrug therapy have revolutionized the treatment of tuberculosis.

### ***EPIDEMIOLOGY:***

Abdominal tuberculosis was recognized as the most common complication of active pulmonary tuberculosis in first half of twentieth century. Enteric involvement was found in 6-90% of patients with pulmonary tuberculosis in necropsy and radiological series.

Blumberg found roentgenographic evidence of tuberculosis in 5-8% of early, 14-18% of moderately advanced, 70-80% of far advanced cases of pulmonary tuberculosis. Mitchell and Bristol indicated the association between pulmonary and enteric tuberculosis to be 1% with minimal, 4.5% with moderately advanced, and 24.7% with far advanced pulmonary tuberculosis.

Now it is the 6<sup>th</sup> most frequent form of extrapulmonary tuberculosis and accounts for 10% to 15% extrapulmonary tuberculosis in non HIV patients, and up to 50% in HIV infected patients.

Tuberculosis (TB) causes some 3 million deaths per year worldwide and is increasing in incidence in developed, and developing countries. Abdominal tuberculosis constitutes upto 12% of extrapulmonary TB and 1-3% of total. [Jadvar.et.al]. Incidence of primary abdominal tuberculosis in unselected autopsy series has been reported to vary from 0.02% to 51% (Trivedi and Gupta 1941, Garlick et al, Pimparkar and Donde 1975).

By the 1970's , it was considered a rare disease in the west. However, starting from mid-1980's a resurgence of tuberculosis occurred, due to a large extent to the epidemic of AIDS. Worldwide number of new patients with tu

berculosis was expected to increase from 8 million annually to over ten million by year 2000.

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 co-infected with HIV and TB. In a study from Mumbai, HIV seroprevalence was found in 16.6% in patients with abdominal tuberculosis as compared to 1.4% in voluntary blood donors. Two large series from UK have reported the prevalence in Asian immigrants as 16 and 36 per 100,000 populations( Singh MM .et.al).

The current frequency of occurrence of enteric tuberculosis has not been assessed. The recent data on TB estimates for India In 2002 are

*Table 1: TB estimates for India (2002)*

Population	1049 million
Global rank	1
Incidence ( all cases/ 100,000 population)	168
Incidence( new smear positive)	75
Prevalence ( smear positive)	156
TB mortality	37

## **PATHOGENSIS**

*Mycobacterium tuberculosis* is the causative organism for abdominal tuberculosis There are three types of mycobacterium, Human Bovine and Avian. Human strains transmitted to susceptible host usually by inhalation of infected droplets coughed by patients with open lesions.

**Microbiology:** *Mycobacterium tuberculosis* is gram-positive, aerobic, non-motile, non-spore bearing organism that is identified by Ziehl-Neelson acid fast differential staining method. The classical method of culturing the organism is in

the solid Lowenstein- Jensen medium, which requires an incubation period of 4-6 weeks. Liquid culture medium, containing growth base, casein and bovine serum, may provide faster result and is more sensitive than solid medium.

***Route of Infection:***

The mycobacterium tuberculosis spreads to abdomen by several routes .

1. spread by means of the ingestion of infected sputum, in patients with active pulmonary TB and especially in patients with pulmonary cavitation and positive sputum smears;
2. spread through a hematogenous route from tuberculous focus in the lung to submucosal lymph nodes; and
3. local spread from surrounding organs involved by primary tuberculous infection (eg, renal TB causing fistulas into the duodenum or mediastinal TB lymphadenopathy involving the esophagus).

**Pathologic findings**

***Histopathogenesis:***

In tuberculous most active inflammation takes place in submucosa , resulting in marked thickening as a result of edema, cellular infiltration, lymphatic hyperplasia, formation of tubercles and fibrosis.. The walls of serosa may be penetrated by lymph channels or direct continuity. The tubercles may visualized, on surface of intestines. They may involve mesenteric nodes also.

Mucosal ulceration results from necrosis of Peyer patches, lymph follicles, and vascular thrombosis. At this stage of the disease, the changes are reversible and healing without scarring is possible. As the disease progress, the ulceration

becomes confluent and extensive fibrosis leads to bowel wall thickening, fibrosis, and pseudotumoral mass lesions. Strictures and fistulae formation may occur.

The serosal surface may show nodular masses of tubercles. The mucosa is inflamed with hyperemia and edema similar to that observed in Crohn disease. In some cases, aphthous ulcers may be seen in the colon. Caseation may not always be seen in the granuloma, especially in the mucosa, but they are almost always seen in the regional lymph nodes.

## **PATHOLOGY**

### **GASTRO INTESTINAL TUBERCULOSIS**

Tuberculosis may involve any region of gut. Tuberculous enteritis may be classified on basis of morphologic appearance.

(1) The ulcerative form of TB is seen in approximately 60% of patients. Multiple superficial ulcers largely confined to the epithelial surface. This is considered a highly active form of the disease with the long axis of the ulcers perpendicular to the long axis of the bowel.

(2) The hypertrophic form is seen in approximately 10% of patients and consists of thickening of bowel wall with scarring; fibrosis; and a rigid, mass like appearance that mimics that of a carcinoma.

(3) The ulcero-hypertrophic form is a subtype seen in 30% of patients. These patients have a combination of features of the ulcerative and hypertrophic forms.

**(1). Ulcerative – Usually Small Intestine and Malnourished individuals**

This is the result of deprivation of Blood supply by endarteritis (Howell JS, Gut 164). There is accumulation of collagenous tissue during the process of ulcer healing and subsequent circumferential structure of bowel lumen (NAPKIN RING ) obstruction other complication are perforation and fistulas etc. The ulcers are characteristically transverse and circumferential are reported do not penetrated muscularis propria. Anand. et al(1962) reported 15 cases of perforation and Dutta Gupta(1968) reported fecal fistula formation.

**(2). Hypertrophic Usually occurs in well nourished individuals**

This usually occurs in ileocecal area and the colon. It features florid inflammatory fibroelastic – reaction in the submucosa, and subserosa. The mesentery, lymph nodes and bowel are matted together, by adhesions to form a mass. This form usually presents as mass in right iliac fossa(or) right lumbar region.

**(3). Ulcerohypertrophic**

This is the combination of above a forms. This features as alcers, nodularity, pseudopolyps, hyperplasia and stenosis. A relationship exists between the number of tubercle Bacilli and the virulence and the resulting lesion, a low density of Bacilli with decreased virulence, forms hypertrophic lesion, whereas a large number of Bacilli with enhanced virulence leads to formation of ulcers.

## **Tuberculosis of peritoneum and nodes:**

Peritoneal tuberculosis occurs in 3 forms:

- Wet type with ascitis;
- Encysted (loculated) type with a localized abdominal swelling; and
- 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.

## **Tuberculosis of Solid organs**

Hepatic tuberculosis has become exceedingly rare these days. The diagnosis is usually made accidentally during exploratory laparotomy or at autopsy in immunocompromised patients. The lesions typically are granulomas, with or without central caseating necrosis, calcified masses, and biliary strictures.

Splenic tuberculosis is also rare and may present as a splenic abscess or with hypersplenism. The presence of multiple hypoechoic lesions on ultrasonography of the spleen in a HIV-positive patient is highly suggestive of disseminated tuberculosis... The diagnosis is usually made following surgical resection of the diseased spleen.

Microscopic changes: Tuberculous granulomas are of variable size and characteristically tends to be confluent in contrast to those seen in Crohn's disease granulomas usually found just beneath the ulcer bed mainly submucosal layers. Ulcers are relatively superficial and usually don't penetrate beyond muscularis. .Ulcer usually transversely oriented. Cicatricial healing of these circumferential girdle ulcer result in stricture. Occlusive endarteritis also produce ischemia and contribute to stricture formation.

## CLINICAL FEATURES

The protean of clinical manifestations and varied complications of abdominal tuberculosis continues to be challenge to diagnostic acumen and therapeutic skill of all physicians. It can mimic many conditions like Inflammatory Bowl Disease, malignancy and other infectious diseases. Untreated or delayed treatment can resulting life long morbid complications.

*Abdominal tuberculosis should be considered as possibility in any patients present with lump, ascites, intestinal obstruction or peritonitis.*

### **Age and Sex**

The average age of presentation in Indian patients is 26 years. Most of the patients belongs to age group 13 – 40 years. More common in woman.

Socio Economic Status : Most of the patients are from low socio economic status and living in over crowded area.

### GENERAL SYMPTOMS

Vague symptoms like generalized weakness, lassitude, loss of appetite, loss of weight, low grade fever with sweating, cough with hemoptysis and menstrual disorders in female etc.

*Table No. 2 : Symptoms reported in cases of Abdominal TB*

Symptoms	Frequency
Weight loss	63( 35-87)
Fever	61 (29-100)
Anorexia	48 ( 10-100)
Amennorrhoea	23 (12-36)
Pulmonary	19 (4-51)

### ABDOMINAL SYMPTOMS

Depends upon the site and nature of the lesion.

The frequency of various clinical features are as tabulated.

Table No. 3 : Abdominal features in Abdominal TB

Symptoms	Frequency
Abdominal pain	86(77-94)
Vomiting	46 ( 33-74)
Abdominal distension	37 (28-45)
Ascites	37 (19-60)
Abdominal mass	33 (17-45)
Diarrhea	22 (11-48)
Constipation	24(12-2)
Haematochezia	4 (2-13)

(1). **Abdominal pain**

Most common presentation. Pain may be dull and vague but when colicky it suggests intestinal obstruction. Pain is often exacerbated by eating and relieved by vomiting or passing flatus.

(2). **Vomiting**

This symptom suggest intestinal obstruction which may be acute chronic or acute on chronic.

(3). **Abdominal distension** may be localized or generalized seen in ascites, persistent subacute obstruction , or perforation with peritonitis.

(4). **Ascites** usually seen in peritoneal disease.

(5). **Abdominal mass** usually seen in ileocecal tuberculosis or peritoneal tuberculosis with rolled up omentum.

(6). **Borborygmi** suggest luminal compromise.

(7). **Diarrhoea** usually occurs in small bowel lesion especially ulcerative type.

(8). **Alternate constipation and diarrhoea** occurs in subacute obstruction .

(9). **Hematochezia** occurs in colonic or rectal tuberculosis.

(10) Rarely discharge from umbilicus or skin, **pneumaturia, faecaluria, and hematuria** due to fistula.

## **Physical signs**

The general survey of the patient reveals an malnourished body with signs of pallor, and lymphadenopathy .

### **PER ABDOMEN**

Abdomen may be abnormal or normal. Visible intestinal peristalsis when found suggest some obstruction of the bowel lumen. Distension, tenderness, mass especially in right iliac fossa or epigastric region [rolled up omentum] , or right lumbar region may be palpable.

A doughy feel of abdomen has become less common now a days. 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. Esophageal TB is the least common site of TB in the GI tract. Till 1997 only 58 cases had been reported in the English literature. Esophageal TB is rare, usually occurring because of spread from TB in the thorax either from mediastinal nodes, lungs, or spine.

The patient usually presents with low grade fever, dysphagia, odynophagia and an ulcer, most commonly midoesophageal. The disease usually mimics oesophageal carcinoma and extra-oesophageal focus of tuberculosis may not be evident.

Dysphagia and retrosternal pain indicate esophageal involvement with ulcerations just above the tracheal bifurcation. A rare granular form of TB occurs in miliary spread of primary TB.

### **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-secretory therapy. It may also simulate gastric carcinoma.

Chowdhary *et al* 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 . 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. Other reported complications by various authors are perforation, fistulae (pyeloduodenal, duodenocutaneous, blind) , excavating ulcers extending into pancreas and obstructive jaundice by compression of the common bile duct.

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 fast 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**

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 bacillus.

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 iliac group of nodes are enlarged .

The most common complication of small bowel or ileocaecal tuberculosis is obstruction due to narrowing of the lumen by hyperplastic caecal tuberculosis, by strictures of the small intestine, which are commonly multiple, or by adhesions.

Adjacent lymph nodal involvement can lead to traction, narrowing and fixity of bowel loops.

In India, around 3 to 20 per cent of all cases of bowel obstruction are due to tuberculosis. In a large series of 348 cases of intestinal obstruction, Bhansali and Sethna found tuberculosis to be responsible for 54 (15.5%) cases; 33 cases were small bowel and 21 large bowel obstruction.

Tandon *et al* 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. 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. Tubercular perforations are usually single and proximal to a stricture. Acute tubercular peritonitis without intestinal perforation is usually an acute presentation of peritoneal disease but may be due to ruptured caseating lymph nodes. Malabsorption is a common complication. Next to tropical sprue, it is the most important cause of malabsorption syndrome in India.

In a patient with malabsorption, a history of abdominal pain suggests the diagnosis of tuberculosis. Pimparkar and Donde studied 40 patients with malabsorption and divided them 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 mal-absorption and found them to be abnormal in, 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* also reported biochemical

evidence of mal-absorption in 75 per cent of patients with intestinal obstruction and in 40 per cent of those without it. The cause of mal-absorption 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. Multifocal involvement is seen in one-third (28 to 44%) of patients with colonic tuberculosis. The median duration of symptoms at presentation is less than 1 yr. Pain is the predominant symptom in 78-90 per cent of patients and hematochezia occurs in less than one third. The bleeding is frequently minor and massive bleeding is less common.

Singh *et al* reported rectal bleeding in 31 per cent of patients with colonic tuberculosis, and it was massive in 13 per cent. Bhargava *et al* reported bleeding in 70 percent cases. Overall, tuberculosis accounts for about 4 per cent of patients with lower gastrointestinal bleeding. 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%) 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. 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. Anal tuberculosis is less uncommon and has a distinct clinical presentation. Tubercular fistulae are usually multiple. Dandapat *et al* 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* 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 . Anal tuberculosis is also seen in paediatric patients .

### **Peritoneal tuberculosis:**

Tuberculous peritonitis usually presents with ascites. Less frequently, fluid collections is mild, but fibrotic component is more prominent, resulting in thickening of peritoneum with adhesions, with fluid loculations, and the classic doughy feel of abdomen. The patient with peritoneal tuberculosis , especially women, often have coexisting tuberculosis of pelvic organs. Since the genital tract is frequently portal of entry of the tubercle bacillus.

## **DIFFERENTIAL DIAGNOSIS**

Disease entities that by their symptoms, signs and radiographic morphologic and histologic features must be differentiated from tuberculosis entities, relative to geographical location include as follows:

### **❖ Small Bowel**

- Crohns disease
- Lymphoma
- Vascular in-sufficiency
- Fungal infections
- Non specific mesenteric lymphadenitis

### **❖ Ileocecal Junction and Colon**

- Acute appendicitis
- Appendicular lump
- Crohn's disease
- Ulcerative colitis
- Malignancy
- Lymphoma
- Ameboma
- Actinomycosis

### **❖ Rectosigmoid**

- Crohn's disease
- Ulcerative colitis
- Ischemic proctocolitis
- Ameboma
- Diverticulitis
- Lympho granuloma venereum

- Endometriosis

## ***Diagnosis and investigations***

Paustian in 1964<sup>4</sup> 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.

## **LABORATORY AIDS**

### **(1). Haematological Test**

- Differential leukocyte count shows lymphocytosis.
- Blood picture may show normocyte normochromic anemia (or) Iron Deficiency anemia.
- Raised ESR.
- Hypoproteinemia.
- Low serum iron levels etc.

### **(2). Bacteriological Test**

Examination of Acid fast bacilli

- (i). From spectrum Gastric aspirate peritoneal fluid tapping, pleural fluid aspiration, urine stools.
- (ii) Culture methods : Culture of acid fast Bacilli from sputum, gastric contents, pleural fluid Aspirate, urine, excretions from fistulas and stools.

### ***Radiological studies***

Play a very important role in the diagnosis of abdominal tuberculosis.

#### ***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* 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%) . In Prakash's series of 300 patients, none had active pulmonary tuberculosis but 39 per cent had evidence of healed tuberculosis. Tandon *et al* 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 & Contrast 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.

*Barium contrast studies* are often rewarding in patients suspected to have intestinal tuberculosis. Sharp and Goldman reported barium meal follow through examination as the best diagnostic test, demonstrating bowel lesions highly

suggestive of tuberculosis such as multiple strictures and distended caecum or terminal ileum in 84% of cases.

*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 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 inflamed 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 emptying into a shortened, rigid or obliterated caecum.

- (vii) “String sign” – persistent 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.

Ultrasonography being a widely available investigation, is now a ‘low threshold’ diagnostic procedure for all patients suspected to have abdominal tuberculosis. It can accurately demonstrate small quantities of ascitic fluid and is an effective method for detection of peritoneal disease.

The following features may be seen, usually in combination :-

- (i) Intra-abdominal fluid which may be free or loculated ; and clear or complex (with debris 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 inflamed 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.

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. 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.

Tariq Sinan .et.al in his study of CT findings in 27 patients had reported that CT reliably demonstrates the entire range of findings which need interpretation in the light of clinical and laboratory data.

*Table no. 4: Summary of CT findings ( Tariq Sinan .et.al)*

Peritonitis (Mesentry, omental and peritoneal disease):	38 (77.5%)
Lymph nodes:	23 (46.9%)
G.I.T:	19 (38.7%)
Solid organ involvement:	10 (20.4%)

*Table No.5 : Distribution of lymph nodes*

Diffuse (Peripancreatic ± Mesentric ± paraaortic)	11 (48%)
Mesentric:	6 (26%)
Peripancreatic/Portal:	3 (13%)
Para aortic:	3 (13%)

*Table No: 6 GI Tuberculosis*

Ileocecal and distal ileum:	10 (50%)
Small bowel:	7 (36.8%)
Large bowel:	2(10.5%)
Stomach (ulcer):	1 (5.2%)
* Perforation:	2
* Fistulae:	1

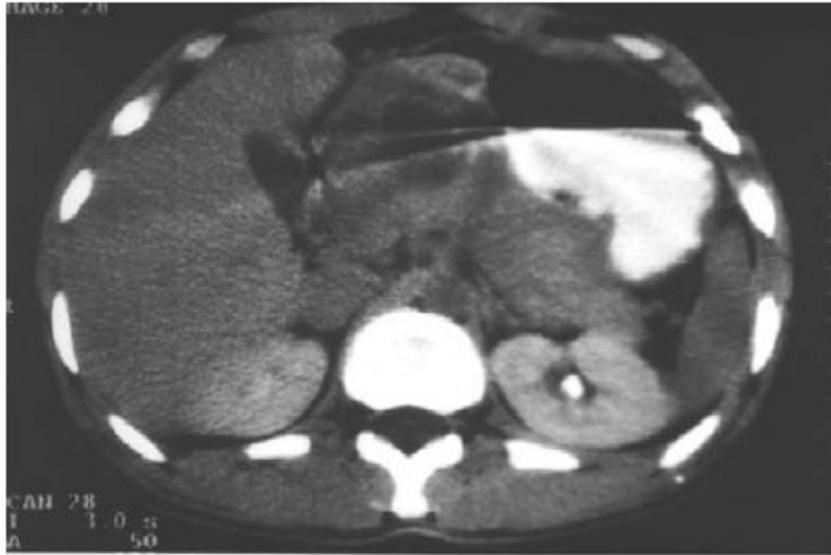


Fig.No.1: CT of the upper abdomen showing large necrotic inflammatory mass in the lesser sac and involving the pancreas. This was due to perforating gastric TB ulcer.

### **Colonoscopy:**

Colonoscopy is an excellent tool to diagnose colonic and terminal ileal involvement 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 pathognomonic. 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. 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.

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 . 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* 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* reported that an optical density(OD) of 0.81 on ELISA and fluorescent 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* 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 .

The amplification of specific DNA sequences by Polymerase chain reaction is a novel tool for the detection of mycobacterial DNA sequences in clinical specimens such as body fluids and tissues. PCR has several advantages over culture, including confirmation of the presence of mycobacterium tuberculosis within 1 to 3 days as compared to six weeks with conventional culture techniques. DNA amplification can be used for tissue specimens which are formalin- fixed and paraffin embedded. Many a times tissue biopsy is send directly for histopathological unsuspecting tuberculosis until the report of microscopis examination comes to be in favour of TB. In those cases PCR amplification can be used to detect mycobacterium tuberculosis in tissue samples even though tissues have been preserved in formalin or pother substances that preclude the possibility of culture.

The application of PCR to the diagnosis of extra-pulmonary TB has the potential to resolve one of the foremost challenges faced by a clinican and a diagnostic laboratory.

PCR, in principle, is a highly sensitive technique that detects DNA from a single to a few microorganisms with overall sensitivity, specificity, and positive predictive value of 97.87%, 100%, and 100% respectively. Acid fast staining alone has positivity of only 15%, and histopathology alone has a positivity of 82.97%.

PCR has a potentially important role in improving the diagnostic accuracy in clinical specimens from extra-pulmonary TB seen in surgical practice.

### **Ascitic fluid examination**

The ascitic fluid in tuberculosis is straw coloured with protein >3g/dl, and total cell count of 150-4000/  $\mu$ l, consisting predominantly of lymphocytes (>70%). The ascites to blood glucose ratio is less than 0.96 and serum ascitic 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* 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*. 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. In the study by Bhargava *et al*, 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. In coinfection with HIV the ADA values can be normal or low. Falsely high values can occur in malignant ascitis. High interferon- $\gamma$  levels in tubercular ascitis have been reported to be useful diagnostically. Combining both ADA and interferon estimations may further increase sensitivity and specificity.

### **Laparoscopic findings**

Bhargava *et al* 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 inoculation (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.

(iv) In peritoneal tuberculosis, *laparoscopic* appearances of thickened peritoneum alongwith whitish to yellowish miliary tubercles studded over the

peritoneum and other viscera have been found to be more helpful in diagnosis of tuberculosis than either histological or bacteriological examination.

### ***COMPLICATIONS***

The complication of Abdominal tuberculosis are :

- ★ Obstruction
- ★ Perforations
- ★ Intra abdominal abscess
- ★ Fistulas
- ★ Haemorrhage
- ★ Enterolithiasis
- ★ Traction diverticula

#### **(1). Obstruction**

This is the most common complication. Incidence is of 10% of cases. This may occur as follows.

Constriction of collagenous tissue following healing of encircling ulceration.

Encroachment of thickened bowel wall upon lumen through scar tissue.

Constriction of intestine by intraperitoneal adhesions.

Retraction of mesentery and shortening of right colon in the healing place.

This is treated by resection and anastomosis or by stricturoplasty.

Bhanshali(1967) reported that in India 5 to 7% of all perforation and 3.4 to 11% of intestinal obstruction and due to intestinal tuberculosis.

## **(2). Perforations**

Free perforations are uncommon due thickening, and adhesions of adjacent of tissue, ileal perforation incidence is about 0-11%. Terminal ileum is the frequent site. It is postulated that lymphoid tissues may develop hypersensitivity rapidly to M.Tuberculosis resulting in ulceration and perforation. Treatment is by simple closure or segmental resection.

## **(3). Fistulas**

This develop in 1 to 33% of patient, they develop between loops of bowel (or) between bowel and abdominal wall or urinary bladder or female adnexal organs. Excision and repair should be carried out depending upon the site of Fistula.

## **(4). Malnutrition**

This is due to malabsorption, this may due to

- ★ Decreased small intestinal mucosal surface.
- ★ Lymphatic obstruction.
- ★ Fistula formation between small and large intestines.
- ★ Do conjugation of bile salts, secondary to bacterial growth.
- ★ Decreased bile salt pool due impaired absorption in distal ileum.

## Management

All patients should receive conventional anti-tubercular 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* 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 . Bypassing the stenosed segment by entero-enterostomy or by ileo-transverse 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. 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*<sup>57</sup> 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 . Similar observations were made by Balasubramaniam *et.al* . The mean time required for the relief of obstructive symptoms was 6 months.

### **I. Conservative Management:**

This mainly by anti tuberculosis drugs. Drugs susceptibility should be done and drugs chosen to which tubercle bacilli is susceptible should be used. More than 2 drugs to be combined to avoid multiplication of drug resistant mutants.

These are first and second line of anti tuberculosis drugs available and 2 regimes available.

- ❖ First line drugs are INH, Rifamycin Ethambutol and streptomycin.
- ❖ Second line drugs are Pyrazinamide, Ethoinamide, Cycloserine, Para-amino Salicylic acid, Kanamycin and Capremycin etc.

Conventional regimens :

These consists of INH + Ethambutol with or without SM (initial +2 months),

Optimum result requires 12-18 months therapy.

**Short course regimens :**

(i) Four Drugs, INH + RMP + PZM + Emb (or) SM daily for 2 months, followed INH + RMP daily for another 4 months, total 6 months.

(or)

(ii) The above four drugs followed by INH + EMB daily for another 6 months-

This is economical.

(iii) Recently : It has been shown that SM/EMB can be eliminated as a routine adjunct unless, INH resistance is suspected. Combination of 3 drugs INH + RMP + PZM for initial 2 months and INH for next 4 months is considered adequate.

## II. Operative Treatment

Indications for surgery in abdominal tuberculosis are two reasons. Like Diagnostic and Therapeutic

### (A). **Diagnostic** laparotomy

Becomes necessary for histopathological/microbiological diagnosis, more often in patients with peritoneal and/or lymph node tuberculosis.

### (B). **Therapeutic** surgery is indicated for complications like

1. Obstruction due to stenosis or kinking of bowel, and
2. Perforation combined perforation with abscess (or)
3. Fistula formation
4. Failure of conservative therapy.

## SURGICAL PROCEDURES

### (1). **Perforation closure : and drainage of peritoneal cavity(Prakash 1975).**

Tuberculosis perforations are usually associated with the structures, simple suture of perforation with drainage of peritoneal cavity as advocated by Prakash (1975) can be done. Suture line may be reinforced with a patch of omentum.

### (2). Segmental resections and anastomosis

Multiple ileal tubercular lesions affecting a short segment of bowel are treated by excision of diseased segment of bowel and end to end anastomosis.

### **(3). Stricturoplasty**

Localised the circumferential stricture either single or multiple are treated by stricture plasty (R.N.Kataria). The bowel is opened longitudinally, on the anti-mesenteric side across the lesion extending the incision on either side of lesion. A portion is trimmed from edge of lesion for biopsy and bowel is closed transversely in two layers.

### **(4) Ileoplasty**

In tubular lesions, of long loop ileoplastics, can be done, In granulomatous lesions oval or rhomboid portion of the lesions is excised on the antimesenteric border with its long of the bowel, and the defect was closed transversely even if perforation. Ileoplasty can be tried including the perforation.

#### **Advantages of Ileoplasty**

- a. Large functional part of ileum is preserved and no part of gut is excised.
- b. Eliminates short bowel syndrome and blind loop syndrome.
- c. Safe, simple, effective in anemic, hypoproteinemic and chronically ill patients.
- d. Procedure can be carried out in patients with active tuberculosis enteritis who have not had antituberculous treatment earlier.
- e. Biopsy can be taken from the edge of incision for HPE.

### **(5). Limited Ileocecal Resection(Right Quarter Colectomy)**

Advocated by Dutta Gupta in 1958, and Sharma and Mehta in 1964, for early ileocecal tuberculosis. The procedure includes mobilization of ileocecal region and limited resection extending 5cm on either side of the lesion and end to end ileocolic anastomosis. Abdomen is closed after reperitonealisation.

#### (6). Bypass procedures

Entero – enterostomy and ileotransverses colostomy were practiced in preantibiotic (Pird, 1957)

- This is likely to produce a blind loops.
- Stricture of remaining segments may produce fistulas or recurrent obstruction (Faulkner 164).
- How even in case of duodenal tuberculosis, Gastro
- Jejunostomy is done in many centers.

### HIV AND TUBERCULOSIS

Abdominal tuberculosis in HIV-infected patients is invariably a manifestation of disseminated disease and results in significant mortality. Extrapulmonary tuberculosis is seen in over 50 percent of patients. Fever, weight loss, lymphadenopathy, and splenic abscesses are seen more commonly in HIV-infected patients than in those without HIV infection.

The diagnostic techniques are the same as in uninfected individuals and include bacteriological testing of body fluids, abdominal ultrasound and CT scanning combined with guided-needle aspiration biopsies, barium examinations, fiberoptic endoscopy, and laparoscopy. Surgical tests such as enzyme-linked immunosorbent assay lack sensitivity due to poor humoral immune response. Most patients respond well to conventional antituberculous drugs use in standard doses. Some experts recommend a longer course of therapy(9 months). Despite treatment, a few patients experience a downhill course, which may be due to drug resistance or the presence of overwhelming infection.

## ***Prognosis***

The prognosis of uncomplicated abdominal tuberculosis is good. Most patients respond well to medical therapy and the outcome of surgical management of complications is generally good. Bowel obstruction or perforation associated with plastic adhesions and the development of enterocutaneous fistulas with intra-abdominal abscesses are associated with increased morbidity and poor prognosis.

## ***MATERIALS AND METHODS***

The present study was conducted as a prospective study from August 2003 to March 2006 at the department of general surgery, Government Stanley Medical College, Chennai.

Fifty patients were pooled in the study who had presented in the surgical outpatient clinic or in the emergency department.

- (i) All these patients were admitted to surgical wards
- (ii) A detail history was obtained.
- (iii) Thorough clinical examination was performed.
- (iv) All patients were subjected to basic lab investigations. Complete blood count , ESR, blood sugar, blood urea, serum creatinine , serum electrolytes, and urine analysis.
- (v) Every patient had an chest x-ray, plain abdominal x-ray done.
- (vi) Barium series, CT scan were done for those patients with inconclusive Ultrasound abdomen. All patients who had been admitted in the emergency wards were not subjected for both barium series and CT scan.
- (vii) Mantoux test was done in all patients with chest x-ray suggestive of TB, postoperatively in patients who had features suggestive of tuberculosis.
- (viii) Conservative measures were started when indicated in the form of NPO, RTA rehydration according to the state of hydration and electrolytes.

- (ix) Tissue diagnosis by histopathological examination had proved all patients to be positive for tuberculosis.
- (x) The details of all the patients were recorded and the results were analyzed.

## RESULTS AND OBSERVATIONS

### Demographics :

#### (1) Age distribution.

The present series involving 50 patients ranging from the second decade of life to the seventh decade of life. The youngest patient of the series was 12 year old and the oldest being 62 year old. The average age of the patients in this series was 30.8 years.

Age group	No. of patients (n)
≤ 20 years	12
21-30 years	16
31-40 years	09
41-50 years	11
Above 50 years	02

#### (2) Sex distribution:

The present study included 26 male patients and 24 female patients.

Fig.13: Sex distribution

## Clinical Presentation:

### Symptomatology .

The patient in this series present with symptoms ranging from abdominal pain to constitutional symptoms alone such as loss of weight, loss of appetite, fever, and altered bowel habits. The frequency of symptoms are tabulated in the table No. 8.

*Table No.8: Symptomatology*

S. No.	Symptoms	No.of patients (n)	Percentage
1.	Pain abdomen	44	88%
2.	Vomiting	26	52%
3.	Abdominal Distension	19	38%
4.	Fever	23	46%
5.	Diarrhea	08	16%
6.	Constipation	19	38%
7.	Weight loss	22	44%
8.	Loss of appetite	27	54%

Abdominal pain was the common symptom with which the patients having abdominal tuberculosis presented. Vomiting and loss of appetite were also common presenting complaints accounting for 52% and 54% respectively.

Diarrhea was present in 16% of the patients.

### Physical signs:

The most common physical finding was abdominal tenderness which was diffuse. 58% patients had presented with abdominal tenderness. Only 18% of patients had ascites and 38% had visible intestinal peristalsis.

*Table No.9 : Physical signs*

S. No.	Signs	No.of patients (n)	Percentage
1.	Abdominal tenderness	29	58%
2.	Mass abdomen	14	28%
3.	Visible peristalsis	19	38%
4.	Ascites	09	18%
5.	Gurgling	18	36%

## Investigations

S. no	Investigations	No. of patients	Positive	Negative	% positive
1.	ESR	50	Raised 27	Normal 23	54%
2.	Mantoux test	35	15	20	42.86%
3.	Colonoscopy	13	05	08	38.5%

.

Plain abdominal x-ray was taken in all patients. 18 films showed dilated loops with multiple air fluid levels three film showed air under diaphragm.

Barium meal series was performed in 5 patients. Barium enema examinations in 6 patients. Various signs of tuberculosis like multiple strictures and distended caecum or terminal ileum displaced loops adherent fixed loops flocculation and fragmentation were seen in few cases These findings were nonspecific and seen in other conditions like Crohn's malignancy and ulcerative colitis etc

Mantoux test was done in 35 patients and it was strongly positive in 15 patients . This test was non specific as it just gives idea about immune status of the patients.

ESR was done in almost all patients and was elevated in 27 patients. this test just reflect ongoing inflammation which also seen in some other conditions

COLONOSCOPY was done in 13 patients biopsy was taken in all patients .Biopsy was positive in 5 patients and in the remaining patients nonspecific inflammatory reaction

ULTRASOUND abdomen was taken in 20 cases in 18 cases various signs of tuberculosis like asymmetric thickening of bowel wall peritoneal thickening nodularity were seen.

ASCITIC fluid examinations were done for 2 patients. Analysis showed all were predominantly lymphocytes, and protein level raised from 3.5 to 5 gm.

CT SCAN was taken in 14 patients. The majority of patients CT picture showed signs of tuberculosis like bowel wall thickening, irregular soft tissue densities and lymphadenopathy, high density ascites and smooth and thick and peritoneum.

Cervical lymph node biopsy was done in two patients and were positive for tuberculosis.

### **Sites of abdominal TB**

Ileocaecal junction was the most common site of abdominal tuberculosis with 32% of the patients, followed by the terminal ileum. Overall the incidence of tuberculosis in the ileocaecal region was 46%.

## INCIDENCE OF ABDOMINAL TB ACCORDING TO THE SITE OF INVOLVEMENT

<b>s.no</b>	<b>Site</b>	<b>n</b>
1.	Ileocaecal TB	16
2.	Ileal TB	07
3.	Jejunal TB	02
4.	Peritoneal TB	10
5	Mesentric Lymphadenitis	13
6	Gastroduodenal TB	01
7	Solid organ TB	01
8	Combination of above	14

### *Accuracy of clinical examinations:*

The clinical accuracy was 50 percent Remaining 50/ patients provisionally diagnosed as recurrent intestinal obstruction, recurrent appendicitis ileocaecal growth, Crohn's disease peptic ulcer and recurrent UTI

### ***ASSOCIATED TUBERCULOSIS***

Associated pulmonary tuberculosis was present in 12 out of 50 patients that is 24%.

### ***TREATMENT***

Surgical treatment was offered to the patients who presented with acute conditions like perforation, and obstruction. Fifteen patients underwent, emergency surgery and 28 patients underwent elective surgery.

Emergency surgery for intestinal obstruction was done in 8 patients. In the form of resection and anastomosis was done in 3 patients limited resection and ileostomy was done in two patients and limited resection and anastomosis in one patient . One patient underwent RT hemicolectomy . one patient underwent adhesiolysis alone

Multiple stricture was present in 4 patients . Two patients presented with multiple strictures of short segment of ileum underwent resection and end to end anastomosis

One patients presented with multiple stricture in ileum and jejunum underwent resection and anastomosis .Another one patients presented with multiple stricture jejunum underwent resection and anastomosis.

Emergency surgery for perforation was done in 5 patients .Of whom 2 patients underwent resection and anastomosis. One patient underwent limited resection and anastomosis. One patient underwent perforation closure and biopsy alone . One patient underwent resection and ileostomy. Three patients underwent emergency appendectomy and biopsy was taken from omentum and mesenteric nodes.

Elective surgery was done in 28 patients, of whom 11 patients underwent laparotomy and biopsy alone . RT hemicolectomy was done in 6 patients. Limited resection and anastomosis was done in 4 patients. Extended Rt.hemicolectomy was done in one patient. Subtotal gastrectomy was done in one patient for suspected malignancy. Laparoscopy was done in 3 patients. Peritoneal and omental biopsy were taken.

One patient with ileovesical fistula underwent disconnection of fistula,, ileostomy and biopsy from bladder wall and mesenteric nodes. One patient with ileoumbilical fistula underwent resection of ileum and excision of fistulous tract. 7 patients were managed conservatively and Antituberculous therapy started. All the post operative patients were put on Antituberculous therapy and regularly followed up.

## DISCUSSION

### ***AGE and SEX***

Age incidence of present series is similar to reported by other workers [Adams and Miller,1946, Dutta Gupta1950,Sharma et al ,1972 B.K Bhansali,1968].

In our series commonest age group affected was 2<sup>nd</sup> and 3<sup>rd</sup> decades of life .Pritam Das and Sukula .et.al 1978 , also reported same age group.

Overall sex incidence is equal in male and female. In the present study males accounted for 26 patients with male to female ratio being 1.08 :1 . thus an almost equal sex incidence. Addison et al 1981 reported high incidence in males. M Ismail.et.al had reported the same male to female ratio in his study on 50 patients.

### **CLINICAL PRESENTATION .**

#### **Symptomatology:**

M P Sharma and Vikram Bhatia stated in their review article about the clinical presentation dominated by constitutional symptoms. 40-70% of patients present with fever, 80-95% with pain, 11-20% with diarrhea, 40 -90% with weight loss.

N.Rangabashyam.et.al cited vague symptoms to be the predominant presenting symptoms. The abdominal pain accounts for in 77-94% of patients, followed by vomiting and abdominal distension.

S. No.	Symptoms	S.K <i>B h a n s a l i e t a l 1 9 7 7</i> n=135	Tariq sinan .et.al 2002 n=49 (%)	Ali Uzunkoy.et.a 1 2004 n=11	<b>Present series 2006 n=50 (%)</b>
1.	Pain abdomen	103	37 (75.5)	8	<b>44 (88)</b>
2.	Vomiting	89	07(14.2)	-----	<b>26 (52)</b>
3.	Abdominal Distension	60	-----	7	<b>19 (38)</b>
4.	Fever	58	32 (65.3)	-----	<b>23 (46)</b>
5.	Diarrhea	21	1(2)	-----	<b>08 (16)</b>
6.	Constipation	55	-----	-----	<b>19 (38)</b>
7.	Weight loss	26	18 (36.7)	9	<b>22 (44)</b>
8.	Loss of appetite	58	-----	5	<b>27 (54)</b>

Majority of patients in the present study had present with chronic symptoms. 15 out of 50 patients had presented with an acute abdomen and underwent emergency surgery for the same. J.M.Findly 1981 found that acute presentation was present in 13.5% patients.

Commonest symptom is vague abdominal pain .Other common symptoms were vomiting, fever, loss of appetite and loss of weight. Pritam Das and Sukula had reported abdominal pain in 94% of cases

Bockus et al 1964 emphasised abdominal pain , anorexia ,loss of weight as common presenting symptoms. Shukula 1970 and L.E Hugas also reported these symptoms as common presentation

Any patient present with vague abdominal pain, vomiting , loss of appetite , loss of weight and not fitting with any other clinical diagnosis , diagnosis of abdominal tuberculosis should be considered and ruled out first

Diarrhoea and constipation were seen in fewer patients. Duration of symptoms before presentation to casualty or outpatient clinic was less than a year Bhansali and Desai[1968] reported a duration of symptoms less than one year in most cases. Other workers [Levine 1968, Sharma et al 1972] reported duration of symptoms 1to 3 years commonest

### ***PHYSICAL SIGNS***

Tenderness and abdominal distension were most frequent local signs. In the present study majority of the patients presented with tenderness, abdominal distension and mass in right iliac fossa. Doughy feel was not present in any case and doughy feeling probably needs experienced hands for diagnosis as it was reported as common clinical entity by many authors.

Most of our patients were illnourished.

The results were more or less similar to reports by Pritam Das et al 1951, Bhansali and Desai 1968 Tailor 1945 ,Sharma et al and J.M.Findley 1978. and Ali Uzunkoy.et.al 2004.

## ***ASSOCIATED PULMONARY TUBERCULOSIS***

Associated pulmonary tuberculosis was seen in 12 patients and rest had primary gastrointestinal tuberculosis. But higher incidence of associated pulmonary tuberculosis was reported by some workers [Hoon et al 1950, Bobro and Friedman 1956, Prakash et al 1970 ] and lower in incidence [3.7%] by Sharma et al 1972.

## ***INVESTIGATIONS***

The haemoglobin level, ESR total leucocyte count was unhelpful since the findings could have compatible with any of the other principle differential diagnosis. Minimal anemia and lymphocytosis could be present in any other chronic disease. ESR is an well non specific.

Mantoux test was positive in 15 of 50 patients indicating its limited value in the diagnosis of Abdominal tuberculosis. Only 30% positive results reported by J. M Findley 1976 and Andreas et al. 1980.

Plain X-ray abdomen showed multiple fluid levels in 18 cases and air under diaphragm 3 cases. Chest X-ray shoed evidence of tuberculosis in 12 patients.

Barium meals and Barium enema studies was done in 5 patients and 6 patients respectively. Certain signs thought non specific were present in few patients suggesting a possibility of tuberculosis.

Peritoneal Biopsy hold promise in diagnosing abdominal tuberculosis in ascitic and non ascitic group of patients. Reports from various centers are encouraging and holds much good for the further as diagnosis tool.

Taylor (1945) and Burrack and Hollister (1960) considered a case tuberculosis. If the ascitic fluid contained more than 250 cell/mm<sup>3</sup> and a protein level more that 2.5 gm%. In the present study, ascitic fluid examination was done

for 2 patient. Analysis of ascitic fluid showed, predominately lymphocytes and protein level more than 5 gm%. We did not employ culture or guinea pig inoculation tests,. Laparoscopy is ideal for diagnosis. With problems in diagnosis, at times exploratory laparotomy becomes essential for confirmation of the disease and tissue diagnosis.

Ever histologically it is at times difficult to distinguish from Chron's disease, When there is an absence of caseation due to probably modification of immune process. The only absolute means of diagnosing tuberculosis is by the actual demonstration of tuberculosis mycobacteria in the tissue section.

Tuberculosis may involve any part of intestinal tract, but ileocecal region is the commonly involved site. Strictures of ileum at the commonest pathology, and commonly associated with intestinal obstruction, we have one case gastric present as peptic ulcer for which subtotal gastrectomy was done

In India about 3 to 20 % of all intestinal obstructions are due to Abdominal tuberculosis ( Jarin and Pradah 1963, Gull and Eggleston, 1965, Bhasali et al 1970). The incidence of perforations of small intestines was 7.6% according to Bhansali et al. (1978) In present study we have 2 patients with perforation at ileal regions.

In the present study, 15 patients underwent emergency surgery. 28 patients underwent surgery, and 7 patients were put to medical treatment with antituberculosis drugs. The above operated patients were continued with antituberculosis regimens, there was no mortality in our series. Modified surgical procedures for tuberculosis were suggested by B.D.Pujarari (1979). Modified procedures like limited ileo-cecal resections and ileoplasty are safe, quick and easy, with preservation of functional portions of the bowel. They are of immense value in the emergencies in chronologically ill and emaciated patients. Blind loop syndrome, the major disadvantage of bypass procedures is eliminated. Chemotherapy is the main modality of treatment and improvement of nutrition and sanitation.

## **CONCLUSION**

- a) ABDOMINAL TUBERCULOSIS IS MOST COMMON AMONG THE MIDDLE AGE GROUP**
- b) THE MOST COMMON SYMPTOM WAS THE ABDOMINAL PAIN**
- c) THE MOST COMMON INVOLVEMENT IN ABDOMINAL TUBERCULOSIS WAS ILEOCAECAL JUNCTION WITH MESENTERIC LYMPHADENITIS.**
- d) INTESTINAL OBSTRUCTION SECONDARY TO STRICTURES IN DISTAL ILEUM AND ILEOCAECAL MASS WAS THE MOST COMMON MANIFESTATION IN THE EMERGENCY.**
- e) NO IMAGING STUDIES WERE CONCLUSIVE OF ABDOMINAL TUBERCULOSIS,**
- f) ABDOMINAL TUBERCULOSIS CAN MIMIC AS MALIGNANT LESIONS.**

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## BIBLIOGRAPHY

1. Addison N.V. & J.M. Findlay. Abdominal Tuberculosis. Current Surgical practice vol. 3 page, 48-61(1982).
2. Ali W, Sikora SS, Banerjee D, Kapoor VK, Saraswat VA, Saxena R, *et al.* Gastroduodenal tuberculosis. *Aust NZ JSurg* 1993; 63 : 466-7.
3. Ali Uzunkoy, Muge Harma, Mehmet Harma. Diagnosis of abdominal tuberculosis: Experience from 11 cases and review of the literature. *World J Gastroenterol* 2004; 10(24):3647-3549
4. Anand BS, Nanda R, Sachdev GK. Response of tuberculous stricture to antituberculous treatment. *Gut* 1988; 29 : 62-9.
5. Anand BS. Distinguishing Crohns disease from intestinal tuberculosis. *Natl Med J India* 1989; 2 : 170-5.
6. Anand S.S. Hypertrophic ileo-caecal tuberculosis in INDIA and record of 50 colectomies. *Ann. R . 19:205-207, (1956)*
7. Anderson K.E. et al. TB of Duodenum. *Am J of Surgery* 1954; 88 : 952-959.
8. Andreas L. Lambrainides et.al. Adbominal tuberculosis Current Surgical Practice 1982; Vol. 3 page.48-61.
9. Arunab, Kapoor , Chattpadhyaya & Sharma L.K. TB peritonitis presenting as acute abdomen. *Ind .J .of Tuberculosis (1986); 33:190-191.*
10. Arya TVS, Jain AK, Kumar M, Agarwal AK, Gupta JP. Colonic tuberculosis : a clinical and colonoscopic profile. *Indian J Gastroenterol* 1994; 13 (Suppl) A 116.
11. Balasubramanian R, Nagarajan M, Balambal R, Tripathy SP, Sundararaman R, Venkatesan P. Randomised controlled clinical trial of

- short course chemotherapy in abdominal tuberculosis: a five-year report. *Int J Tuberc Lung Dis* 1997; 1 : 44-51.
12. Balasubramanian R, Ramachandran R, Joseph PE, Nagarajan M, Thiruvengadam KV, Tripathy SP, *et al.* Interim results of a clinical study of abdominal tuberculosis. *Indian J Tuberc* 1989; 36 : 117-21.
  13. Barutcu O, Erel HE, Saygili E, Yildirim T, Torun D. Abdominopelvic tuberculosis simulating disseminated ovarian carcinoma with elevated CA-125 level: report of two cases. *Abdom Imaging* 2002; 27: 465-470
  14. Berney T, Badaoui E, Totsch M, Mentha G, Morel P. Duodenal tuberculosis presenting as acute ulcer perforation. *Am J Gastroenterol* 1998; 93 : 1989-91.
  15. Bhansali S.K & Desai. Abdominal tuberculosis- 135 cases review. *Indian J Surg* 1968, 30: 218.
  16. Bhansali SK, Sethna JR. Intestinal obstruction: a clinical analysis of 348 cases. *Indian J Surg* 1970; 32 : 57-70.
  17. Bhansali SK. Abdominal tuberculosis. Experiences with 300 cases. *Am J Gastroenterol* 1977; 67 : 324-37.
  18. Bhargava DK, Dasarathy S, Shriniwas MD, Kushwaha AKS, Duphare H, Kapoor BML. Evaluation of enzyme315 linked immunosorbent assay using mycobacterial salineextracted antigen for the serodiagnosis of abdominal tuberculosis. *Am J Gastroenterol* 1992; 87 : 105-8.
  19. Bhargava DK, Kushwaha AKS, Dasarathy S, Shriniwas, Chopra P. Endoscopic diagnosis of segmental colonic tuberculosis. *Gastrointest Endosc* 1992; 38 : 571-4.
  20. Bhargava DK, Shriniwas, Chopra P, Nijhawan S, Dasarathy S, Kushwaha AK. Peritoneal tuberculosis: laparoscopic patterns and its diagnostic accuracy. *Am J Gastroenterol* 1992; 87 : 109-12.

21. Bhargava DK, Tandon HD, Chawla TC, Shriniwas, Tandon BN, Kapur BM. Diagnosis of ileocecal and colonic tuberculosis by colonoscopy. *Gastrointest Endosc* 1985; 31 : 68-70.
22. Bilgin T, Karabay A, Dolar E, Develioglu OH. Peritoneal tubercuosis with pelvic abdominal mass, ascites and elevated CA 125 mimicking advanced ovarian carcinoma: a series of 10 cases. *Int J Gynecol Cancer* 2001; 11: 290-294
23. Bouma BJ, Tytgat KM, Schipper HG, Kager PA. Be aware of abdominal tuberculosis. *Neth J Med* 1997; 51: 119-122
24. Chattpadhyaya et al. Peritoneal biopsy in TB ascites. *JIMA* 1981; 76:206.
25. Chaudhary A, Gupta NM. Colorectal tuberculosis. *Dis Colon Rectum* 1986; 29 : 738-41.
26. Chawla S, Mukerjee P, Bery K. Segmental tuberculosis of the colon: a report of ten cases. *Clin Radiol* 1971; 22 :104-9.
27. Chowdhary GN, Dawar R, Misra MC. Coexisting carcinoma and tuberculosis of stomach. *Indian J Gastroenterol* 1999; 18 : 179-80.
28. Demir K, Okten A, Kaymakoglu S, Dincer D, Besisik F, Cevikbas U, Ozdil S, Bostas G, Mungan Z, Cakaloglu Y. Tuberculous peritonitis-reports of 26 cases, detailing diagnostic and therapeutic problems. *Eur J Gastroenterol Hepatol* 2001; 13: 581-585
29. DiFebo G, Calabrese C, Areni A, Savastio G, Grazia M, Miglioli M. Oesophageal tuberculosis mimicking secondary oesophageal involvement by mediastinal neoplasm. *Ital J Gastroenterol Hepatol* 1997; 29 : 564-8.
30. Dorairajan LN, Gupta S, Deo SV, Chumber S, Sharma L. Peritonitis in India – a decade’s experience. *Trop Gastroenterol* 1995; 16 : 33-8.
31. Dutta Gupta A.K. Intestinal Tuberculosis. *Indian J Surg*1958;20:386-400.

32. Dwivedi M, Misra SP, Misra V, Kumar R. Value of adenosine deaminase estimation in the diagnosis of tuberculous ascites. *Am J Gastroenterol* 1990; 85 : 1123- 5.
33. Findly .J.H Gastrointestinal tuberculosis- Resent Advances in surgery vol. 10, Ed. Selwyn Taylor.
34. Gill SS, Eggleston FC. Acute intestinal obstruction. *Arch Surg* 1965; 91 : 589-91.
35. Gupta OP, Dube MK. Tuberculosis of gastrointestinal tract: with special reference to rectal tuberculosis. *Indian J Med Res* 1970; 58 : 979-84.
36. Gupta SK, Jain AK, Gupta JP, Agrawal AK, Berry K. Duodenal tuberculosis. *Clin Radiol* 1988; 39 : 159-61.
37. HK Ha, JI Jung, MS Lee, Choi BG, Lee MG, Kim YH. CT differentiation of tuberculosis peritonitis and peritoneal carcinomatosis. *Am J Roentgenol* 1996; 167: 743-8.
38. Jadvar H, Mindelzun RE, Olcott EW, Levitt DB. Still the great mimicker: abdominal tuberculosis. *Am J Roentgenol* 1997; 168: 1455-1460
39. JainA.K:LalRK Guptha S:Singhal .M,Guptha J.P., ELISA in GUT tuberculosis. *Indian J Gastroenterol* 1986.5:175-177.
40. Kapoor VK. Abdominal tuberculosis : the Indian contribution. *Indian J Gastroenterol* 1998; 17 : 141-7.
41. Kapoor VK. Abdominal tuberculosis :Misconception, myths and facts. *Indian J Tub.*, 1991; 38 : 119-122.
42. Kapoor VK. Abdominal tuberculosis. *Postgrad Med J* 1998; 74 : 459-6.
43. Kataria .R.N et al.:Strictureplasty for TBstrictures of GItract, *Br J Surg* 1977 ;64:496-498.
44. Lal N, Soto-Wright V. Peritoneal tuberculosis: diagnostic options. *Infect Dis Obstet Gynecol* 1999; 7: 244-247

45. Lantheaume S, Soler S, Issartel B, Isch JF, Lacassin F, Rougier Y, Tabaste JL. Peritoneal tuberculosis simulating advanced ovarian carcinoma: a case report. *Gynecol Obstet Fertil* 2003; 31: 624-626
46. Mahdavi A, Malviya VK, Herschman BR. Peritoneal tuberculosis disguised as ovarian cancer: an emerging clinical challenge. *Gynecol Oncol* 2002; 84: 167-170
47. Malik A, Saxena NC. Ultrasound in abdominal tuberculosis. *Abdom Imaging* 2003; 28: 574-579
48. Martin JR, Whitted R, Latchaw GA, Yebara S. Complications of operative and diagnostic laparoscopy: a retrospective study. *Obstet Gynecol* 2001; 97: S20
49. Moatter T, Mirza S, Siddiqui MS, Soomro IN. Detection of Mycobacterium tuberculosis in paraffin embedded intestinal tissue specimens by polymerase chain reaction: characterization of IS6110 element negative strains. *J Pak Med Assoc* 1998; 48: 174-178
50. 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
51. Nair KV, Pai CG, Rajagopal KP, Bhat VN, Thomas M. Unusual presentations of duodenal tuberculosis. *Am J Gastroenterol* 1991; 86 : 756-60.
52. Ozalp S, Yalcin OT, Tanir HM, Kabukcuoglu S, Akcay A. Pelvic tuberculosis mimicking signs of abdominopelvic malignancy. *Gynecol Obstet Invest* 2001; 52: 71-72
53. Panoskaltis TA, Moore DA, Haidopoulos DA, McIndoe AG. Tuberculous peritonitis: part of the differential diagnosis in ovarian cancer. *Am J Obstet Gynecol* 2000; 182: 740-742

54. Paustian FF. Tuberculosis of the intestine. In: Bockus HL, editor. *Gastroenterology*, vol.11, 2nd ed. Philadelphia : W.B. Saunders Co.; 1964 p. 311.
55. Peda Veerraju E. Abdominal tuberculosis. In: Satya Sri S, editor. *Textbook of pulmonary and extrapulmonary tuberculosis*. 3rd ed. New Delhi: Interprint; 1998 p. 250-2.
56. Pimparkar BD, Donde UM. Intestinal tuberculosis II. Gastrointestinal absorption studies. *J Assoc Physicians India* 1974; 22 : 219-28.
57. Pimparkar BD. Abdominal tuberculosis. *J Assoc Physicians India* 1977; 25 : 801-11.
58. Piura B, Rabinovich A, Leron E, Yanai-Inbar I, Mazor M. Peritoneal tuberculosis-an uncommon disease that may deceive the gynecologist. *Eur J Obstet Gynecol Reprod Biol* 2003; 110: 230-234
59. Piura B, Rabinovich A, Leron E, Yanai-Inbar I, Mazor M. Peritoneal tuberculosis mimicking ovarian carcinoma with ascites and elevated serum CA-125: case report and review of literature. *Eur J Gynaecol Oncol* 2002; 23: 120-122
60. Prakash A. Ulcero-constrictive tuberculosis of the bowel. *Int Surg* 1978; 63 : 23-9.
61. Protopapas A, Milingos S, Diakomanolis E, Elsheikh A, Protogerou A, Mavrommatis K, Michalas S. Miliary tuberculous peritonitis mimicking advanced ovarian cancer. *Gynecol Obstet Invest* 2003; 56: 89-92
62. Pujari BD. Modified surgical procedures in intestinal tuberculosis. *Br J Surg* 1979; 66 : 180-1.
63. Puri AS, Vij JC, Chaudhary A, Kumar N, Sachdev A, Malhotra V, *et al.* Diagnosis and outcome of isolated rectal tuberculosis. *Dis Colon Rectum* 1996; 39 : 1126-9.

64. Rai S, Thomas WM. Diagnosis of abdominal tuberculosis: the importance of laparoscopy. *J R Soc Med* 2003; 96: 586-588
65. Ranjan P, Ghoshal UC, Aggarwal R, Pandey R, Misra A, Naik S, *et al.* Etiological spectrum sporadic malabsorption syndrome in Northern Indian adults at a tertiary hospital. *Indian J Gastroenterol* 2004; 23 : 94-8.
66. Rathi PM, Amarapurakar DN, Parikh SS, Joshi J, Koppikar GV, Amarapurkar AD, *et al.* Impact of human immunodeficiency virus infection on abdominal tuberculosis in western India. *J Clin Gastroenterol* 1997;24 : 43-8.
67. Rowell.J.S *et al.* Ileocaecal TB. *Gut* 1964; 5:254.
68. Sathar MA, Simjer AE, Coovadia YM, Soni PN, Moola SA, Insam B, *et al.* Ascitic fluid gamma interferon concentrations and adenosine deaminase activity in tuberculous peritonitis. *Gut* 1995; 36 : 419-21.
69. Schwake L, von Herbay A, Junghanss T, Stremmel W, Mueller M. Peritoneal tuberculosis with negative polymerase chain reaction results: report of two cases. *Scand J Gastroenterol* 2003; 38: 221-224
70. Shah P, Ramakantan R, Deshmukh H. Obstructive jaundice - an unusual complication of duodenal tuberculosis : treatment with transhepatic balloon dilatation. *Indian J Gastroenterol* 1991; 10 : 62-3.
71. Shah P, Ramakantan R. Role of vasculitis in the natural history of abdominal tuberculosis - evaluation by mesenteric angiography. *Indian J Gastroenterol* 1991; 10: 127-30.
72. Sharma AK, Agarwal LD, Sharma CS, Sarin YK. Abdominal tuberculosis in children : experience over a decade. *Indian Peadiatr* 1993; 30 : 1149-53.
73. Sheer TA, Coyle WJ. Gastrointestinal tuberculosis. *Curr Gastroenterol Rep* 2003; 5: 273-278

74. Sinan T, Sheikh M, Ramadan S, Sahwney S, Behbehani A. CT features in abdominal tuberculosis: 20 years experience. *BMC Medical Imaging* 2002; 2: 3-16
75. Singh V, Kumar P, Kamal J, Prakash V, Vaiphei K, Singh K. Clinicocolonoscopy profile of colonic tuberculosis. *Am J Gastroenterol* 1996; 91 : 565-8.
76. Tandon HD, Prakash A. Pathology of intestinal tuberculosis and its distinction from Crohn's disease. *Gut* 1972; 13 : 260-9.
77. Tandon RK, Bansal R, Kapur BML, Shrinivas. A study of malabsorption in intestinal tuberculosis : stagnant loop syndrome. *Am J Clin Nutr* 1980; 33 : 244-50.
78. Tandon RK, Sarin SK, Bose SL, Berry M, Tandon BN. A clinico-radiological reappraisal of intestinal tuberculosis – changing profile? *Gastroenterol Jpn* 1986; 21 : 17-22.
79. Tassios P, Ladas S, Giannopoulos G, Larion K, Katsogridakis J, Chalarelakis G, *et al.* Tuberculous esophagitis. Report of a case and review of modern approaches to diagnosis and treatment. *Hepatogastroenterology* 1995; 42 : 185-8.
80. Thakur V, Mukherjee U, Kumar K. Elevated serum cancer antigen 125 levels in advanced abdominal tuberculosis. *Med Oncol* 2001; 18: 289-291
81. Tzoanopoulos D, Mimidis K, Giaglis S, Ritis K, Kartalis G. The usefulness of PCR amplification of the IS6110 insertion element of *M. tuberculosis* complex in ascitic fluid of patients with peritoneal tuberculosis. *Eur J Intern Med* 2003; 14: 367-371
82. Vij JC, Malhotra V, Choudhary V, Jain NK, Prasaed G, Choudhary A, *et al.* A clinicopathological study of abdominal tuberculosis. *Indian J Tuberc* 1992; 39 : 213-20.

83. Vij JC, Ramesh GN, Choudhary V, Malhotra V. Endoscopic balloon dilation of tuberculous duodenal strictures. *Gastrointest Endosc* 1992; 38 : 510-1.
84. Voigt MD, Kalvaria I, Trey C, Berman P, Lombard C, Kirsch RE. Diagnostic value of ascites adenosine deaminase in tuberculous peritonitis. *Lancet* 1989; 1: 751-754
85. Wadhwa N, Agarwal S, Mishra K. Reappraisal of abdominal tuberculosis. *J Indian Med Assoc* 2004; 102 :31-2.
86. Wig KL, Chitkara NK, Gupta SP, Kishore K, Manchanda RL. Ileocecal tuberculosis with particular reference to isolation of *Mycobacterium tuberculosis*. *Am Rev RespirDis* 1961; 84 : 169-78.
87. Wu JF, Li HJ, Ni YH, Yu SC, Chang MH. Tuberculous peritonitis mimicking peritonitis carcinomatosis: a case report. *Eur J Pediatr* 2003; 162: 853-855
88. Zaidi SN, Conner M. Disseminated peritoneal tuberculosis mimicking metastatic ovarian cancer. *South Med J* 2001; 94: 1212-1214
89. Zhang Z, Shi X, Li J. Abdominal tuberculosis is diagnosed as tumor. *Zhonghua Jiehe He Huxi Zazhi* 2001; 4: 400-403
90. M Ismail, Farmanullah, Mumtaz Khan. Surgical management of abdominal tuberculosis. *J Postgraduate of Medical Institute*. 2003 ; Vol.17 (1) : 32-41.
91. M.P. Sharma & Vikram Bhatia. Abdominal Tuberculosis. *Indian J Med Res* 120, October 2004, pp 305-315.
92. Tariq Sinan, Mehraj Sheikh, Salwa Ramadan, Sukhpal Sahwney and Abdulla Behbehani. CT features in abdominal tuberculosis: 20 years experience. *BMC Medical Imaging*. <http://www.biomedcentral.com/1471-2342/2/3>.
93. Kesarwani RC, Pandey A, Mishra A, Singh A. Polymerase chain reaction (PCR): its comparison with conventional techniques for diagnosis of extra-pulmonary tubercular diseases. *Indian J Surg*. 2004;66:84-88.

# PROFORMA

Name :

Age :

IP No :

Sex :

Address :

Chief Complaints :

## History of presenting complaints

- Abdominal pain
- Vomiting
- Abdominal distension
- Loss of appetite
- Loss of weight
- Low grade fever, night sweats
- Diarrhea / Constipation
- Bleeding per rectum

Past History

h/o DM / HTN / T B/ Ba / Epileptic / Previous surgery

Personal History

Mixed diet / veg.

Alcoholic / smoker

## General Physical examination

- Concious
- Oriented / Disoriented
- Well / Moderately / poorly / Nourished
- Febrile / Afebrile

- Pallor - yes / no
- Icterus – yes / no
- Hydration- adequate / Dehydrated
- Pedal edema – yes / no
- Lymphadenopathy –yes / no

**Systemic Examination:**

**Per Abdomen.**

1. distension
2. soft / rigidity
3. visible intestinal peristalsis
4. tenderness – yes/no
5. abdominal mass - size/ site/ consistency/ mobility.
6. presence of free fluid
7. presence of gurgling
8. bowel sounds – present / absent.

**Respiratory System :**

1. Chest – for any abnormality
2. breath sounds – normal /abnormal
3. any added sounds
4. fluid if present

**Cardiovascular system for heart status**

**Central Nervous System –**

1. higher mental functions
2. spinal tenderness.
3. any focal neurological deficit

**Investigations.**

1. Haemoglobin
2. total and differential white cell count
3. ESR
4. Blood sugar, Urea, Creatinine.
5. Serum Electrolytes.
6. Urine Routine analysis
7. Blood Grouping
8. X-ray Chest
9. Mantoux test.
10. X-ray Abdomen- Erect
11. Ultrasound abdomen/ CT Scan Abdomen
12. Barium meal series
13. colonoscopy