A CLINICO PATHOLOGICAL STUDY AND VARIOUS MODALITIES OF MANAGEMENT OF ABDOMINAL TUBERCULOSIS IN G.R.H. MADURAI

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CERTIFICATE

This is certify that dissertation entitled **"A CLINICO PATHOLOGICAL STUDY AND VARIOUS MODALITIES OF MANAGEMENT OF ABDOMINAL TUBERCULOSIS"** Submitted by **Dr.M.BOOMINATHAN** to the Tamil Nadu Dr. M.G.R Medical University, Chennai, is in partial fulfillment of the requirement for the award of **M.S Degree Branch – I (General Surgery)** and is a bonafide research work carried out by him under direct supervision and guidance.

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DECLARATION

This is consolidated report on **"A CLINICO PATHOLOGICAL STUDY AND VARIOUS MODALITIES OF MANAGEMENT OF ABDOMINAL TUBERCULOSIS"** based on 60 cases treated at Govt. Rajaji Hospital, Madurai, during the period July 2004 to September 2006.

This is submitted to the **Tamilnadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the rules and regulations for the **M.S.** Degree Examination in General Surgery.

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CONTENTS

INTRODUCTION

Tuberculosis is a disease which has affected mankind for many centuries. In spite of considerable advances in prophylaxis and chemotherapy, tuberculosis at various sites, continues to remain a health hazard in our country.

In many countries, especially the developed ones, the situation has progressively improved because of various prevention measures introduced in this century. It has been estimated that every year, about one million people in India die of tuberculosis. Tuberculosis was seen as a common disease associated with the poor in overcrowded conditions. In the developed countries there is a change in pattern with a greater predominant of non-pulmonary tuberculosis.

Abdominal tuberculosis is one of the commonest forms of extra pulmonary tuberculosis. An early reference to probably intestinal tuberculosis was made in 1643 when an autopsy on King Louis XIII show ulcerative intestinal lesions associated with a large pulmonary cavity.

Mycobacterium bovis was previously a common cause of abdominal tuberculosis, but now it has been controlled in many countries by detection and elimination of diseased animals and pasteurization of milk.

Tuberculosis is an important socio-economic problem in our country and it is closely linked with health education, health consciousness and preventive awareness. Because of effective control measures, abdominal tuberculosis has become a rare entity in the west, but it is still more common in our country than other non-specific granuloma.

Although abdominal tuberculosis is quite common, it is still ill understood and is being neglected all too often by clinicians and researches. Although some of the patients may have co-incident pulmonary disease, there is an increased proportion who have intra abdominal disease alone. It is in this group that the diagnostic problems are particularly difficult.

HISTORICAL MILESTONE

Hippocrates 460 BC father of medicine called the tuberculosis as phthisis which means 'to dry up'. He noted intestinal involvement in pulmonary tuberculosis and also its seriousness for he declared Diarrhoea attacking a person with phtisis is mortal symptom (Pimparker 1977).

Manget in 1700 reported that tuberculosis is also found in lived spleen and mesenteric glands described as being the size of millet seeds (**Webb** 1948), later known as miliary tuberculosis.

Lannec 1819 described pulmonary tuberculosis and differentiated from the other chronic lung lesions.

Barkhanson in 1824 reported the first case of gastric tuberculosis **Rokintonsky** in 1855 first described the giant cell which was characterized as a tubercle.

Langhans in1868 described a giant cell in a more detailed manner and now called with his name.

Robert Koch in March 1882 discovered the mycobacterium tuberculosis and in May 1882.

Enrlich for the first time showed the bacilli were acid fast. In the same year, **Ziehl** and modified by **Neelsen**.

Hartman and **Pileit** in1891 published a report on surgery of intestinal tuberculosis. **Hutman** and **Pilliet** published the first report of cases of hyperplastic tuberculosis of the caecum for which operation was performed.

Wilhelm konard bon rotgen in 1895 used radiation for examine the progression of TB and the severity of the illness

Theobald Smith in 1896 differentiated between human and bovis strains of tubercle bacilli (Day 1970).

Von pirquent in 1907 described the tuberculin test.

Calmette and **Guerin** of Pasteur institute in 1921 discovered to B.C.G. vaccine and BCG vaccine was introduced in India 1949.

Liehtheium in 1869 described the tuberculous catcall tumor and 1883 he was the first man to demonstrate the tubercle bacilli in stools.

Carmen (1920) performed the radiolgocial study of barium energy with good results. Fischer in 1923 introduced double contrast enema.

Selmen A waksman in 1943 discovered the antibiotic streptomycin was purified from streptomyces griseus to treat TB

REVIEW OF LITERATURE

The term abdominal tuberculosis should ideally refer to tuberculous disease of intra abdominal organs. In clinical abdominal tuberculosis it is classifies as follows affecting 1. Gastro intestinal tract and biliary system 2. Peritoneum 3. Mesenteric nodes. Gastro intestinal tuberculosis is once considered as a rare disease in U.K. But recently the incidence is increased due to immigrants.

Gastrointestinal tuberculosis is common in India (**S.S.Minhas** 1992 Chuttani 1970). 5-7% of intestinal perforations have been associated with intestinal tuberculosis (**Bhanasali** SK 1967). Obstructive jaundice follow by tuberculosis of pancreas was reported by **Major Chaudhary** 1992. Primary pancreatic tuberculosis was first reported by **JA Allen** in1991 Missouri. Role of vasculities in the natural history of abdominal tuberculosis evaluation by mesenteric angiography was carried by **Piriti Sha** 1991.

Tuberculous abscess of spleen was reported by Narrate 1992. The relation of Tuberculosis in AIDS patients were reported by shaver **RM Brooklyn, Soriano V**, Barcelona 1991. Evaluation of enzyme linked immunosorbant assay using mycobacterium saline extracted antigen for the serodiagnosis of abdominal tuberculosis was carried out by **Bargava** DK 1992. A change in acute phase proteins in-patients with abdominal tuberculosis was studies by **Emmanuel C** 1990. Diagnostic values of Ascitic Adenosine deaminase in Tuberculosis peritonitis was studied by M.**Dwivedi 1990 and Violet** MD 1989 South Africa.

ETIOLOGY

The causative organism, mycobacterium tuberculosis was discovered by Koch 1882. It is a rod shaped, non-sporing, gram positive, acid fast organism. There are many types 1.Human 2.Bovine 3.Avial 4. Urine 5.Cold blooded. But only human and bovine strains are known to produce lesions in human being.

Intestinal tuberculosis was thought to be the result of infection with bovine strain in USA and UK when unpasteurised milk was being consumed.

AGE AND SEX INCIDENCE

The disease is more common in young adults and females are more often attached than males (VK Kapoor and L.K.Sharma 1988). In india the disease has a higher incidence in female (Wig 1961 Tandon and Prakash 1972). Many affected females had history of contact with open cases of pulmonary tuberculosis (Chutani 1970).

ROUTES OF INFECTIONS OF GASTROINTESTINAL TUBERCULOSIS

GIT tuberculosis is divided into primary and secondary forms. It is called primary form, when there is no focus of tuberculosis seen any where else in the body and secondary from when there is established lesion found in lungs or elsewhere in the body. There are possible routes.

1. **Direct contact**
    a. Most of the investigators believed that the greater number of intestinal tuberculosis is due to direct contact between the mucosa and contaminated intestinal contents.

b. Consumption of infected milk from tuberculous cattle. In a western countries, or ingestion of infected food from human sputum as a probable cause of primary of intestinal tuberculosis.
c. The serial section studies of involved intestine by Gardner (1928) showed that the initial lesions were at the tip of the lymphoid follicles.

2. Entergenous – direct swallowing of infected sputum (Gardner 1930KL Wig and BNTandon).
3. Hematagenous – blood born infection from a distant focus through mesenteric arteries (patterson 1920 and KLWig BN Tandon).
4. Lymphatic and direct spread.
5. Intestinal involvement can occur from infected female genitalia (cullen JH 1940)
6. Peritoneum is infected from intestinal lesions, female genitalia, mesenteric nodes tuberculosis, and from lungs and pleura through the lymphatics passing through the diaphragm and from bronchial lymph nodes.

ABDOMINAL TUBERCULOSIS AND HUMAN IMMUNO DEFICIENCY VIRAL INFECTION

Patients with AIDS having tuberculosis are considered as opportunities infection which are mostly extra pulmonary forms. They have atypical clinical features and the incidence of abdominal tuberculosis is 18%. Lymph node involvement was most common type.

The diagnosis of tuberculosis was difficult, often delayed they have high fever, decrease in tuberculin activity and atypical chest radiographic pattern. Acid fast bacilli demonstration by biopsy and aspiration from visceral lymph nodes, liver and bone marrow provide the highest

immediate diagnostic yields with rate between 50-90% (shatter RW 1991).

The increased incidence of extra pulmonary military tuberculosis in HIV patients were reported by Feinberg BB 1992.

HIV infected patients had abdominal pain, weight loss and fever are the most frequent physical findings (Guth AA 1991).

Major abdominal procedures in patients with AIDS should not be withheld due to fear of excessive morbidity and morality. Appropriate management requires recognition of wide range of surgical pathology and attention to details of safe intra operative conduct (Deziel DJ 199).

Formation of tuberculous abscesses is a common complication in tuberculosis patients with AIDS (Lupatkin 1992).

CLINICAL FEATURES

According to Bhansali 1968 an acute or chronic intestinal obstruction is the commonest mode of presentation.

Due to vide variation in the clinical profile the correct diagnosis is possible only in half of the cases, the early adulthood 11-20 years is the commonest age of involvement and between 21-30 years is the most affected group.

Females are affected more than male (Shukla HS 1978 Das P1976). But Hoon report higher incidence among males.

The sex incidence also varies according to site of involvement with ration 8.5:1 female – male hyperplastic ileocaecal tuberculosis and for chronic miliary peritonitis is 1.5:1.

Symptoms

Das and Shukla 1976 classified the presentation as obstructive and non obstructive.

General symptoms include fever 65%, anorexia, weight loss 63% weakness, night sweating, chronic cough and haemoptysis (Bilbao 1992).

Abdominal pain is the specific symptoms in 65% (Bilbao 1992) which may be colicky in nature in obstructed cases and may be dull aching chronic cases, it may increase after meals and relieved after vomiting or after passage of flatus or faeces (Das P 1976 Banerjee 1950).

Loud borborgymi can be heard some times.

Vomiting is the second common symptom. Constipation may occur in 20-45% (Das P 1976).

Diarrhoea is also common in abdominal tuberculosis (Hoon JR 1950).

Distress after meal is common and it may contribute to anorexia (Bhansali 1968).

Intestinal bleeding and internal or external fistulae are rare.

An umbilical fistula may occur (Das P1976). Tuberculous fistula ano can occur (Shukla HS 1988).

PATHOLOGY

Abdominal tuberculosis may involve the gastrointestinal tract, peritoneum and mesenteric lymph nodes. The commonest sites of involvement are terminal ileum and ileocaecal regions followed by jejunum and colon (VK Kapoor LK Sharma K 1998). Approximately 85% of the lesions are located in the ileocaecal region (Boyd 1961).

The focus of infection to occur is in areas of

1. Increased physiological stasis.
2. Regions with more lymphoid tissues
3. Areas of increased rate of absorption
4. In areas where the small bowel contents are more completely digested, permitting closer contact with acid fast bacilli with the mucuosal surface.

Common sites involved is terminal ileum where slow peristalsis favours a prolonged period of close contact between infected gut contents and ileal mucosa (HS Shukla 1986).

The bacilli become localised in the depths of mucosal glands and initiates an inflammatory reaction, the bacilli is then carried from the epithelial layer by phagocytes to submucosa where the initial characteristic lesions form in the lymph follicles of peyers patches.

As a result of mucosal swelling and mucosal end-arteritis leading to mucosal oedema, sloughing ensues and ragged ulcer is formed which is swallow with undermined edges, the base of the ulcer may be formed by the submucosa, the muscularis or the serosa. In the large bowel followed by crypt abscess the ulcer is shallower and seldom penetrates the muscularis layer. Small tubercles may be seen on the serous coat or they may be covered by plastic exudates.

In the process of ulcer healing there is accumulation of collagenous tissue which subsequently contracts and may causing intestinal narrowing. The tubercle bacilli are carried along the lymph channels to invade the mesenteric nodes. These nodes show complete the range of changes from hyperplasia to caseation necrosis and calcification producing the pathological lesion called the “Tabes mesenterica”.

In the end stage lymphatic obstruction results and eventually the mesentery as well as involved bowel becomes a thick florid tuberculous mass.

HISTOPATHOLOGY

The histopathological pattern in human tuberculosis is apparently the result of relationship between the virulence of the organism and degree of immunity and the allergic reaction of the host. Polymorphonuclear leucocytes are the first to arrive in response to invasion by tuberculous bacilli and this response is much more active if the patient is previously infected.

The congression of the polymorphonuclear cells is short lived and within 24 hours they are replaced by mononuclear phagocytes or histiocytes (Aird 1957) the bacilli as well as the polymorphonuclear cells containing the bacilli are phagocytosed by mononuclear cells which break down the lipid envelop of the bacilli with dispersion of the lipid through the cytoplasm. This result in the transformation of the mononuclear cells into epitheloid cells which is the most characteristic single feature in tuberculous reaction (Boyd 1961).

Giant cells are formed by the fusion of epitheloid cells and they may attain a larger size and contain larger number of nuclei usually arranged either in periphery or at one or both pole these are typical langhan giant cells. By the end of a week lymphocytes appear and form a ring

around the periphery of the lesion.

Small mass of newly formed cells constitute tiny transulucent nodules visible to naked eye and are called tubercles. It may disappear or it may be surrounded first by fibroblasts and they fibrous capsule in which calcium may be deposited, such a tubercle is called “healed”. But tubercle bacilli may be alive within it and may be awakened to activity later.

If the virulence is low or resistance is high there will be little casceation but a liberal formation of granulation tissue results in hyperplastic or hypertrophic lesions are seen in ileocaecal area (Tandon 1981 VK Kapoor LK Sharma 1988). There may be spread of infection through the tissues, tubercles fusing to give large areas of casceation. If the infection is virulent there may be violent acute inflammatory reaction and an early fatal issue.

Histopathological studies of mesenteric nodes have become very important since the description of regional enteritis by Crohn 1932. Because marked morphological and histological similarity is seen between hypertrophic tuberculous enteritis and regional enteritis, both are proliferative, subacute or cronic inflammatory condition with lymphoid follicles and hyperplastic germinal centers.

The most significant feature in tuberculous enteritis is casceation necrosis. Casceation necrosis is the only feature in the lymph node thus emphasing the importance of study of mesenteric nodes in the diagnosis of tuberculosis (Paustian 1964).

Tuberculosis of stomach

Tuberculosis of the stomach is rare even in the presence of florid pulmonary tuberculosis and intestinal tuberculosis. The first case was reported in 1824 by Barkhanson.

For the rarity of Gastro duodenal tuberculosis the reasons are given by Morris and

Seaber 1948.

1. Rapid expulsion of the bacilli from the stomach by peristaltic waves.
2. Relative scarcity of lymphoid follicle in the gastric wall.
3. Gastric acidity
4. Presence of intact gastric mucosa
5. Inherent resistant of tissues to infection

The ulcer is usually situated along the lesser curvature on the pyloric end of the stomach with a tendency to appear on the posterior wall. 1 Undermined edges of the ulcer, 2. Serpigenous outline of the ulcer and there may be fistulous openings in the adjacent mucosa, 3. Superficial tubercles 4. Gastric motility is disturbed less by tuberculosis than by carcinoma.

TUBERCULOSIS OF DUODENUM

Extremely rare condition and has been reported as occurring in as few as 0.5% of the patients with abdominal tuberculosis (Feldman 1957). It is reported thirty times rarer than intestinal tuberculosis (Findlay 1980) Gupta 1971, reported a case of duodenal tuberculosis.

Prakash 1978 found duodenal involvement in 2 cases. Duodenal tuberculosis causing obstructive jaundice was reported by Piriti Shah, Ravi Ramakandan in 1991. They found a patient on treatment for dueodenal tuberculosis who developed obstructive jaundice due to benign stricture of the terminal common bile duct and treated by percutaneous trans-hepatic balloon dilatation.

Tuberculosis of duodenum is essentially by intrinsic involvement by ulceration and stricture formation. Extensive compression by enlarged tuberculous lymph nodes. While both of these commonly present with symptoms of gastric outlet obstruction. The ulcerative type

may mimic symptoms of peptic ulcer (Gupta SK 1988).

The obstructive jaundice due to duodenal tuberculosis is rare. However, compression of the CBD leading to jaundice has been seen with enlarged tuberculous lymph node at the porta hepatis and in the peri pancreatic region.

TYPES

Ulcerative type can cause perforation, mimic peptic ulcer perforation (Gupta SK 1988).

Hyper plastic.

Infiltrative and obstructive types. Duodenal tuberculosis is more common in first part or the duodenum and may perforate.

TUBERCULOSIS OF ILEOCAECAL REGION

Ileum and ileo caecal regions are more commonly affected.

PATHOLOGY

Three types of lesions were described. 1. Hyperplastic 2. Ulcerative, 3. Fibrotic

Tuberculosis is the common cause of stenotic intestinal lesions in India (Tandon 1981) Ulcertive lesion is characterised by multiple ulcers, the long axis of the ulcer lying transversely in the direction of lymphatic flow. So they are horizontally placed. The ulcer may extends to mucosa, musclaris propria of the serosa, adhesions of the involved segment of the bowel precedes ulceration and therefore perforations either confined or fistula develop. An associated

end arteritis lessen the chance of serious haemorrhage (Boyd 1964) since the ulcer is transversely placed in the direction of lymphatic flow, subsequent fibrosis produce stricture which may be enhanced by Antitituberculous treatment. The affected segment of the bowel will be indurated, mesenteric fat may be increased, nodules containing casceation will be present and adhesions occur. The related mesenteric nodes will be enlarged. tuberculous ulcer may be solitary or multiple or circumferential, or irregular and may be longitudinal (Hoon JR 1950).

HYPERPLASTIC TYPE

More commonly seen in ileocaecal region according to Sharma the incidence of caecal tuberculosis is about 85 to 97.4%. There may be lesion in ascending and transverse colon either in continuity or in skip lesions.

The contributory factors are:

1. Degree of virulence
2. Volume of infection
3. Host resistance
4. Fibrous tissue reaction on healing. The mesenteric fat increased lymph node enlarged, Gut wall thickened up to 3 cm, mucosa shows pseudo polyposis, cobble stone appearance and furrows.

HISTOPATHOLOGY

Lymphoid follicular overgrowth, hyperplastic germinal centers and infiltration of bowel wall by eosinophils lymphocytes, plasma cells monocytes, neutrophils and giant cells. Caseastion is not always seen in the gut often seen in the mesenteric nodes.

TUBERCULOMA OF INTESTINE

Bacterial granuloma, mycobacterium tuberculosis resembles Benign neoplasm, slow progression, low recurrence rate, tendency to calcify microscopically conglomeration of tubercles in patients with high resistant to the organism appear as mass lesion, can produce intestinal obstruction, the mass projecting into the lumen.

TUBERCULOSIS OF APPENDIX

Appendix is frequently found to be involve secondary to ileocaecal tuberculosis. However tuberculosis of appendix alone is rare (Parkin an Robinson 1964, Bobrow and Friedman 1956, Bansali 1978). Three presentation. 1. A chronic disease with low grade intermittent pain and occasional vomiting and diarrhoea. Examination will reveal tenderness guarding and occasionally mass. 2. Acute case indistinguishable from acute appendicitis. 3. Incidentally found on microscopic examination (find la 1980).

TUBERCULOSIS OF HEPATOBILIARY SYSTEM

Virchow described miliary tuberculosis in liver in the autopsy material obtained from patients dying of tuberculosis. The incidence of hepatic involvement in pulmonary tuberculosis was described by (Saphir 1929). Gupta and Thomas 1967 reported and incident of 2.8% for granulomatous lesions of which 68% had evidence of tuberculosis. Tuberculosis in liver may be seen in the presence of pleural effusion (sarin 1957) Liver function tests may occasionally reveals abnormalities even in the absence of hepatomegaly or any other clinical evidences.

Clinically hepatomegaly or hepatosplenomegaly, are seen associated with miliary tuberculosis or intestinal tuberculosis. Hepatic tuberculosis is usually associated with elevated alkaline phosphatase, hypergammaglobulinemia.

TUBERCULOSIS OF GALL BLADDER

Tuberculosis of gallbladder is rare. This is diagnosed by histologial examination after cholecystectomy. Less than 60cases are reported so far in the world. The gall bladder infection by miliary tuberculosis in children or adult or disseminated tuberculosis of biliary tract. Isolated case of gall bladder in uremia because of immunological energy favoured the infection (piper C 1987).

TUBERCULOSIS OF SPLEEN

Can be seen as splenomegaly or associated with hepatosplenomegaly. The usual spread is haematogenous. Pedro Botet in 1991 report that splenic tuberculosis in three patients with AIDS, Enlarged, Nodular and multiple hypoehoeic area in ultrasonography, hypodense area in CT scan.

Naresh K 1992 in India reported a tuberculous abscess of spleen, multiple abscess cavity containing caseous material, histology showed casseation and acid fast bacilli. Traumatic rupture of a caseous spleen was encountered by summer in 1978.

Ultrasonography showed multiple hypoehoeic area in relation to tail of pancreas. CT scan showed pancreatitis with psedocyst, CBD dilation and of pancreatic duct. There were large retroduodenal lymph nodes which were confirmed as tuberculous adenitis, after laprotomy. The genesis of tuberculous lesion in head and tail of pancreas are due to patchy tuberculous lesion of pancreatic duct which inturn cause accumulation of pancreatic secretion and debris and granuloma, of pancreatic parenchyma could have produced enlarged cyst with obstructive jaundice.

COLONIC TUBERCULOSIS

Tuberculous colitis is a granulomatous disease that involves all layers of the bowel, producing deep ulcers which may progress to form fistula or stricture formation. It occurs in two forms 1. Ulcerative 2. Hyperplastic Ahcya 1976. Ulcerated lesion produce stricture similar to carcinoma of the colon. Tuberculous peritonitis is classified as acute and chronic.

TUBERCULOUS PERITONEUM

Tuberculous peritonitis may be due to i) Generalised miliary tuberculosis ii) Rupture of caseous mesenteric nodes iii) Directly from the fallopian tubes. Tuberculous peritonitis is classified as acute and chronic.

ACUTE

Rare condition, peritoneal cavity is filled with straw coloured fluid and peritoneum studded with tubercules.

CHRONIC

1. **Ascitic** –onset is insidious, peritoneum studded with tubercles, transverse mass rolled up omentum palpable, fluid is pale yellow, specitic gravity 1.020 containing lymphocytes, culture may be positive.
2. **Encysted –** Loculated form, one part of the abdominal cavity alone involved. Later intestinal obstruction may occur.
3. **Fibrous or plastic form –** Produce widespread adhesion, coils of intestines are matted together and distended, may produce blind loops.

4. **Purulent form –** They are rare usually secondary to tuberculous salphingitis may produce cold abscess.

TUBERCULOUS MESENTERIC ADENITIS

Is more common in childhood and adolescence. It is involved by tubercle bacilli by way of peyers patches, they may get matted together production so called Tabes mesenterica. They may caseate of appear a lump in the abdomen, produce intestinal obstruction due to adhesions pressure symptoms in ileocaecal or pyloroduodenal junctions or the first part of duodenum (Gupta 1977 Bhanasali 1978). Rupture of casceating node may produce peritonitis and present as emergency (Bhansali 1978).

COMPLICATION OF INTESTINAL TUBERCULOUS

1. Intestinal obstruction

It may be due to (a) fibrosis and stricture following ulcerative lesions, (b) encroachment on the lumen by hyperplastic lesions, (c) followed by the effect of treatment of intestinal lesions, (d) Pressure of the enlarged node on the fixed portion of the intestine, (e) during retraction of mesentery following healing process, (f) peritoneal band, adhesions and plastic form, (g) Kinking at the ileocaecal angle which accompanies the cephaled displacement of the caecum and change in the angle of entery of ileum into caecum (Foreshortening of caecum).

2. Perforation of the bowel is uncommon because of fibrous adhesion and in duration of base of the ulcer perforation can occur mainly in ileum perforation of ileal strictures also reported 30% by Ashon 1990.

3. Malabsorption: causes

1. Intestinal hurry
2. Decreased effective mucosal surface
3. Fistula formation
4. Blind loops
5. Due to toxic effect of tuberculosis itself

4. Fistula formation occur between adjacent loops of bowel

5. Intussuception may occur but it is a rare complication

6. Massive bleeding per rectum is rare but now recently cases are reported in a case of tuberculosis of transverse colon.

7. Pressure on the duodeno jejunal flexure by tuberculous lymph node may produce **Duodenal ileus.**

8. Tuberculosis of head of pancreas cause **obstructive jaundice.**

9. Duodenal tuberculosis may case **stricture of CBD** and produce **obstructive jaundice.**

INVESTIGATIONS

HAEMATOLOGICAL TEST

ERYTHROCYTE SEDIMENTATION RATE

E.S.R. is a non-specific reaction like leukocytosis and is a general manifestation of disease or tissue destruction. It is therefore, not of specific diagnostic value in tuberculosis. The rate reflects only the degree of activity of the infectious process. It is an useful prognostic test.

BLOOD - Hb

Patients with abdominal tuberculosis may present with evidence of anaemia, which may be iron deficiency type or of non-specific normocytic normochromic pattern.

LEUCOCYTE COUNT

An average count of 8000 to 10,000 leucocytes with moderate rise of lymphocytic count may be observed.

SPUTUM

Sputum for tuberculous bacilli examination is significant review of aetiological relationship of swallowed positive sputum to intestinal tuberculous.

RADIOLOGICAL INVESTIGATIONS

Plain X-ray abdomen may show inclusive evidence of the lesion like lymphonode calcification. Fluid level, and abnormal gas shadows.

BARIUM STUDIES

Barium meal, barium enema and gastrografin studies show the following one or more findings made out like ulcer, irregularity, spasm, mottling, mucosal oedema, defect of motility. It is very difficult to differentiate crohn's disease and intestinal tuberculosis by radiological examination.

ASCITIC FLUID EXAMINATION

It is essentially exudates which is straw coloured, contain protein more than 2.5 grams% and lymhocyte count more than 1000cell/mm^3. Some times cells more than 250/mm^3 are taken as positive. Now much more recently (Shukla 1982) cells more than 100/mm^3 is taken as positive.

HISTOLOGICAL EXAMINATION

100% accuracy in needle biopsy of peritoneum (Levine 1968). Needle biopsy of liver shows casceation necrosis in 2-3% as reported by Das P 1973.

Culture of AFB in Lowenstein Jenson medium takes about 6-8 weeks.

RECENT INVESTIGATIONS

1. ***ENZYME LINKED IMMUNOSORBANT ASSAY***

   ELISA level of absorbance more than 0.7 serum suggest tuberculosis with a sensitivity of 81% specificity 88% and diagnostic accuracy in 84%. This is used to diagnose the abdominal TB from other non tuberculous lesions (Bhargave DK 1992).

2. ***ACUTE PHASE PROTEINS IN ABDOMINAL TUBERCULOSIS***

There may be decrease in the C-reactive protein, ceruloplasmin, heptaglobin, Alpha 1 and glycoprotein. There may be increase in concentration of transfer in (Immanuel 1990). Further evaluation has to be done in the study of diagnostic value of Ascitic adenosine, deaminase in TB peritonitis advocated by Voigt MD 1989. The mean activity of adenosine deaminase is more than 36.7 in TB abdomen.

3. ***PCR***

Polymerize chain reaction is a Highly sensitive (92-95%) and specific (90-100%) test.

PCR is based on 123 bp fragment of IS 6110 is specific for the Mycobacterium tuberculosis complex there are many occasions when biopsy material is scanty and even in intestinal resection cases histological evaluation fails to confirm or rule out tuberculosis

It can be performed on any body fluid specimen including sputum blood CSF urine ascitic fluid or even the fixed tissue specimen (paraffin block) after making fresh sections.

4. ULTRASONOGRAPHY

May be useful in making diagnosis is abdominal tuberculosis with enlarged lymphnodes in pancreatic, mesenteric, perivascular, and hepatic pedicle area (Beliaiche 1986). Hypoechoeic area in splenic tuberculosis (Pedro Botet 1991).

Mass lesion and omental adhesion and ultrasonographic guided needle biopsy may give a compliment to the diagnosis.

5. C.T. SCAN

Also useful in diagnosis to fine

1. Diffuse lymphadenopathy
2. Low density lymph node with multilocular appearance following intravenous contrast.
3. High density ascitis
4. Mottled low density masses in the omentum thickening of the Bowel wall adjacent to mesentery
5. Hepatic pseudo tumors

6. ***COLONOSCOPY***

Lesions in the wall of large intestine can be made out early and tissue diagnosis can be made out by colonscopy. Sharma in 1990 reports that the following findings.

1. Deformed ileocaecal valve contracted caecum
2. Ulcer, mucosal nodules and stricture
3. Histopathological examination

7. ***LAPARASCOPY***

Many studies recently show the lesion in the peritoneum. Ascities was detected in 70% cases and intra peritoneal tubercle in31% and adhesion in 22% tubercle with adhesion in 47% and is useful to find hepatomegaly and also for biopsy (Parikh 1990 and Hussain J 1992).

8. MESENTRIC ANGIOGRAPHY

Role of vasculitis in abdominal tuberculosis was diagnosed by mesenteric angiography.

They show that in stricturous lesion, there is occlusion of vasa recta, and peritoneal lesion can be made out by stretching and overcrowding of vessels

TREATMENT

1. General treatment
2. Medical treatment
3. Surgical treatment

1. GENERAL TREATMENT

A. Heliotherapy

The open air treatment in abdominal tuberculosis is valuable and is best carried out in hospitals and Sanitoria. The patient must be nursed in an air room with wide and open windows and plenty of sunlight. Heliotherapy was first used by Roller in 1903. Natural and artificial Heliotherapy was used in graduated dosages offered in important aid in its management (Goldberg, Moyer, Tuber 1935). As symptoms improve with increase in weight and improvement in pulse rate, temperature, E.S.R. returning to normal a gradual increase of patient's activity depending upon the severity of the disease vigorous treatment may need to be continued for several years after resuscitation of normal life.

B. Diet

i. Highly nutritious, fully digestible with low residue diet so as to lessen the stimulation and mechanical trauma to the bowel is recommended (Armstrong).

Medical treatment

In the absence of acute obstruction and complications a conservative medical treatment is recommended.

Now according to RNTCP (Revised National Tuberculosis Control Programme) short term therapy is commonly used for 6-9 months.

Medical treatment should be continued whatever the surgical procedures were contemplated.

RNTCP categories and treatment

There are 3 categories. Patient those who are having extra pulmonary TB and are not seriously ill are categorized under Category III patient those who are having extra pulmonary TB and seriously ill are categorized under Category I.

CATEGORY III

FIRST 2 MONTHS

Rifampicin	450mg	3 doses in a week (alternate days)
Isoniazine	600mg	for 8 weeks (24 doses)
Pyrizinamide	1500mg	

Followed by

NEXT 4 MONTHS

Rifampicin	450mg	3 doses in a week
Isoniazide	600mg	for 18 weeks (54 doses)

CATEGORY I

FIRST 2 MONTHS

Rifampicin	450 mg	3 doses in a week
Isoniazide	600 mg	(alternate days)
Pyrazinamide	1500 mg	for 8 weeks (24 doses)
Ethambutol	1200 mg	

Followed by

NEXT 4 MONTHS

Rifampicin	450mg	3 doses in a week
Isoniazide	600mg	(alternate days)
		For 18 weeks (54 doses)

SURGICAL TREATMENT

It is varied the emphasis is to conserve as much tissue as possible, to avoid blind loops and drain any abscess. Extensive resection for mesenteric lymph node tuberculosis is not recommended. In emergency simple closure of perforation and bypass, to circumvent stricture is adequate, even at the cost of blind loop syndrome and recurrent obstruction at a later date.

The principal indications for surgery are the

1. Management of complications of tubercular disease and

2. As a diagnostic procedure

The list of following indications for surgical treatment

17. Free perforation of a tuberculosis ulcer.

18. Perforation with localised abscess formation.

19. Obstruction by ciccatrical stenosis of the bowel or shortening of the mesentery resulting in kicking of the bowel / gangreneous bowel.

20. Localised tuberculosis involvement of hypertrophic type resulting in marked diminution of the caliber of the lumen.

21. Gastrointestinal haemorrhage

22. Tuberculous illeo-caecal mass with entero cutaneous fistula.

Intestinal stricture is best treated with stricturoplasty and it is done in ileocaecal region also without damaging ileocaecal valve and in perforation just proximal to the stricture (S.S. Minyhas 1992 Chuttani 1970 Bhansali 1967).

In multiple strictures which are situated close to each other, it is better to resect and continuity to be resorted.

Tuberculous ileal perforation is treated by primary perforation closure.

Ileocaecal tuberculosis are treated are treated Right hemicolectomy followed by Ileotransverse anastomosis.

Anti Tuberculous Treatment

It is better to stress once again that Anti Tuberculous treatment to be continued whatever the surgical procedures were contemplated.

AIM OF THE STUDY

1. To find out Age and sex incidence of abdominal tuberculosis

2. To study of various clinical presentation of abdominal tuberculosis.

3. To find out various sites and commonest sits of tuberculous lesion is the abdomen.

4. To find out appropriate indication and various modalities of treatment in tuberculous abdomen.

MATERIALS AND METHODS

MATERIAL

This study consist of 60 cases of abdominal tuberculosis, which were treated in various surgical units in Government Rajaji Hospital, Madurai. This study does not include the patients in paediatric age group and open pulmonary tuberculosis who were treated in T.B. ward. The period of study was between July 2004 to September 2006.

METHOD

All patients who were diagnosed as abdominal tuberculosis, a detailed clinical history, through clinical examination and previous history of treatment for tuberculosis were taken, relevant laboratory and radiological investigations were done. When operated histopathological examination of specimens were carried out. The criteria used to establish the diagnosis were,

1. Symptomatology, radiological evidences of tubercular lesion in GIT with histopathological evidence of tuberculosis in lymph nodes in other site of the body.

2. Pulmonary tuberculosis with GIT problems
3. Patients with abdominal tuberculosis with previous history of typical scar or evidences of sinuses.

4. Histopathological examination of operated specimens.

5. Colonoscopic examination and biopsy

6. Diagnostic lap – for patient with peritoneal and or lymph node tuberculosis

PROFORMA

Case No.:		I.P. No.:
Name :	Occupation:	D.O.A.:
Age:	Religion:	D.O.D.:
Sex:	Wt. OA:	
Address:	Wt. OD:	

1. DIAGNOSIS:

2. COMPLAINTS:

3. HISTORY OF PRESENT ILLNESS:

a. Pain abdomen Present / Absent

- Present attack from
- Previous attack from
- Duration of attack
- Type of pain
    - Dull aching / colickly
    - Discomfort / Non specific
- Site
- Onset of Pain: Sudden / Insidious
- Radiation: Present / Absent

Back / Any other part

- Relation to food: Relieves / Aggravates

  No change

- Relation to bowel movements

b. Vomiting Present / Absent

- Duration - Frequency - Projectile
- Spontaneous - Nature - Contents
- Amount - Relation to food - Hemetemesis
- Relation to pain

c. Cough Duration - Expectoration

d. Haemoptysis Present / Absent

- Duration - Nature - Quantity
- Fresh - Altered blood

e. Bowel movement - Increased / Decreased / No change

1. Diarrhoea - Duration - Nature
   - Frequency - Blood & mucus
   - Malena Present / Absent
     - Frequency
     - Quantity

2. Constipation – Present / Absent
   - Duration
3. Alternate diarrhoea & constipation - Present / Absent

f. Appetite - Increased / Decreased / No change

g. Fever - Present / Absent

Mild

- Duration -Nature - Severity - Moderate

- Chills & Rigors

- Evening raise of temps - Present / Absent

h. Loss of weight - Decreased / No change

By Kgs or %

i. Distension of abdomen

j. Moving mass in the abdomen

k. Borborygnii

l. Post prandial distress

m. Mass in abdomen - Duration progress Site

n. Urinary symptoms

4. History of previous illness

- Similar complaints before – Present / Absent
- H/O having suffered from pulmonary tuberculosis
- Drug history: taken treatment or not taken
- Patient undergone any operation or not: Yes / No
- Details of operation if yes.

5. Personal history -Diet: Veg/Mixed

- Habits : Regular / Occasional / Never

- Tobacco: Chewing / smoking –Beedies, Cigarettes
- Appetite Good / Decreased
- Bowel movements Normal / Increased / Constipated
- Micturation
- Obestetric history
- Menstrual history

6. Family History Present / Absent

- Any number of the family suffered from pulmonary tuberculosis
- Marital status

7. Socio Economic history

8. Physical Examination

General examination

a. Built & Nourishment	b. Pedeal oedema or anasarca dehydrations
c. Lymphadenopathy	d. Skin manifestation of T.B.
e. Anaemia / Jaundice	f. Pulse
g. Respiration	h. B.P.
i. Temperature	j. Any others

9. Local Examination

a. Abdomen	- Inspection	Small bowel V.P.	+/-
		Distended abdomen	+/-
		Mass abdomen	+/-
	- Palpation	Mass abdomen	

Doughly abdomen

Liver & Spleen

Ascites

Hernial orifice

b. P.V. & P.R. examination

10. Systemic Examination

Cardiovascular system

Respiratory system

11. Provisional Diagnosis

12. Differential Diagnosis

13. Investigation

URINE	BLOOD	STOOL
Albumin	Hb	Ova
Sugar	Tc	Cyst
Deposits	Dc	Cytology
	ESR	

BLOOD: Grouping & cross matching

HIV

ECG

BLOOD

Urea

Ascitic fluid analysis

Cell type

Cell count

Proteins

Sugar Specific gravity

Creatinine

Mantoux test

Sputum for AFB Stool for AFB

X ray chest PA view

Plain X ray Abdomen erect

X ray barium meal Barium enema

Ultrasound

CT

Colonoscopy examination

Laparoscopic examination

14. Final Diagnosis:

15. Management:

1. Emergency management of acute abdomen
2. Planned treatment
3. Surgery – operation notes
4. Post – operative treatment
5. Post – operative complications
6. Follow – up

16. Discussion:

RESULTS OF STUDY

The present clinical analysis has been prepared from the study of 60 cases.

The following Table shows the age and sex incidence

Age group	Male	Female	Total
10-20	4	6	10
21-30	13	8	21
31-40	11	4	15
41-50	6	3	9
51-60	3	-	3
61-70	2	-	2
Total	39	21	60

AGE INCIDENCE

It is found that abdominal tuberculosis is more common in 3rd and 4th decades which constitute 60% of the total cases.

SEX INCIDENCE

There was male dominance as against the female. Out of 60 patients 39 patients were male 21 patients were female.

CLINICAL STUDIES

ACUTE GROUPS

Out of 60 cases, 12 patients were presented with acute symptoms. All patients had constant symptoms of abdominal pain, vomiting, constipation and fever, either alone or in combination of symptoms. Among the 12 cases in addition to abdominal pain 8 patients had both vomiting and constipation and 4 patients had fever.

Local examination revealed tenderness in all 12 cases, guarding and rigidity in 8 cases, distension and visible intestinal peristalsis in 7 cases and lump in 4 cases.

CHRONIC GROUP

48 patients presented as non emergency condition. The following table shows the various clinical signs and symptoms.

ABDOMINAL SIGNS

When we look into abdominal signs alone in abdominal tuberculosis the following findings were noted.

- Abdominal distension in 34 cases
- Abdominal tenderness in 32 cases
- Lump in various quadrants of abdomen 32 cases
- Ascities 14 cases
- Visible intestinal peristalsis 12 cases
- Hepatosplenomegaly in 2 cases

Sl. No.	Symptoms	No. of cases
1	Pain abdomen	42
2	Lump in the abdomen	23
3	Loss of weight	38
4	Loss appetite	38
5	Diarrhoea	18
6	Constipation	16
7	Vomiting	18
8	Respiratory complaint	6
9	Jaundice	2

DISTRIBUTION OF MASSES IN 20 CASES

Right iliac	25 cases
Right lumbar	5 cases
Left iliac fossa	2 cases

INVESTIGATIONS

Hemoglobin %	**No. of cases**
Below 8gm%	4
Below 8-9gm	9
Below 9-10gm	25
More than 10gm	22

Total count / mm^3	**No. of cases**
6000-7000	6
7000-8000	8
8000-9000	24
9000-10000	16
More than 10000	6

Lymphocytes	No. of cases
20-30	16
30-40	28
40-50	12
More than 50	4

ESR	No. of cases
Less than 20mm per hour	12
20-40	15
40-60	14
60-80	8
80-100	6
More than 100	5

HIV	No. of positive case
Reactive	2

RADIOLOGICAL INVESTIGATION

It is decided on the merit of the cases. Barium meal series was done in 7 cases, which revealed strictures in the ileum.

Barium enema was done in 22 cases of leo-caecal tuberculosis which showed filling defect in the caecum and foreshortening in 14 cases.

Ultrosonography was done for all cases and C.T. scan were done in selected cases, also helpful in diagnosis of abdominal tuberculosis. In the 18 cases they revealed, lump in 16 cases in right iliac fossa and hepatosplenomegaly in 2 cases.

Based upon the investigations, the diagnosis is confirmed as follows:

Barium meal series	7 cases
Barium enema	14 cases
History of previous Anti Tuberculous Treatment	11 cases
Colonoscopy & Biopsy	10 cases
Mesenteric node biopsy	16 cases
Biopsy from the ileal perforation	2 cases

DISTRIBUTION OF LESIONS

Site	**No. of cases**
Ileum	7
Ileocaecal region	14
Peritoneal and omentum	20
Ascitis with lymphnodes	14

MANAGEMENT

Patient who presented with obstructive signs and symptoms, peritonitis, lump in the abdomen with malabsorption were operated.

Out of these 60 cases, 19 cases were not operated because they were non obstructive, 41 cases were operated. Among these 12 cases as emergency and 29 cases as elective.

In the emergency the following surgical procedures were contemplated.

1. Adhesion release - 8 cases
2. Limited resection - 2 cases
3. Perforation closure for Ileal perforation - 2 cases

Among the 29 patients taken up for elective surgery the following surgical procedure was done.

Sl. No.	Indication	Type of operation	No. of cases
1	Ileal stricture	A. Stricturoplasty	3
2	Ileo caecal tuberculosis involving ascending colon	Right hemicolectomy followed by ileo transverse anastomosis	14
3	Tuberculous mesenteric lymph adenitis with adhesions	A. Diagnostic Lap – Adhesiolysis (release of adhesions) and mesenteric node biopsy B. Adhesiolysis – (lapratomy) release of adhesions and mesenteric node biopsy	6 6
Total no. of elective cases			**29**

FOLLOW UP

Cases taken up for surgery and non operated cases were taken follow up for a period of 3 months to 18months. Among these 26 patients completed the medical treatment for 9 months and were symptom free. Cases still under treatment are 14. Cases lost to follow up 20 cases.

MORTALITY - Nil

SUMMARY

INTRODUCTION

According to Shukla 1988 the incidence of abdominal tuberculosis is high in India. But recent literature indicates that the disease is also prevalent all over the world (Chen ws Taiwan 1992). Shafer RW New York reports those 47 cases in 1983 and 113 cases in 1988. In Government Rajaji Hospital, Madurai, Tamilnadu over 24 months of study, 60 cases are reported in various surgical units and treated as inpatients. In United Kingdom the frequency has recently increased due to arrival of Asian immigrants (VK Kapoor and L.K. Sharma 1988).

MODE OF INFECTION

According to Medlar EW 1964, Shukla 1978 the main source of infection is swallowed sputum. But in our study in 60cses only 6 cases are having pulmonary tuberculosis. These low incidences may probably due to exclusion of open pulmonary tuberculosis in this study.

According to Cullen J.H.1940 spread can occur in miliary tuberculosis, 2 cases had hepatosplenomegaly and multiple lymphadenopathy. Intestinal TB spread to the peritoneum and mesenteric lymph nodes.

LESION OF ABDOMINAL TUBERCULOSIS

Ulcerative form or hyperplastic forms are common (Tandon HD 1981). In our study, right iliac fossa mass-hyperplastic ileocaecal TB in14 cases, 5 cases of ileal stricture, 2 cases of ileal perforations, Tuberculous mesenteric lymphadenopathy with adhesion in 20 cases. Tuberculosis ascities in 14 cases (Hussain J 1992).

CLINICAL FEATURES

According to Das P 1976 early adulthood tuberculosis between the age of 11-30 is more common and 21-30 years being the most affected group. In our study between the age of 20-30 total number of cases are 35%.

SEX INCIDENCE

According to Shukla 1978 females are more affected than male. In our study 39 cases are male and 21 cases are female. 65% and 35% respectively.

According to Das P, Shukla HS 1975 the incidence of associated pulmonary tuberculosis has diminished from 77% to 15%. In our study only 6 cases had pulmonary tuberculosis which were not open cases (10%).

In tuberculous peritonitis the concomitant pulmonary tuberculosis is found about 50%. In our study of 14 cases having ascitis, only 6 cases are having pulmonary tuberculosis (Das P, Shukla HS 1975).

SYMPTOMS

Colicky abdominal pain in obstructed cases is about 50% (Bhansali 1968, Das P 1964). In our study of 60 cases there were 12 emergency patients, all having pain abdomen 100%. In

non emergency group 44 cases out of 48 patients were having abdominal pain 92%.

VOMITING

Vomiting in 10 cases of acute group and in chronic group 8 cases had vomiting.

CONSTIPATION

According to Shukla HS, Das P 1975 the incidence is 20-45%, in our study 10 cases of acute group and 8 cases in chronic group had constipation.

PHYSICAL SIGNS

According to Das P, Shukla HS 1978 the incidence of abdominal distension was about 2-58%. In our study 35 cases were having abdominal distension 58%.

The incidence of visible intestinal peristalsis may vary (Bokhus HL 1964). In our study 12 cases were having VIP 20%.

According to Bhansali 1968, Pimparkar 1967 ascitis is rare in intestinal tuberculosis but common in tuberculous peritonitis. In our study about 14 cases were having ascitis 23%.

COMPLICATIONS

According to Anand SS, 1956, Bhansali, 1968 the incidence of intestinal obstruction is 12.5-60%. In our study, 29 cases presented with intestinal obstruction 48.3%.

Peritonitis can occur (Bokhus 1964). In our study 2 cases presented as Perforative peritonitis.

INVESTIGATIONS

BLOOD EXAMINATION

ESR raised and moderate anemia found by Prakash 1970. In our cases, ESR raised more than 20mm per hour in 48 cases 80%. Anemia in 38 cases 63.3%.

HISTOPATHOLOGICAL EXAMINATION

Total no. of surgery done	Specimen sent for exam	Histopathological positive cases	Non specific lesion
41	40	38	2

Feature suggestive of caseating granuloma in 38 cases.

Feature suggestive of non specific inflammation in 2 cases.

According to Das P 1973, the confirmation by needle biopsy is 2- 3%. According to Mehrotra MP 1966, Jain SC 1982 peritoneal biopsy prove 24-42% accuracy.

According to Mehrotra MP 1966, Levome 1968 ascitic fluid aspiration cytology were helpful in 7-36%. In our 14 cases only 6 cases showed positive results.

The recently advocated investigations like 1.Estimation of ascitic adenosine deaminase estimation in ascitic fluid by Voigt MD 1989, Dwivedi 1990.

Enzyme linked immunosorbant assay by using saline extract tuberculous antigen by Bargava DK 1992.

The usefulness of mesenteric artery angiography in diagnosing vasculitis in abdominal tuberculosis by Piriti Sha, Ravi Rama Kandan, 1991 has to be evaluated in future.

Colonoscopy examination and biopsy was taken for 10 cases which showed positive results.

TREATMENT

Medical Treatment; Rifampicin, INH, Pyrazinamble combination are given to our patients for 6 months whatever the surgical procedures were done.

SURGICAL PROCEDURE

Sl. No.	Indication	Type of operation	No. of cases
1	Ileal stricture	B. Stricturoplasty	3
2	Ileo caecal tuberculosis involving ascending colon	Right hemicolectomy followed by ileo transverse anastomosis	14
3	Tuberculous mesenteric lymph adenitis with adhesions	A. Diagnostic Lap – Adhesiolysis (release of adhesions) and mesenteric node biopsy B. Adhesiolysis- (lapratomy) release of adhesions and mesenteric node biopsy	6 6
Total no. of elective cases			**29**

In the emergency the following surgical procedures were contemplated.

1. Adhesion Release 8 cases

2. Limited resection 2 cases

3. Ileal perforation - 2 cases
   Perforation closure

Hence elective surgeries done in 29 cases, emergency surgery were done in 12 cases and total cases are 41.

19 cases were managed conservatively.

MORTALITY Post operative mortality; Nil.

CONCLUSION

1. 60 cases of abdominal tuberculosis involving the various organs were discussed.
2. The maximum incidence was noted in 3rd and 4th decades. But according to study report early adulthood 11-20 year is the commonest age of involvement and between the ages of 21-30 year is the most affected group.
3. In our study males are more affected than female. But according to study reports (Shukla HS 1978 Das P1976) Female are more often affected than male.
4. The commonest site of lesion is Ileocaecal region.
5. Only 20% of the total cases were presented as emergency condition.
6. 32% of cases were treated with Anti Tuberculous Drugs only.
7. 3 patients had stricturoplasty
8. 2 patients had Ileal perforations for that perforation closure was done.
9. As far as possible conservative surgery was contemplated.
10. 10% of patients associated with active pulmonary tuberculosis.
11. 2 patients associated with HIV.
12. 2 patients had ATT induced hepatitis (jaundice).
13. The mortality in the series in NIL.

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