Review Article

Abdominal Tuberculosis

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Abstract

Abdominal tuberculosis constitute up to 12% of extrapulmonary TB and is sixth frequent site of extrapulmonary involvement. The most common sites of involvement is the ileocaecalregion. Other site of involvement in descending order are ascending colon jejunum, appendix, duodenum, stomach, esophagus, sigmoid colon and rectum. Abdominal TB has diagnostic dilemma due to its diverse and non-specific clinical presentation and has no single most specific, sensitive diagnostic test. A high index of suspicion, common and rare clinical feature, adequate imaging study, endoscopy, enteroscopy, laparoscopy, laparotomy, biopsy with histopathology, Mycobacterial isolation, Quantiferon-TB Gold, GeneXpert Assay, MULTIPLEX PCR and clinical response to anti TB therapy are considered for early diagnosis to reduce morbidity and mortality. Six month antiTB regime is effective as nine or 12month therapy. MDR TB and frequent interruption of therapy should considered in nonresponder to standard therapy. Surgery is required for minority cases that developed complications not responding to medical therpy.

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Introduction

Tuberculosis is a infectious disease which has affected mankind since Neolithic era[1]. It was recognized and described by Hippocrates in his book I of the epidemics, as consumption, phthisis(waste away). Scrofula, Pott's disease, and the White Plague are other terms used to refer to tuberculosis and thought as hereditary disease. Aristotle disagreed, believing the disease was contagious. Abdominal tuberculosis usually occur in following four forms depending on the affected parts(in descending incidence) nodal, peritoneal, gastrointestinal and visceral or solid organ excluding genitourinary system[2]. The gastrointestinal tract from mouth to anus, peritoneum and pancreatobiliary system may be involved in abdominal TB.

Epidemiology of tuberculosis

Globally, TB incidence has fallen by an average of 1.5% per year since 2000 and is now 18% lower than the level of 2000. From 2016, the goal is to end the global TB epidemic by implementing the End TB Strategy with targets linked to the newly adopted SDGs, the strategy serves to reduce the number of TB deaths by 90% by 2030 (compared with 2015 levels), cut new cases by 80% and ensure that no family is burdened with catastrophic costs due to TB[3].

Rates per100 000 population

BANGLADESH POPULATION (THOUSANDS)a 159 078, MORTALITY 51 (37-68), HIV-POSITIVE-TB-MORTALITY 0.1 (0-0.1), PREVALENCE -404 (211-659), INCIDENCE-227 (200-256), HIV PREVALENCE IN INCIDENT TB CASES (%) 0.2 (0.1-0.2)[4].

Exact epidemiology of extrapulmonary TB in Bangladesh is not known. One retrospective study was done by Rouf HMA in general Hospital, Sirajgonj[5] with intestinal obstruction (25%), and 27% chronic symptoms. Faiz M.A. did another retrospective study in 1989 in IPGM & R on extrapulmonary tuberculosis and found intestinal tuberculosis in 5 cases out of 47 patients having extrapulmonary tuberculosisa[6a]. For Bangladesh, a joint reassessment of estimates of TB disease burden will be undertaken following completion of the national TB prevalence survey.

Pathogenesis

There are various ways tuberculosis can spread into the abdomen and gastrointestinal tract. One route is through ingestion of the tuberculous bacilli. The second Path is from hematogenous spread from a primary lung focus during childhood with subsequent reactivation, another mechanism is through the lymphatics from infected nodes. Lastly, tuberculosis of the gastrointestinal tract can also come from direct spread from adjacent infected structures[7]. Organisms are Mycobacterium tuberculosis and Mycobacterium bovis. Primary intestinal tuberculosis is usually caused by M. bovis and results from ingestion of contaminated milk from infected cows[8]. 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. Abdominal lymph nodal and peritoneal tuberculosis may occur without gastrointestinal involvement in about one third of the cases[9]. Enlarged

Lymph nodes at the root of the mesentery may cause obstruction to the third part of the duodenum. Portal hypertension due to portal vein compression and obstructive jaundice due to compression of the common bile duct due to tuberculous nodes, have been reported.

Pathology

Tuberculous granulomas are initially formed in the mucosa or the Peyer's patches and tend to beconfluent. Tubercular ulcers are relatively superficial and usually do not penetrate beyond the muscularis[10] and are usually transversely oriented. Cicatrical healing of these circumferential 'girdle ulcers' results in strictures. Mesenteric lymph nodes may be enlarged, matted and may caseate. Characteristic granulomas may be seen only in the mesenteric lymph nodes[11]. Hoon[11] et al originally classified the gross morphological appearance of the involved bowel into ulcerative, ulcerohyperplastic and hyperplastic varieties. Ulcerohypertrophic types usually seen at Colon and ileocaecal regions and presents with a right iliac fossa lump.

Tuberculousperitonitis(PT)

Typed with (1) wet type with ascites, (2) encysted (loculated) type with a localized abdominal swelling; and (3) fibrotic type with abdominal masses. Clinically PT may be acute or chronic. The peritoneum is studded with multiple yellow-white tubercles. It is thick and hyperaemic with a loss of its shiny luster. The omentum is also thickened[12].

Disseminated Abdominal Tuberculosis

Involving the gastrointestinal tract, peritoneum, lymph nodes and solid viscera has also been described[13].Chen et al [14] reported disseminated involvement of the abdomen in 21 Out of 60 patients with large bowel tuberculosis, while most of the 96 patients with tuberculous hepatitis reported by Essopet al'[15] had disseminated disease. Multiple Lesions are common. Bhansali[8] reported that small intestinal strictures were multiple in 71 Out of 119 patients.

Hyperplastic tuberculosis

Usually occurs in the ileocaecal region, solitary or multiple lesions in the distal ileum are sometimes seen and early involvement of regional lymph nodes which may caseate.

Clinical presentation

Abdominal tuberculosis is predominantly a disease of young adults. Two-thirds of the patients are 21-40 yr old and the sex incidence is equal, although some Indian studies have suggested a slight female predominance. The clinical presentation of abdominal tuberculosis can be acute, chronic or acute on chronic. Symptoms and signs of tuberculosis are non-specific and protean. Weight loss, Fever (low grade), chronic cough, malaise, anorexia, night sweets are features developed due to cytokines released by activated macrophages. i.e. IL-1, TNF^{a} and not by the organism itself.

Table-I: The clinical presentation depends upon the site and type of involvement[16]

Clinical features

Site	Type	Clinical feature
Small intestine	Ulcerative	Diarrhoea, malabsorption
	Stricturous	Obstruction
Large intestine	Ulcerative	Rectal bleeding
	Hypertrophic	Lump, obstruction
Peritoneal	Ascitic	Pain, distension
	Adhesive	Obstruction
Lymph nodes	-	Lump, obstruction

Table -II: Case series of intestinal tuberculosis[17,18,19]

Symptoms	Mukewar et al	Makharia et al	Khan et al
Abdominal pain	80.6%	90.5%	93%
Weight loss	74.6%	83%	47%
Loss of appetite	62.7%	69.8%	52%
Fever	40.30%	41.5%	46%
Diarrhoea	16.4%	37.7%	12%
Constipation	25%	49%	31%
Bleeding Per rectum	11.9%	16.9%	14%

Esophageal Tuberculosis:

It is rare, constituting about 0.3% of GIT TB. Additonal symptoms are dysphagia, odynophagia, pain at mid third of esophagus, near carina due to proximity to mediastinal lymph node[20].

Gastroduodenal Tuberculosis

About 1%, usually involves antral region, prepyloric area, and fundus and usually non healing ulcer or hypertrophic lesion with or without the gastric outlet obstruction[21,22] or perforation.

Duodenal Tuberculosis

Most common in third part and may be intrinsic (ulcerative, hypertrophic or ulcerohypertrophic) orextrinsic (periduodenal lymph nodes compression from the outside). The largest published series of duodenal tuberculosis reported 30 cases from India[23]; most patients (73%) had symptoms of duodenal obstruction (mostly extrinsic compression). 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[24] fistulae (pyeloduodenal, duodenocutaneous, blind)[25] and obstructive jaundice by compression of the common bile duct. Recently Mohite et al[26] from Mumbai reported a case of duodenal tuberculosis presenting with choledocho-duodenal fistula.

Ileocaecal Tuberculosis

Patient may present with acute tubercular abdomen-(intestinal obstruction - acute or acute on chronic. Peritonitis with or without perforation, Acute mesenteric

lymphadenitis, Acute tubercular appendicitis)[27]. Or As Subacute intestinal obstruction - colicky abdominal pain, distension, vomiting, gurgling, feeling of a ball of wind moving in the abdomen, and visible loops and peristalsis; thats relieved spontaneously after passage of flatus. Abdominal examination may reveal no abnormality or a doughy feeling to a well defined, firm, usually mobile mass palpablein the RLQ 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 aorticor illiac group of nodes are enlarged. Or as complication-small intestinal perforation(5-9% all small intestinal perforation in india)[28] malabsoption[29].

Isolated or segmented colonic tuberculosis

It may (9.2% of abdominal TB) presents with pain, mild hematochezia usually involve sigmoid, ascending and transverse colon[30].

Rectal and Anal tuberculosis

Haematochezia is the most common symptom (88%) followed by constitutional symptom (75%) and constipation (37%).[31] The high frequency of rectal bleeding may be because of mucosal trauma caused by 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.[31] Also presents as stricture, fistula-in-ano[32] or fissure-in-ano. Tubercular fistula-in-ano are multiple.

Investigations

Routine test commonly includes CBC- reveal mild anaemia and increased ESR in 50 to 80% of patients. The white blood count is usually normal, Hypoalbuminemia common. Sputum analysis can reveal associate PTB in less than 20% cases[4]. Chest x-ray found positive for active or healed lesion of 46%, 39%, 25% by Sharma et al, Prakashseries,Tendon et al in a case series of 70,300 abdominal TB[54] respectively. TST positive in 53%[33]. Ascitic fluid examination, X-rays and barium studies, CT scan of Abdomen with contrast, QFT-G, Gene-Xpert MTB RIF assay, PCR for tubercular peritonitis, Diagnostic Laparoscopy for abdominal tuberculosis in peritoneal seedling and extraluminal intestinal TB, Endoscopy for upper Gastrointestinal tuberculosis can be done according to site and specific area. Chest radiographs may show hilar lymphadenopathy or tuberculous lesion in case of concurrent active pulmonary TB. However a normal chest radiograph does not rule out the possibility of abdominal tuberculosis[34]. **Ultrasound:** It is useful for imaging peritoneal tuberculosis. The following features may be seen, usually in combination[35]. i. Free or loculated Intra-abdominal fluid; Pelvic collection may be septated and mimic ovarian cyst. ii. "Club sandwich" or "sliced bread" sign. iii. Lymphadenopathy may be discrete or conglomerated (matted). The echotexture is mixed Heterogenous. Small discrete anechoic areas representing zones of caseation may be seen within the nodes. 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. v. Pseudo kidney sign - involvement of the ileocaecal region which is pulled up to a subhepaticposition. Several radiographic signs (barium meal and enema) have been described for ileocecal TB :**(barium meal)**. 1. Chicken intestine"-hypersegmentation of the contrast column. 2. Hourglass stenosis"-luminal stenosis with smooth but stiff contoursin small intestine **Barium Enema**. 3. "Fleischner" or "inverted umbrella" sign-thickening of the valve or wide gaping of the valve with narrowing of the terminal ileum[36]. 4. "Goose neck deformity"-loss of normal ileocecal angle and dilated terminal ileum, appearing suspended from a retracted, fibrosed caecum. 5. "Stierlin sign"-lack of barium retention in the ileum, caecum and ascending colon, with a normal configured column of barium on either side; narrowing of the terminal ileum with rapid emptying into a shortened, rigid or obliterated caecum.
6. "String sign"-persistent narrowing indicating stenosis of the terminal ileum, sometimes annular, ("napkin ring") or with dilatation of the more proximal ileal segment("purse string") for enema. Both Stierlin and String signs can also be seen in CD and hence are not specific for TB. "Conicalcaecum", due to contraction and fibrosis of mesocolon caecum reduced in size and pulled out of iliac fossa, hepatic flexure is also pulled down.
CT scan: Iliocaecal tuberculosis is usually hyperplastic and well evaluated on CT scan. In the early, slight symmetric circumferential thickening of caecum and terminal ileum. Laterileocaecal valve and adjacent medial wall of the caecum is asymmetrically thickened. In more advanced disease gross wall thickening, adherentloops, large regional nodes and mesenteric thickening can together form a soft tissue mass centered around ileocaecal junction[37]. CT scan can also pick up ulceration or nodularity with in terminal ileum, along with narrowing and proximal dialtaion. Wall thicking, ulceration and narrowing of lumen may involve in other part of small and large bowel. In the colon, involvement around the hepatic flexure is common. Complication of perforation, abscess and obstruction are also common. Tuberculararascitic fluid is of high attenuation value(25-45HU) due to high protein content. Strands of fine septa and debris with in fluid are the characterstics, but are better appreciated on USG. Thicken peritoneum and enhenching peritoneal nodules may be seen. Omental thickening is well seen as an omental cake appearance. CT showed that abdominal tuberculous lymphadenopathy involved predominately the mesenteric, upper and lower para-aortic, periportal, and pancreaticoduodenal regions. **Capsule Endoscopy and Enteroscopy:** There is limited data regarding capsule endoscopy in intestinal TB . Colonoscopy: Shah et al[38] has described the frequency of distribution of colonic TB based on the colonoscopy as follows: 32% disease

confined to the ileocaecal region, 28% ileocaecal and contiguous involvement of variable lengths of the ascending colon, 26% segmental colonic tuberculosis with involvement of the ascending colon in 10%, transverse colon in 12%, and descending colon in 4%; 10% ileocaecal and non-confluent involvement of another part of the colon and in 2% the entire colon was affected. The Colonoscopic findings in various series in patients of GI tuberculosis are high lightened in Table III.[39-40] The main differential diagnosis at endoscopy is Crohn's disease (CD). As use of steroids for a misdiagnosis of CD may have disastrous consequences in patients with TB enteritis. Colonoscopy with retrograde intubation of the ileum is the initial procedure of choice to differentiate. In patients with suspected or proven CD, ileocolonoscopy provided similar sensitivity (67% vs. 83%) but significantly higher specificity (100% vs. 53%) compared to video capsule endoscopy[41]. The diagnostic yield of histology increases with increasing number of biopsies from up to four segments in the colon. Endoscopic biopsies from segments upstream after dilating a stricture, and also from the normal looking ileum, increase the yield in patients with suspected TB.

Table -III : Colonoscopic findings

	Alvareset al	Misra SP et al	SinghVet al	DasHS
Ulceration	70%	92%	83%	47%
Nodularity	56%	88%	79%	42%
Deformed Caecum and IC valve	40%	42%	55%	NA
Strictures	23%	25%	27%	14%
Polypoidlesions	14%	6%	5%	4.7%
Segmental Involvement	19%	22%	19%	14%
Fibrous bands	7%	8%	NA	NA
Lesions mimicking Carcinoma	16%	NA	20%	NA

USG & EUS guided FNA: Suri et al[42] in his series of abdo. Lymphadenopathy found 58% positive diagnosis of abdominal tuberculosis at FNAC. Puri et al[43] found 90.8% positive diagnosis(of failed USG guided FNAC) patients; 76.1% were found to have tuberculosis. Dhir et al[43] studied the utility of EUS-FNA in evaluating intra-abdominal lymph nodes of unknown etiology, in the setting of high endemicity of tuberculosis. Sensitivity, specificity, PPV and NPV for diagnosing tuberculosis via EUS-FNA were 97.1%, 100%, 100% and 96.9%, respectively. **Ascitic Fluid examination:** The tubercular ascitic fluid has protein more than 3 g/dL, with a total cell count of 150-4000/μL and consists predominantly of lymphocytes. The ascitic fluid to blood glucose ratio is less than 0.96 and serum ascitic albumin gradient is less than 1.1 g/dL[44]. The yield of organismson smear and culture is low. Staining for acid fast bacilli is positive in less than 3% of cases and a positive culture is seen in only 20% of cases. Ascitic fluid adenosine deaminase (ADA) levels are elevated in tubercular ascites. Serum ADA level above 54 U/L, ascitic fluid ADA level above 36 U/L and an ascitic fluid to serum ADA ratio more than 0.98 are suggestive of tuberculosis[45]. However, in cases of co-infection with HIV, ascitic ADA levels can be normal or low. Also, falsely high values can be seen in malignant ascites. Interferon-γ levels are also elevated in tubercular ascites[46]. The sensitivity and specificity increased by combining ascitic fluid ADA and interferon-γ assay. **Histopathology:** Microbiological Diagnosis of abdominal tuberculosis is difficult; the yield of organisms from abdominal lesions is low because extra pulmonary disease is paucibacillary. Acid-fast Bacilli were seen on histological examination by Ziehl Nielson Staining in only 6-8% of patients[47]. The Diagnosis of abdominal tuberculosis is therefore mainly histological-Epithelioid cell granulomas with Langhan's Giant cells, peripheral rim of lymphocytes and plasma cells, and central caseation necrosis. Non-caseating granulomas, as seen in Crohn's disease, may be present in tuberculosis due to low virulence of organisms and increased host resistance. Mycobacterial Culture should be performed in all cases (although Results take 6 weeks) because it may be positive even in the absence of a characteristic histological picture[48]. Alvares et al in his study demonstrated well-formed granulomas in 23 patients (54%). 14 of the patients (61%) had caseation and 11 (48%) had confluence of the granulomas. Acid-fast bacilli were present in the biopsies from two patients (5%). Recently Ihama et al[49]. demonstrated the diagnosis of intestinal tuberculosis using amonoclonal antibody to Mycobacterium tuberculosis. The antibody being to the CD 68 present in the granuloma. **Quantiferon-TB Gold (QFT-G) and GeneXpert Assay:** The QuantiFERON-TB Gold In-Tube (QFT-G) is a blood test for use as an aid in diagnosing Mycobacterium tuberculosis infection (both latent and active tuberculosis disease). The test is approved by the U.S. Food and Drug Administration (FDA) for use with adult patients. Interferon-Gamma Release Assays (IGRAs) may be used in place of (but not in addition to) a TST in all situations in which the CDC recommends TST as an aid in diagnosing M. tuberculosis infection. IGRA is preferred for testing persons who have received BCG, to test recent contacts of persons with infectious tuberculosis with follow-up testing. In contact investigations, negative results obtained prior to 8 weeks typically should be confirmed by repeat testing 8-10 weeks after the end of exposure. The **QFT-G** is an indirect test for M. tuberculosis infection that is based on measurement of a cell-mediated immune response. A cocktail of 3 mycobacterial proteins (ESAT-6, CFP-10,

and TB 7.7) stimulate the patient's T-cells in vitro to release interferon-gamma, which is then measured using ELISA technology. The test detects infections produced by the M. tuberculosis complex proteins; thus, patients either vaccinated with BCG or infected with environmental mycobacteria should test negative. Results should always be interpreted in conjunction with other clinical and laboratory findings. In a review of meta-analysis[50] the pooled sensitivity, specificity, positive likelihood ratio, and negative likelihood ratio of IGRA for the diagnosis of ITB was 81% (95% CI, 75-86%), 85% (95% CI, 81-89%), 6.02 (95% CI: 4.62-7.83), and 0.19 (95% CI: 0.10-0.36) The AUC was 0.92. IGRAs do not have high accuracy for the prediction of active TB, although use of IGRAs in some populations might reduce the number of people considered for preventive treatment. Several longitudinal studies show that incidence rates of active TB, even in IGRA-positive individuals in high TB burden countries, are low, suggesting that a vast majority (>95 percent) of IGRA-positive individuals do not progress to TB disease during follow-up[51]. The latest guidelines from the United States, Canada,the European Centre for Disease Prevention and Control (ECDC), the United Kingdom, and World Health Organization (WHO) do not support the use of QFT-G in the setting of active TB. **WHO recommendation 2013; Gene Xpert MTB/RIF** may be used as a replacement test for usual practice (including conventional microscopy, culture or histopathology) for testing specific nonrespiratory specimens (lymph nodes and other tissues) from patients suspected of having extra pulmonary TB (conditional recommendation, very low-quality evidence). **Gene Xpert MTB/RIF** test is a simple method, and routine staff with minimal training can use the system. The test appeared to be as sensitive as culture with smear-positive specimens but less sensitive with smear-negative pulmonary and extrapulmonary specimens that include low numbers of bacilli[52]. **MULTIPLEX PCR:** Multiplex PCR using MPB64 and IS6110 are useful in rapid diagnosis of gastrointestinal TB. Multiplex PCR has sensitivity and specificity of 90% and 100%, respectively in confirmed (AFB/culture/histopathology) cases of gastrointestinal TB and positive results in 72.41% of the suspectedgastrointestinal TB cases[53].

Lapaorscopy: Laparoscopy provides a good deal of visual confirmation of findings, taking biopsy and collecting ascitic fluid for further investigations. Bhargava et al[54] reported laparoscopic findings in 38 proven cases of peritoneal tuberculosis. The laparoscopic appearances can be classified into three types: thickened peritoneum with military yellowish white tubercles with or without adhesions (n = 25), only thickened peritoneum with or without adhesions (n = 8), and fibro adhesive pattern (n = 5). Biopsies were avoided from fibro adhesive lesions due to risk of complications. Visual diagnosis was accurate in 95% of patients. In comparison, in 27 (82%) of 33 patients, the examination enabled a histological diagnosis to be made on the basis of typical granuloma.

Diagnosis

Timely diagnosis based on a high index of suspicion in areas and in populations in which tuberculosis is common, an algorithmic diagnostic approach using radiology, imaging and endoscopy (table-II), and management with a judicious combination of anti-tubercular therapy and conservative surgery (table- III), can reduce the mortality of this 'easily curable yet potentially lethal'disease[55].

Table IV: Diagnostic algorithm

Possible lesions			
Presentation	**Site**	**Type**	**Investigations**
Intestinal Obstruction	Small Intestine	Stricturous	Small Bowel Enema
	Peritoneum	Adhesive	CT
Lump	Ileocaecalreg., Colon		Hypertrophic DCBE Colonscopy+Biopsy
	Lymph Nodes		US, CT, FNA
Diarrhoea	Small Intestine	Ulcerative	Small Bowel Enema, Ileoscopy+Biopsy
Rectal Bleeding	Large Intestine	Ulcerative	Colonoscopy+Biopsy
Ascites	Peritoneum		Ascitic US, CT, Ascitic tap, Laparoscopy+Biopsy +ADA.

Other tests:Quantiferon-TB Gold (QFT-G)(WHO-Recommondation)

GeneXpertAssay(replacment of conventional, microscopy, culture or histopathology)

Interferon-Gamma Release Assays (IGRAs)(Replacement of TST).

MULTIPLEX PCR (Rapid diagnosis)

Table -V: Management

Site	Type	Suggested Treatment
Any Site	Acute Abdomen	Emergency Surgery
Intestinal	Ulcerative	ATT
	Stricture	Strictureplasty, resection
	Hypertrophic	Resection
Peritoneal	Ascitic, Adhesive	ATT+Steroids
Lymph node		ATT

Management

All Patients with abdominal tuberculosis should receive a full course of anti-tubercular therapy. Conventional Regimens include anti-tubercular therapy for 12 To 18 months. Short-course Regimens including ethambutol, rifampicin and isoniazid for 3 months followed by rifampicin and isoniazid for 6 Months or pyrazinamide, ethambutol, rifampicin and isoniazid for 2 months followed by rifampicin and isoniazidfor 4 months, are effective for abdominal tuberculosis[56]. It is important to administer a correct and complete course, as inadequate

drugs, dose or duration is the most important cause of emergence of multi-drug-resistant tuberculosis. As many as 20%[57] to 40% of patients with abdominal tuberculosis who present with acute abdomen require emergency surgical intervention[58]. Acute-on-chronic intestinal obstruction usually responds to conservative management; these patients can then be electively investigated and managed accordingly[59]. Tubercular Perforations are usually ileal and are associated with distal strictures; if the two are close to each other the segment should be resected. Parikh' Described stricture plasty in such situations the incision through the stricture encircling the perforation. If they are far apart the perforation may be closed after freshening the edges and the stricture may be resected or treated with strictureplasty[60]. In Patients with acute tuberculous peritonitis and acute mesenteric lymphadenitis, biopsy alone is performed and the abdomen is closed without drainage."Peritoneal Toilet should be performed. Patients with ulcerative intestinal disease and those with peritoneal and lymph node involvement may be treated with anti-tubercular therapy if no complications are present. In patients with peritoneal disease, the addition of steroids may reduce the subsequent complications of adhesions. Since most patients with strictures and hypertrophic lesions have obstructions, surgical treatment is recommended.

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