37 Abdominal Tuberculosis

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Tuberculosis remains one of the major health problems in the world. WHO estimates that each year 8 million new cases of tuberculosis occur and approximately 3 million people die from the disease (WHO 1996). Tuberculosis is a disease of developing countries; however, its incidence is increasing in developed countries as well, mainly in the immigrant population and in patients with AIDS (McKenna et al. 1995; Barnes et al. 1991).

37.1 Epidemiology

Abdominal tuberculosis is still prevalent in developing countries (Tandon and Prakash 1972; Bhansali 1977; Kapoor 1998). There is confusion on the exact incidence of abdominal tuberculosis in such countries due to problems of actual reporting, difficulty in diagnosis, and inability to separate tuberculosis from Crohn's disease, which can closely resemble it in its clinical manifestations. Abdominal tuberculosis was common in the United States early in the 20th century (Horvath and Whelan 1998). It was the cause of most cases of small intestinal obstruction and stricture. However, by the middle of the century all forms of tuberculosis had declined dramatically. This decline was caused by a number of factors, which included an increased standard of living, pasteurization of milk, control of bovine tuberculosis. and introduction of antituberculous treatment (O'Reilly and Daborn 1995). In fact, frequency of abdominal tuberculosis in the United States in 1960s and 1970s dropped to such low levels that the disease was classified as a "rare" or Third World disease. However, since 1985 the number of reported cases of abdominal tuberculosis has dramatically increased. This was due to two reasons: (1) an increased incidence of all cases of tuberculosis (Brudney and Dobkin 1991; Cantwell et al. 1994) and (2) an increased proportion of extrapulmonary disease, especially abdominal tuberculosis (Farer et al. 1979; Alvarez and McCabe 1984). From 1980 onward, reported cases of tuberculosis in the United States increased. The majority of these cases were in Hispanics, blacks, prisoners, immigrants, refugees, and nursing home patients (McKenna et al. 1995; Cantwell et al. 1994; Nardell et al. 1986; Raviglione and O'Brien 2001; Bradney and Dobkin 1991). Multidrug-resistant tuberculosis in AIDS patients contributed significantly to this increase in the occurrence of the disease (Edlin et al. 1994; Bloch et al. 1994; Gordin et al. 1996; Frieder et al. 1993; Selwyn et al. 1989; CDC 1990, CDC 1991; Small et al. 1993; Anand 1956). The impact of the disease was seen particularly in urban areas. In 1979, there were 1,530 new cases of tuberculosis in New York City, and by 1991 the city had 3,673 new cases of tuberculosis, a yearly increase that is three times the national average. The number of cases continued to increase and peaked in 1992. As a result of aggressive health care control policies, the number of cases has shown a gradual downward trend.

Another reason for high occurrence of abdominal tuberculosis was high proportion of extrapulmonary disease (Farer et al. 1979; Alvarez and McCabe 1984). In 1960s only 8% of patients with tuberculosis had extrapulmonary manifestations. By 1986, extrapulmonary disease constituted 25% of all cases of tuberculosis. The lung is the commonest site (over 85%) of

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involvement in immunocompetent persons, while the disease predominantly affects extrapulmonary sites (over 50%) in patients with AIDS. As the urban epidemic of tuberculosis in the United States occurred in AIDS patients, abdominal tuberculosis revealed a significant resurgence.

Mycobacterium tuberculosis is the pathogen for most cases of abdominal tuberculosis. *Mycobacterium bovis*, an organism transmitted by unpasteurized diary products, is the cause of a small percentage of cases in developing countries (Raviglione and O'Brien 2001; Marshall 1993).

The route of infection occurs by one of the following mechanisms (Kapoor 1998; Horvath and Whelan 1998; Marshall 1993):

1. Swallowing of infected sputum. This occurs in patients with sputum-positive pulmonary disease and those with laryngeal involvement. This was the most important route of infection before the era of effective treatment. Autopsies in patients with pulmonary tuberculosis in the pretreatment era demonstrated intestinal disease in 55% to 99%. The frequency of intestinal disease was related to the severity of pulmonary involvement: 1% of patients with minimal pulmonary tuberculosis, 4.5% with moderately advanced disease, and 25% with far-advanced disease. In modern series, this mode of infection is less important and chest radiograph is completely normal in the majority of patients with intestinal tuberculosis.
2. Hematogenous spread from active pulmonary, miliary tuberculosis or silent bacteremia during the primary phase of tuberculosis. Most cases of abdominal tuberculosis occur as a result of reactivation of a latent focus in the small bowel or peritoneum. This focus is established perhaps previously, because of hematogenous spread from a primary focus in the lungs that subsequently healed completely (as they usually do without leaving any radiologic evidence of a lung lesion). Less commonly, hematogenous spread can occur from active pulmonary focus tuberculosis. Hepatosplenic tuberculosis almost always follows miliary seeding and is a manifestation of dissemination throughout the body.
3. Ingestion of contaminated milk or milk products. This mode of infection had been a common cause of spread of bovine tuberculosis in the past. However, pasteurizing and/or boiling milk has controlled this mode of transmission (O'Reilly and Daborn 1995). At present, *Mycobacterium bovis* is involved in a small percentage of intestinal disease in developing countries, and this form of disease is rare in the West (Anand 1956).
4. Contiguous spread from adjacent organs. Occasional cases of abdominal tuberculosis are related to contiguous spread from tuberculous lesions of adjacent organs. Peritoneal spread can occur from lesions in the fallopian tubes and intestines. Recent data showed that this is an infrequent mechanism in most patients with abdominal tuberculosis. More often, lymph node lesions spread the infection to the bowel wall or pancreas.
5. Tuberculosis in patients with AIDS. Tuberculosis occurs with increased frequency in AIDS patients as the CD4 count drops below 400 cells per µl (Jones et al. 1993). An autopsy study in West Africa found that 50% of adults dying of AIDS had active tuberculosis and in 85% of them the liver was involved. In fact tuberculosis is the most common specific hepatic HIV-associated lesion in such patients (Lucas 1994). The pathology of tuberculosis varies with the immune status of the patient (Bhargava et al. 1984; Edwards and Kirkpatrick 1986). In patients with intact immune systems, granulomas with Langhans giant cells and caseation or non–giant cell epitheloid granulomas are usually seen. In patients with extreme immune deficiency as commonly seen in terminal AIDS patients, the histologic pattern is that of nonreactive tuberculosis. Foci of granular necrosis are surrounded by degenerate swollen macrophages, and a large number of acid-fast bacilli are seen. An analysis using restriction-fragment-length polymorphisms to study the mode of infection of tuberculosis in patients with AIDS has shown that the disease is readily spread from index patients and progresses rapidly to active disease. There was no evidence that disease occurs from reactivation of a latent focus (Daley et al. 1992; Small et al. 1994).
6. Liver disease and tuberculous peritonitis. Patients with cirrhosis of the liver with ascites have a higher chance (around 10%) of concomitant tuberculous infection (Aguado et al. 1990). In the United States, half of the patients with tuberculous peritonitis have underlying alcoholic cirrhosis as a cause of ascites formation (WHO 1990; Raviglione and O'Brien 2001; Lucas 1994). The mechanism of this infection in patients with liver disease is not known. It may be due to reactivation of a latent tuberculous focus in the peritoneum facilitated by lower immunity and coexistent ascites.
7. Tuberculous peritonitis in patients undergoing long-term or continuous ambulatory peritoneal

dialysis (CAPD). Tuberculous peritonitis has been reported as a complication of CAPD (Holley and Piraino 1990; Cheng et al. 1989; Lui et al. 1996; Lam et al. 2000). Talwani and Horvath (2000) reviewed the English-language literature and found 51 reported cases of CAPD-associated tuberculous peritonitis and added a 52nd case from their own experience (Lui et al. 1996). Defects in local immunity unique to CAPD may predispose to active tuberculosis in such patients. Removal of the CAPD catheter is not considered necessary for cure of the infection.

Abdominal tuberculosis can occur at any age and is equally prevalent in males and females (Bhansali 1977; Kapoor 1998; Horvath and Whelan 1998). The majority of patients have symptoms present for 1 month to 1 year; however, around 20% of patients have symptoms for 1 month or less at the time of presentation. Low-grade fever, night sweats, anorexia, weight loss, general lassitude, and weakness occur in around two thirds of patients. Symptoms of disease at other sites occur in patients with active disease in extraabdominal organs. This is of particular significance in patients with active pulmonary tuberculosis or disseminated tuberculosis. Laboratory results reveal mild normocytic or microcytic anemia and normal white blood cell count (Pouchot et al. 1997). PPD is positive in most of the patients; however, it may be negative in immunosuppressed and malnourished patients (Bass et al. 1985; Huebner et al. 1993; American Thoracic Society 1981; Markowitz et al. 1993). Chest X-rays show active disease in about one fifth of patients.

The evolution of the disease in a patient with abdominal tuberculosis depends upon route of infection, site of involvement, and underlying immune status of the subject. Abdominal tuberculosis may affect the gastrointestinal tract, peritoneum, lymph nodes, liver and spleen and pancreas singly or in combination. Abdominal tuberculosis in immunosuppressed patients poses special problems as disease has distinct bacteriologic and clinical characteristics. Gastrointestinal tuberculosis affects, in order of frequency, the ileocecal region, jejunum/ileum, colon, anorectum, stomach, appendix, duodenum, and esophagus (Marshall 1993). Reported sites of involvement of abdominal tuberculosis are shown in Fig. 37.1.

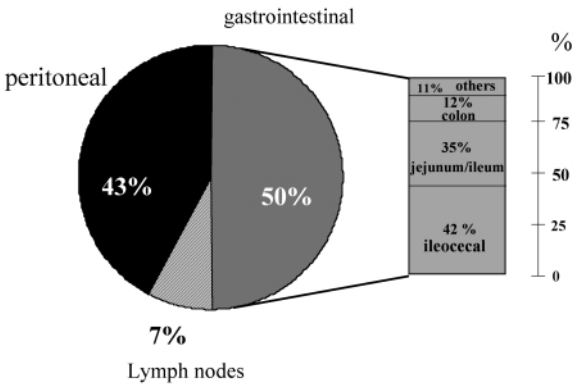


Fig. 37.1. Sites of organ involvement in abdominal tuberculosis. The data are based on 596 patients with abdominal tuberculosis

37.2 Tuberculosis of Small Bowel and Colon

Pathogenesis (Tandon and Prakash 1972; Anand 1956). After the tubercle bacilli enter the gastrointestinal tract, they traverse the mucosa to lodge in the submucosa. There, the presence of the bacilli induces inflammatory changes, including serosal and subserosal edema, cellular infiltrate, and lymphatic hyperplasia. Eventually, the appearance of granulomata causes small papillary mucosal elevations. Lymphangitis, endarteritis, and fibrosis ensue, which lead to mucosal ulceration, caseating necrosis, and narrowing of the intestinal lumen. Mucosal ulceration may occur as a result of endarteritis of submucosal vessels. Infection can spread to mesenteric lymph nodes.

As mentioned earlier, the most common site of involvement is the ileocecal region. The affinity of the bacilli for this site may be due to its relative stasis and abundant lymphoid tissue. The macroscopic appearances of intestinal lesions can follow one of the below-mentioned patterns. Such lesions are usually segmental, and multiple sites of involvement are common. Rarely, diffuse colonic involvement may simulate ulcerative colitis and Crohn's disease. Other characteristics include increased mesenteric fat and mesenteric lymphadenopathy, which can cause traction diverticula with narrowing, local fixation, and sinus tract development.

a. An ulcerative lesion is characterized by multiple superficial ulcers. Ulcers are circumferential and usually surrounded by inflamed mucosa. This is the most common lesion, occurring in around 60% of such patients and is associated with a virulent clinical course.
b. A hypertrophic lesion is characterized by scarring, fibrosis, and pseudotumor formation. This is seen in around 10% of such patients.

c. An ulcerohypertrophic lesion is characterized by an inflammatory mass with thickened and ulcerated mucosa. The lesion is most commonly seen in the ileocecal region. It causes cone-shaped deformity of the cecum, shortening of the ascending colon, and thickening of the ileocecal valve, where a wide gape is created. Overall this lesion is seen in 30% of such patients.
d. Fibrous stricture occurs in some patients as a result of healed ulceration causing luminal narrowing and gut obstruction. In some cases this occurs after effective antituberculous therapy. Luminal narrowing may also occur due to extraintestinal lymph node involvement without intrinsic intestinal lesions.

Clinical Manifestations. Abdominal symptoms depend upon the site of involvement of disease, pattern of pathologic changes, and underlying immunologic status of the host (Bhansali 1977; Kapoor 1998; Horvath and Whelan 1998; Marshall 1993). Involvement of small bowel and colon leads to single or multiple strictures through a number of underlying pathogenic mechanisms (see as above). Abdominal pain in such patients is characteristically described as a "ball of wind" moving around the umbilicus. It is associated with abdominal distension, inability to pass wind, and borborygmi. Following an episode of pain, diarrhea usually ensues. Steatorrhea and significant weight loss can occur due to bacterial overgrowth. Right lower quadrant mass and pain can occur in patients with hypertrophic ileocecal tuberculosis. Rarely, diffuse colonic disease can simulate symptoms of ulcerative colitis. Perforation and fistulae occur in a small percentage of patients. Massive bleeding from the lesion in the gut has been reported. Clinical examination reveals distended bowel loop and exaggerated bowel sounds. Plain X-ray of the abdomen reveals distended bowel loops with multiple fluid levels.

Diagnosis. Intestinal tuberculosis can be difficult to diagnose. The reasons for this include absence of a particular pattern of symptoms and signs. In fact, symptoms of the disease may be vague and signs nonspecific. Thus, a high degree of suspicion is needed. Even with adequate imaging, endoscopic examination and bacteriologic tools, diagnosis can correctly be made in only around 50% of patients with intestinal tuberculosis. The dominant reason for this is the inaccessibility of common sites of disease segments of the bowel, namely the ileum and ileocecal region. Moreover, the hallmark of tuberculous pathology, namely caseating granulomas, may be absent in the bowel wall and present in the draining lymph nodes (Tandon and Prakash 1972; Anand 1956). Laparotomy and resection of the involved segments with culture and animal inoculation of the organisms have been performed to make a diagnosis with precision in endemic areas. Therapeutic trial with antituberculous drugs is commonly used in developing countries to make a diagnosis.

A number of clinical conditions closely simulate intestinal tuberculosis. These include Crohn's disease, amebiasis, carcinoma colon, *Yersinia enterocolitis*, gastrointestinal histoplasmosis, and periappendiceal abscess. A number of features may help to differentiate intestinal tuberculosis from Crohn's disease and *Yersinia* infection. These have been detailed in Table 37.1.

The diagnostic algorithm to be followed for intestinal tuberculosis may vary with the exact site of disease involvement (Fig. 37.2). X-ray chest, PPD skin test, and flat abdominal films are usually used for the

Table 37.1. Differentiating features of abdominal tuberculosis from Crohn's disease and Yersinia infection

Feature	Tuberculosis	Crohn's disease	Yersinia enterocolitica
Clinical course	Prolonged	Long intermittent	Several weeks
Stool culture	Negative	Negative	Positive
Serology for Yersinia	Negative	Negative	Positive
PPD	Positive	Negative	Negative
X-ray of chest	Positive	Negative	Negative
Ileal disease	Short	Long	Short
Ulcers	Circumferential	Linear	Normal endoscopy
Fistulae	Unusual	Common	Nil
Granulomas	Large, many, caseating	Small, few, noncaseating	Intramural, multiple, large, with satellite Abscess
Anal lesions	Rare	Frequent	Nil
Strictures	Usually <3 cm	Long	Localized
Nodal involvement	Often, independent of mural disease	Only with transmural disease	In children with ileitis

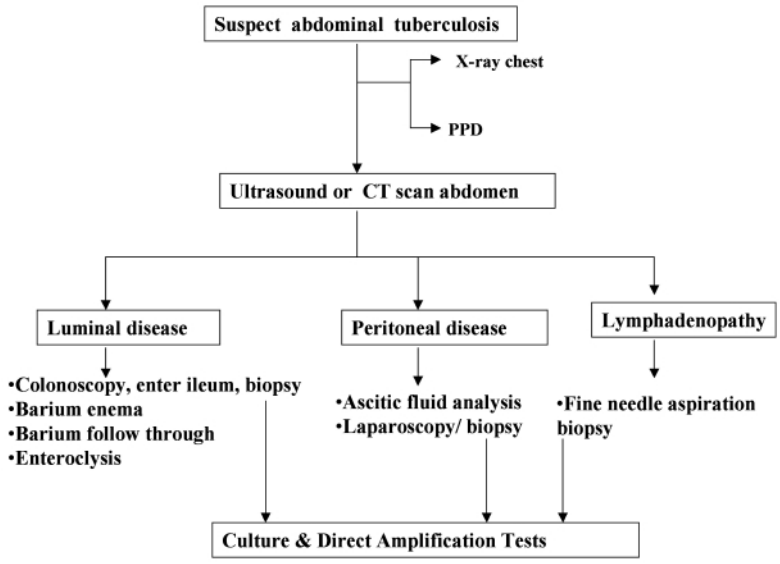


Fig. 37.2. An algorithm which is useful to investigate most cases of abdominal tuberculosis

initial investigations. Active pulmonary disease may help; however, it is seen in only a minority of patients. PPD skin test is positive in the majority of patients with abdominal tuberculosis, but is of limited value because it does not differentiate between active disease and previous exposure or vaccination. Furthermore, the PPD skin test may be negative in older or immunosuppressed patients (Lui et al. 1996; Lam et al. 2000). Careful examination of the flat abdominal films may give important clues to the nature and site of underlying pathology. Calcification of lymph nodes is of the speckled type, and rarely calcification of peritoneum may coexist. Episodes of abdominal pain are usually associated with dilated bowel loops with air fluid levels proximal to the site of stricture.

Abdominal imaging by ultrasound, computed tomography, or magnetic resonance imaging is useful to define the bowel wall, abdominal lymph nodes, and changes in peritoneum, mesentery, and omentum. CT, with its ability to provide a comprehensive overview of abdominal structures, is the imaging modality of choice for such evaluations (Suri et al. 1999; Balthazar et al. 1990; Ha et al. 1999). The most common CT findings are mural thickening affecting the ileocecal region, either limited to the terminal ileum or cecum or, more commonly, simultaneously involving both regions (Fig. 37.3, 38.10, 38.12). This mural thickening is usually concentric, but is occasionally eccentric, and it predominantly affects the medial wall. In some patients, low-density areas, most likely to represent necrosis, may be noted within the thickened wall. Ileocecal involvement is usually associated with enlarged hypodense nodes in the adjacent mesentery. Skip areas of concentric mural thickening

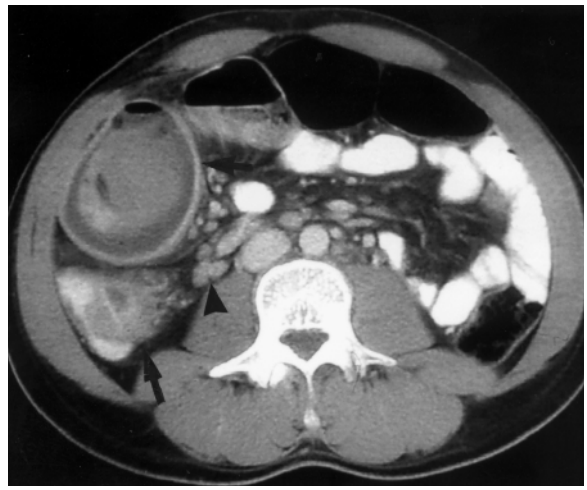


Fig. 37.3. Tuberculosis of colon. A 28-year-old male presenting with fever, abdominal pain, and loose motions of 18 months duration. He had a strong history of contact with an open case of pulmonary tuberculosis. General physical examination was unremarkable. Abdominal examination revealed fullness and vague tender mass in the right iliac fossa. ESR was 10 mm and PPD skin test was 7 mm. Plain X-rays of chest and abdomen were normal. Computed tomography of abdomen with oral and IV contrast shows thickened cecum *(short arrow)*, a mass below the cecum *(long arrow)* and 2 lymph nodes of 1 cm diameter each *(arrowhead)*. A barium enema (Fig. 37.4a), colonoscopy (Fig. 37.5c), colonic biopsies from the lesion in the cecum (Fig. 37.6b) were performed. Colonic biopsy samples grew *Mycobacterium tuberculosis* on culture. He made rapid clinical improvement with antituberculous treatment

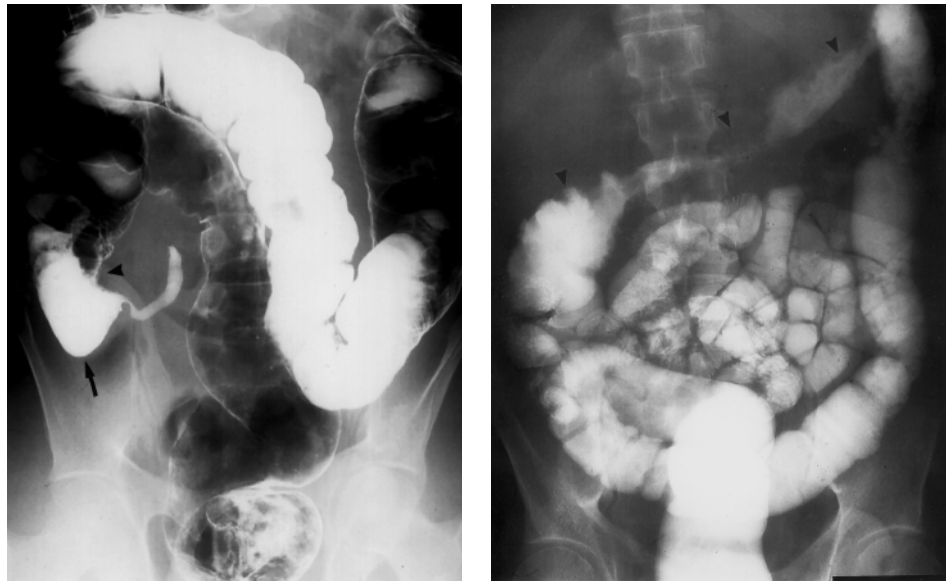
may be seen elsewhere in the small bowel, usually affecting the ileal loops. These segments may also show luminal narrowing with or without proximal dilatation. The presence of such lesions in combina-

tion with ileocecal involvement should strongly suggest the diagnosis of tuberculosis.

Barium enema and small bowel follow-through may show mucosal ulceration, strictures, deformed cone-shaped and retracted cecum, incompetent ileocecal valve, a wide gap between a thickened ileocecal valve and a narrowed ileum (Fleischner's sign), and a fibrotic terminal ileum that empties into a rigid contracted cecum (Stierlin's sign) (Fig. 37.4, 38.9) (Suri et al. 1999; Balthazar et al. 1990; Ha et al. 1999). Small bowel enema (enteroclysis) has a special advantage in defining the site and number of small bowel strictures (Fig. 38.5, 38.6, 38.7)

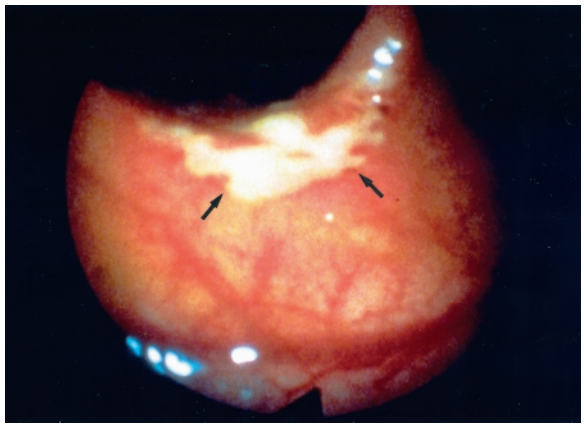
Colonoscopy has been used in patients with colonic and ileocecal tuberculosis (Singh et al. 1998; Bhargava et al. 1992; Shah et al. 1992; Misra et al. 1999; Kalvaria et al. 1988). It has the advantage that targeted biopsies from endoscopic abnormalities can be taken for histology, culture, and molecular techniques (Kochhar et al. 1991; Pulimood et al. 1999; Jost et al. 1995; Anand et al. 1994; Kashima et al. 1995; Pfyffer et al. 1996; Yajko et al. 1995; Tevere et al. 1996; Rich et al. 1996; Simon et al. 1993; Schluger et al. 1994; Bradley et al. 1996; Wobeser et al. 1996; Carpentier et al. 1995; Vlaspolder et al. 1995; Shah et al. 1998). Colonoscopic examination in 50 patients of colonic tuberculosis revealed ileocecal disease in 16, ileocecal and contiguous ascending colon disease in 14, segmental colonic disease in 13, ileocecal disease and nonconfluent involvement of another part of the colon in 5, and pancolitis in two patients (Singh et al. 1996). The colonoscopic appearances include mucosal nodules and ulcers, stricture with nodules and ulcerations, and mucosal nodules with or without pseudopolypoid folds (Fig. 37.5). Nodules vary in size from 2 to 6 mm and have a pink surface. These are scattered and at places densely packed. Friability of mucosa over nodules is unremarkable. Ulcers may be from a few millimeters to 2 cm long and are superficial with sharply defined irregular margins. Ulcers are covered with slough, which is difficult to wash away. The surrounding mucosa is nodular and hyperemic and blends imperceptibly with normal mucosa. When the ileocecal valve is involved, it is edematous, deformed, patulous, and easily admits the endoscope into the diseased terminal ileum. With diffuse colonic involvement, mucosa from rectum to cecum is hyperemic and friable and shows areas of circumferential ulcerations of different sizes along the entire length of the colon. Biopsy samples should be taken from ulcer edge, ulcer base, nodules, and from adjacent normal mucosa.

Endoscopic mucosal biopsies from the colon and terminal ileum may show a mixture of pathologic changes and include (1) characteristic and diagnostic caseating granulomas in about 25% of patients, (2) noncaseating granulomas in about 35%, (3) ulceration with nonspecific granulation tissue and infiltration with polymorphs forming microabscesses in around 60%, (4) variable mucosal reparative changes in around



a b

Fig. 37.4a, b. Tuberculosis of colon. **a** Barium enema shows filling defect *(arrowhead)* and cone shaped deformity (*long arrow*) of the cecum. Appendix is normally filled. **b** Barium enema shows lack of distensibility and nodular defects of the hepatic flexure, transverse and splenic flexure *(arrowheads)*



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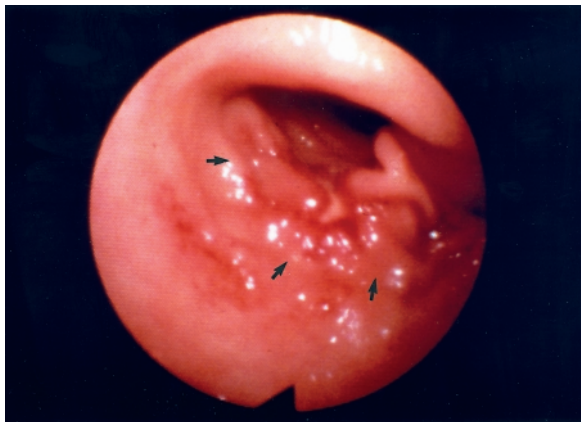

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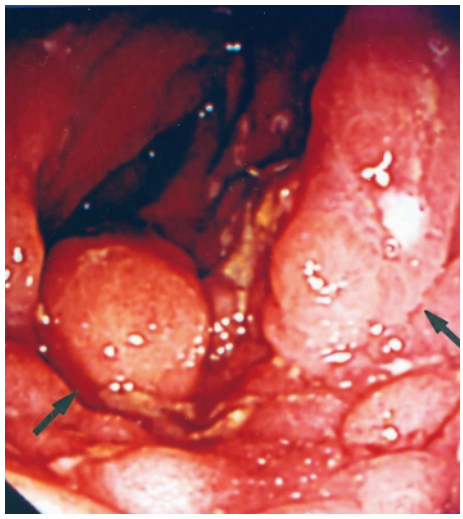
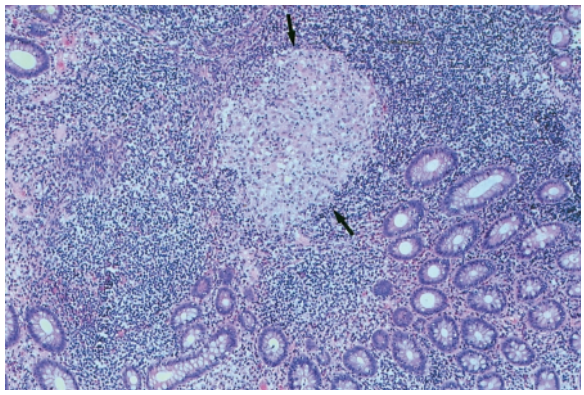


c

Fig. 37.5a, b. Tuberculosis of colon. Colonoscopic views in three patients. **a** A transversely placed ulcer in the descending colon *(arrows)*. **b** An infiltrative and nodular lesion in the transverse colon *(arrows)*. **c** Hypertrophic nodular mass in the cecum *(arrows)*

20%. Characteristic granulomas show caseous necrosis in the center, are often large, with marked variations in size, and usually tend to be confluent (Fig. 37.6). The granulomas seem to be enlarged by expansion of individual granulomas or by confluence of numerous satellite granulomas. This is in sharp contrast to sarcoid granulomas seen in Crohn's disease which are small in size, closely adjacent but discrete, and do not become confluent (Tandon and Prakash 1972).

Endoscopic mucosal biopsy rarely shows *M. tuberculosis* organisms on smear, and routine culture yields a growth of bacilli in only 6% to 40% of speci-



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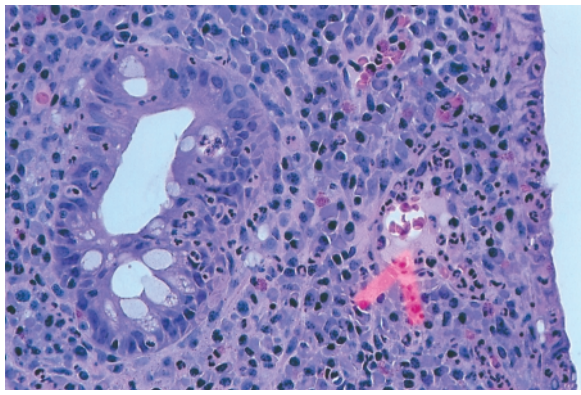


b

Fig. 37.6a, b. Tuberculosis of colon. **a** Histologic examination of colonic biopsy revealed dense lymphoplasmocytic infiltrate in lamina propria and a well-formed granuloma *(arrows)* consisting of epitheloid histiocytes and multinucleated giant cells. **b** Histologic examination of colonic biopsy showing moderate lymphoplasmocytic infiltrate in the lamina with infiltration and destruction of crypts *(arrow)*. No granulomas were seen

mens (Singh et al. 1996; Bhargava et al. 1992; Shah et al. 1992; Misra et al. 1999; Kalvaria et al. 1988). Recent technologic developments have introduced a number of improvements in the ability of clinical laboratories to cultivate and identify *Mycobacterium tuberculosis* complex more quickly and precisely than previously. These developments include more rapid detection of growth (Jost et al. 1995) and tests to identify RNA or DNA of *M. tuberculosis* complex directly in clinical samples (Anand et al. 1994; Kashima et al. 1995; Pfyffer et al. 1996; Yajko et al. 1995; Tevere et al. 1996; Rich et al. 1996; Simon et al. 1993; Schluger et al. 1994; Bradley et al. 1996; Wobeser et al. 1996; Carpentier et al. 1995; Vlaspolder et al. 1995; Shah et al. 1998). Exploitation of such tools for intestinal tuberculosis will make the diagnosis easier and more frequent.

37.3 Tuberculous Peritonitis

Pathogenesis (Marshall 1993; Singh et al. 1969). Peritoneal seeding by tubercle bacilli causes granulomas, which appear as multiple, whitish miliary nodules (<5 mm) scattered over the visceral and parietal peritoneum. In addition, the peritoneal lining along with the omentum and mesentery is thickened and adhesions develop with abdominal organs. A majority (>95%) of patients develop exudative free or loculated ascites; however, a small group of patients may have a more advanced dry fibroadhesive (plastic) or purulent form of disease. Plastic peritonitis causes adhesions and matting of bowel loops, mass formation due to matting of bowel loops, adenopathy, mesenteric and omental thickening (omental cake). Purulent peritonitis is usually secondary to tuberculous salpingitis and causes abscess formation due breakdown of caseous lesions in lymph nodes, mesentery, or omentum. These abscesses are present within matted bowel loops and thickened omentum and mesentery. Fistulae, both cutaneous and enteric, are common when such abscesses rupture either through the skin or into the bowel.

Clinical Manifestations (Marshall 1993; Singh et al. 1969; Manohar et al. 1990). Tuberculous peritonitis in its ascitic form presents insidiously with progressive abdominal distension. Diffuse abdominal pain (65%), fever (71%), and weight loss (38%) are seen in a variable percentage of patients. Clinical examination reveals shifting dullness, abdominal tenderness, and transverse solid epigastric intra-abdominal mass. The last is caused by rolled-up, thickened omentum infiltrated with tubercles. The encysted form of the disease produces a localized cystic mass usually in the central or lower abdomen, resembling a mesenteric cyst in children and ovarian cyst in females. Plastic peritonitis produces matted small bowel loops with thickening of, and adhesions with omentum and mesentery. Patients often present with recurrent attacks of subacute intestinal obstruction. Acute intestinal obstruction may sometime supervene. Dilated bowel loops produce bacterial overgrowth and cause steatorrhea and wasting. Abdominal examination reveals single or multiple bowel masses which are resonant to percussion (thickened and matted bowel loops). Solid mass may be caused by thickened mesentery. Patients with purulent peritonitis are very sick, wasted, and in moribund clinical status. Abdomen examination reveals tenderness, guarding, multiple bowel masses, and usually a fecal fistula commonly near the umbilicus.

Patients with tuberculous peritonitis with cirrhosis of the liver present with similar clinical features to those without liver disease. However, patients with liver disease are younger (42±8 years vs 54±15 years, $p<0.01$) and have a higher maximum-recorded temperature (102±107 vs 100.5±1.3, $p<0.01$). In addition, clinical examination reveals hepatomegaly (48%) and splenomegaly (20%) due to underlying liver disease and portal hypertension (Aguado et al. 1990; Shakil et al. 1996).

Tuberculous peritonitis in patients with long-term or continuous ambulatory peritoneal dialysis present with fever, abdominal pain, and cloudy dialysate. Peritoneal fluid has predominance of polymorphonuclear cells as against lymphocytic predominant cells in tuberculous peritonitis associated with other conditions. Diagnosis is made at culture of the fluid, which grows tubercle bacilli in two thirds of such patients (Holley and Piraino 1990; Cheng et al. 1989; Lui et al. 1996; Lam et al. 2000; Talwani and Horvath 2000).

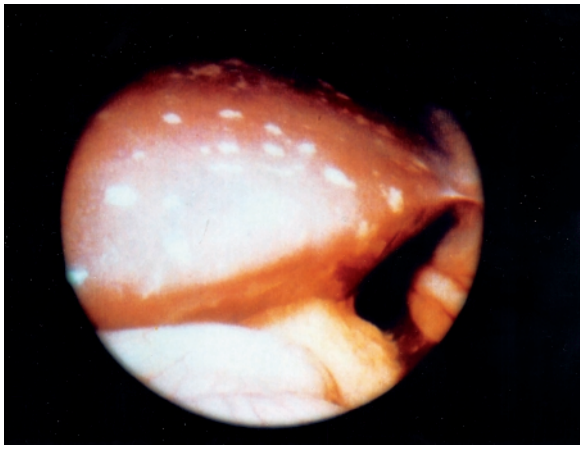
Diagnosis. Diagnosis of tuberculous peritonitis is mainly focused on the differential diagnosis of ascites and a well-established algorithm has been developed in clinical practice to do so (Runyon et al. 1992). The index of suspicion of tuberculous peritonitis should be high in following circumstances:

a. Residence in developing countries or immigration to a Western country from a developing country
b. Recent exposure to open tuberculosis
c. Underlying cirrhosis
d. Patients on long-term or continuous ambulatory peritoneal dialysis
e. Immunosuppressed patients, especially AIDS, and patients with liver or renal transplants

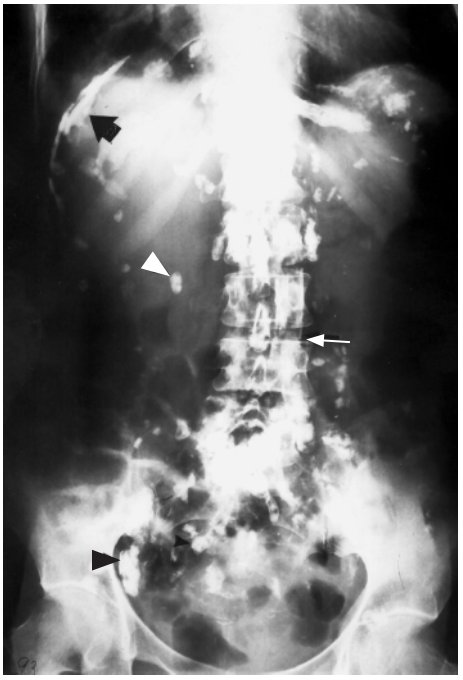
The value of a chest X-ray, PPD, and flat abdominal films has been discussed (Fig. 37.7). Abdominal imaging, especially CT scan, is useful for an initial investigation to give a comprehensive view of the abdominal organs. Ascitic fluid analysis gives an important lead to the possibility of infectious etiology. Laparoscopy and peritoneal biopsy is the investigation of choice to confirm the diagnosis of tuberculosis.

CT findings include changes in the peritoneal lining and cavity, mesentery, and omentum (Suri et al. 1999). Peritoneal lining shows smooth uniform thickening. Nodular implants with irregular thickening of the peritoneum are unusual and more often suggest peritoneal carcinomatosis (Fig. 37.8, 38.19a,b). Peritoneal fluid may be free or loculated and shows high-density signals (25–45 HU), possibly explained by high protein and cellular contents of the fluid. However, tuberculous ascites may also be near water density, perhaps reflecting an earlier transudative stage of immune reaction. Mesenteric infiltration can range from mild involvement in the form of linear soft tissue strands, thickened and crowded vascular bundles, a "satellite" appearance, and subtle increase in mesenteric fat density, to more extensive involvement resulting in diffuse infiltration with soft tissue density masses involving the leaves of mesentery surrounding the adjacent bowel loops. Omentum infiltration may cause thickening, smudged appearance, or omental "cake " formation. Retroperitoneal and mesenteric nodes may be enlarged and caseate to form large mesenteric abscesses (Fig. 37.9).

Ascitic fluid may be collected from either flank or centrally below the umbilicus with a blind peritoneal needle puncture and aspiration (Runyon 1986). In patients with minimal fluid collection or those with thick abdominal wall due to obesity, ultrasound-guided fluid collection may be done (Goldberg et al. 1970). Ascitic fluid examination should include gross inspection, biochemical tests, cytology, and smear and culture for tuberculosis. Fluid for a cell count should be sent to the laboratory in an anticoagulant tube (i.e., containing heparin/ethylenediaminetetraacetic acid) to prevent clotting (Hoefs 1990). Before the 1980s, the ascitic fluid total protein concentration



a



b

Fig. 37.7a, b. Tuberculous peritonitis. A 50-year-old woman presenting with low-grade fever, weight loss, diffuse abdominal pain, and abdominal distension of 6 months duration. Clinical examination revealed abdominal tenderness and free fluid in the peritoneum. ESR was 60 mm and PPD skin test was 25 mm. X-ray chest revealed right apical infiltration and scarring. Ascitic fluid analysis revealed low-gradient lymphocytic exudate. **a** Laparoscopic examination revealed adhesions, peritoneal exudates, and multiple small (3 to 5 mm), whitish, elevated lesions on the visceral and parietal peritoneal surface. In this photograph multiple such lesions are shown on the liver surface. The results of a peritoneal biopsy from this patient are shown in Fig 37.10 **b** Plain X-ray of abdomen showing plaque-like calcification in the right and left upper quadrant (peritoneum – *thick arrow*), nodular calcification in the abdomen (lymph nodes – *arrowheads*), and incidental atherosclerotic linear calcification along the aortic wall (*arrow*).