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Gastrointestinal Radiology

Intestinal Tuberculosis

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Abstract. Intestinal tuberculosis is a disease with protective clinical manifestations. The radiographic changes in the bowel are similarly varied but often highly suggestive if not characteristic.

The key radiologic changes in the bowel, with emphasis on the ileocecal area, are described and the clinical and pathologic features are reviewed.

Key words: Tuberculosis — Intestinal tuberculosis — Ileocolitis.

In the United States abdominal tuberculosis is seldom suspected preoperatively, or seriously considered in the differential diagnosis of bowel disease. This contrasts sharply with medical practice in countries such as India or South Africa where tuberculosis is endemic and considered early in the diagnosis [1–3]. Although the clinical, roentgenographic, and pathologic manifestations of intestinal tuberculosis are not unique, it is possible to identify those patients in whom the diagnosis of tuberculosis should be suspected so as to institute effective therapy as soon as possible.

Enterocolitic Tuberculosis

Pathogenesis

The distal ileum and colon are the most common sites of tuberculous infection in the gastrointestinal tract. Three major routes of infection of the bowel have been proposed:

1. *Via* infected bile. Although theoretically possible, no compelling evidence has been presented to support this pathway [4].

2. *Via* ingestion of heavily infected material (milk or sputum). This is the most widely accepted mechanism for intestinal infection. *Mycobacterium bovis*, the causative organism of bovine tuberculosis, has been isolated from intestinal lesions in patients exposed to contaminated milk [5]. In addition, a strong positive correlation between intestinal disease and the extent of pulmonary tuberculosis has been described [5, 6]. Crawford and Sawyer [7], furthermore found that 96.7% of patients with tuberculous laryngitis, as a complication of pulmonary infection, had intestinal infection as well.

3. *Via* hematogenous spread to submucosal lymphatic structures from a distant primary infection. This is an attractive hypothesis for a variety of reasons. The early intestinal lesion is generally found in the submucosa with a normal overlying mucosa [8]. There is evidence that a silent bacillemia can occur early in primary tuberculosis [9]. Mitchell [10] uncovered a strong relationship between intestinal tuberculosis and the bilateral, symmetric, diffuse nodular pattern of pulmonary tuberculosis, which is considered a hematogenous disease. In addition, many series indicate that a substantial number of patients with gastrointestinal tuberculosis, but *not* selected from known tuberculous populations, have negative chest radiographs [1, 3, 11–16].

Relationship to Pulmonary Disease

The relationship of intestinal tuberculosis to pulmonary tuberculosis is not clear. In the large early American series from tuberculosis sanatoria, there was a strong direct correlation between the two conditions. Intestinal involvement occurred much more often in patients with advanced, chronic pulmonary disease than in patients with less severe pulmonary tuberculosis [16, 17].

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More recent experience does not support this relationship so clearly. In the series reported by Werbeloff et al. [3], and in several other studies [13, 15, 18] consistently less than 50% of the patients with intestinal tuberculosis had radiographic evidence of pulmonary disease.

The disparity in the above-mentioned statistics may relate to the populations studied, the criteria for diagnosis, as well as the basic disease process. The key point is that intestinal tuberculosis, as it is seen today, frequently occurs without radiologic evidence of pulmonary tuberculosis. Consequently, a negative chest radiograph is not helpful in excluding intestinal tuberculosis from the differential diagnosis.

Several of the large series accumulated prior to 1950 provide insight into the pattern of gastrointestinal tuberculosis. There is a striking tendency to preferentially affect the distal small bowel and proximal colon. In a series of over 1,000 autopsied cases of pulmonary tuberculosis [19] evidence of intestinal tuberculosis was found in 70.4% of patients. Approximately 89% of patients with intestinal lesions had disease in the ileum and 75.8% had disease in the cecum. Other important sites of infection included the jejunum (34.6%), ascending colon (51.2%), transverse colon (33.7%) and descending colon (22.8%), and the appendix (33.9%). The duodenum, stomach, and rectosigmoid colon were involved less frequently.

This series demonstrates another important point. The small bowel alone was involved in only 17.8% of cases and the large bowel alone in only 8.96% of cases. The cardinal pattern was entercolitis involving the distal small bowel and proximal colon, centering in the ileocecal region [19].

More recent, smaller series from countries where intestinal tuberculosis is common tend to confirm the earlier observations made by Cullen [19]. In the series reported by Gupta and Dube [20] in 1970, Lewis and Kolawole [2] in 1972, and Tandon and Prakash [21] in 1972, there is a striking preponderance of ileocolitis, with the major involvement in the proximal colon.

Clinical Features

Clinical manifestations of tuberculous entercolitis are varied, deceptive, and generally of little help in the differential diagnosis. The main features are those of any subacute or chronic systemic infection associated with vague, nonspecific abdominal symptoms. Anemia, nausea and vomiting, and ascites (with peritoneal disease) may also occur. Hypertrophic ileocolitic tuberculosis may produce an abdominal mass that is somewhat softer, smaller, and more tender than a neoplasm.

An entirely different set of clinical manifestations can occur if the infection has progressed to a late stage. Patients with intestinal strictures can develop intestinal obstruction [2, 13, 21]. Other presentations include intestinal perforation [22], enterocolitic fistula [13], and intestinal hemorrhage [4], and (in one reported case) a malabsorption syndrome [24].

Pathology

The histologic features of tuberculous infection are quite similar throughout the bowel, depending on the stage or type of infection.

The histology of tuberculous enterocolitis reflects the evolution of the tuberculous lesion. The initial response to infection is a local acute inflammatory reaction which, by 10 to 20 days, becomes compact and forms an epitheloid tubercle with a surrounding lymphocyte infiltrate. Caseous necrosis begins within 15 to 30 days and fibrous scarring may follow. Throughout this process viable tubercle bacilli can be found both in phagocytic cells and in the center of a caseous area [25].

Three gross forms of intestinal tuberculosis are classically described: ulcerative, hypertrophic, and ulcerohypertrophic [12, 14, 21, 26–29]. The ulcerative form is generally considered the most common [28]. The ulcerations are classically described as perpendicular to the axis of the bowel. They are generally quite short, often 3 to 6 mm in length. The ulcers are frequently multiple and can be parallel when adjacent to one another.

Although the ulcers generally do not extend through the muscularis, the crater may be deep because of considerable thickening of the surrounding mucosa and submucosa. Circumferential orientation is the rule, but they may also be longitudinal, stellate, or nondescript. The base of the ulcer is often shaggy and necrotic, and margins may be irregular. Spasm of the bowel in an area of ulcerative enteritis with functional stenosis is typical in the acute phase of infection. In the more chronic phase, "napkin ring" strictures may develop in the areas of previous ulceration [21, 22, 26, 28, 29].

Orientation of the ulcers may be due to the transverse orientation of submucosal lymphatic structures, which may be the primary site of tuberculous infection of the bowel. Inflammation and tubercle formation would cause the overlying mucosa to necrose and slough in a transverse pattern. It has been noted that when a Peyer's patch alone is involved, a longitudinal ulcer can result [22].

The hypertrophic form of intestinal tuberculosis is more common in the ileocecal area and colon than

in the mesenteric small bowel. This particular form is frequently associated with a negative chest radiograph and is considered by many to be a primary tuberculous infection [1, 5, 14, 31]. There is an abundant inflammatory response with a large amount of reactive tissue. An abdominal mass is frequently present with adhesions and fibrosis. The bowel wall and surrounding nodes are thickened and matted together. Hyperplastic nodular mucosal patterns (cobblestoning) and a large intraluminal inflammatory mass, resembling a neoplasm, may occur. Long areas of involvement showing a narrowed, "pipe-stem" lumen, long areas of contraction, and stenosis are also considered part of the hypertrophic form. Extensive involvement of the adjacent mesentery and mesenteric nodes are classic features [2, 14, 21, 27, 30, 32].

Combined features of both forms of tuberculous enterocolitis can occur in any area and lead to an ulcerohypertrophic pattern. This type not only demonstrates hyperplastic, cobblestone areas but also multiple ulcers in the affected segment [2, 14, 21, 31]. Histologically, inflammatory thickening of the bowel wall, mixed infiltrates, and granuloma formation are present. The granulomas may be relatively superficial or may extend through the bowel wall into the mesentery.

Caseous necrosis most commonly occurs in the mesenteric lymph nodes rather than in the bowel wall [14, 21, 26,30, 31]. Intense fibrosis with collagenous strictures is often prominent. Tubercle bacilli may occasionally be demonstrated in the mesenteric nodes but rarely in the bowel lesions. Preoperative tests are of little value in establishing the diagnosis of intestinal tuberculosis [3]. Bacteriologic examination of the stool or intestinal contents may well be negative. Gastric aspirates may show tubercle bacilli if the patient is actively ingesting infected material, but this is more helpful in diagnosis of pulmonary than gastrointestinal disease (which is primarily an infection of the deeper layers of bowel). Of particular importance in the differential diagnosis is the fact that caseation is usually absent in Crohn's disease. The microscopic pathology of intestinal tuberculosis sometimes reproduces Crohn's disease in such detail that the two entities are indistinguishable. Gross changes are also similar so that even endoscopy may not be helpful. If a classic ulcer pattern is seen radiologically in the colon and tuberculosis is suspected, caseous necrosis of acid-fast bacilli must be sought [21, 26, 30, 31, 33].

Complications

Complications of intestinal tuberculosis are rare but may include intestinal fistulas, tuberculous appendicitis, and enterolithiasis. Patel and De [23] found 90 reported cases of intestinal fistula, most commonly involving the sigmoid. Similarly, Prout [22] reported a patient with multiple tuberculous perforations of the distal small bowel. The thickening of the intestinal wall and marked tendency to sclerosis so prominent in intestinal tuberculosis may account for the rarity of perforation.

Tuberculous appendicitis (Fig. 1) usually presents as a chronic process. It is an unusual lesion; the changes in the appendix may result from involvement of surrounding nodes as well as intrinsic disease of the appendix [2, 20, 33].

Enterolithiasis may be a late sequel to intestinal tuberculosis. Chawla [31], analyzing 400 cases of tuberculous enterocolitis, found multiple opaque enteroliths in 13 patients. Bery et al. [34] also reported multifaceted opaque enteroliths proximal to tuberculous intestinal strictures where stasis of the bowel content occurs. These stones may vary in size and shape and tend to be, in part, radiopaque.

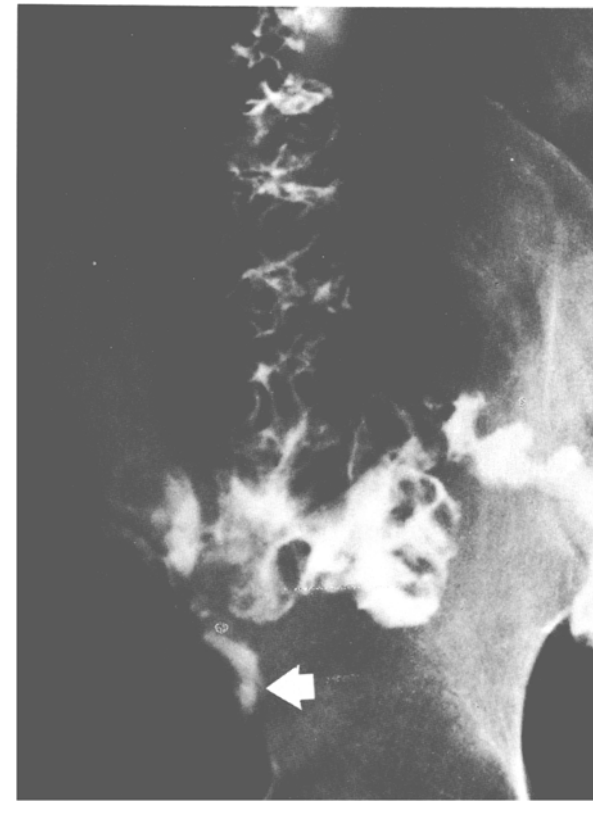
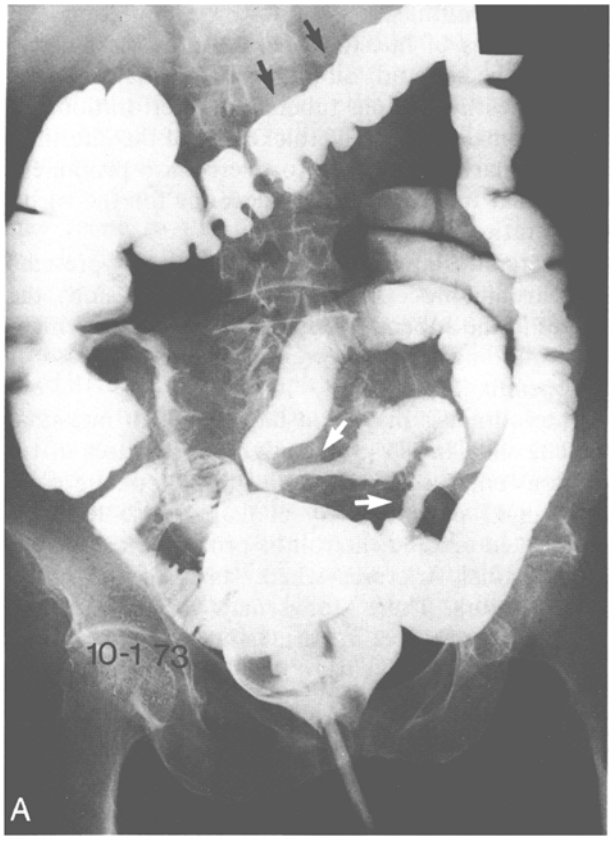


Fig. 1. *Tuberculous appendicitis.* This 46-year-old man with known pulmonary disease developed a fecal fistula that drained acid-fast bacilli postoperatively. On this postevacuation film the mucosa of the cecum is nodular. The fistulous tract extending from the lateral cecal wall is clearly opacified. On biopsy this tract showed typical tuberculous lesions



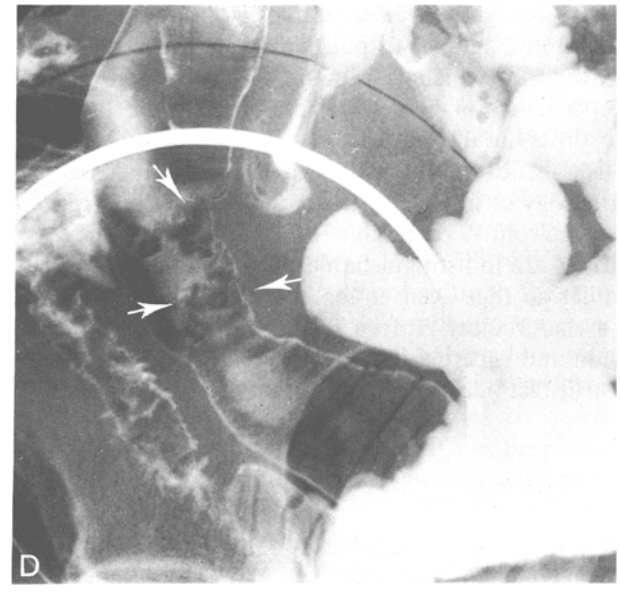
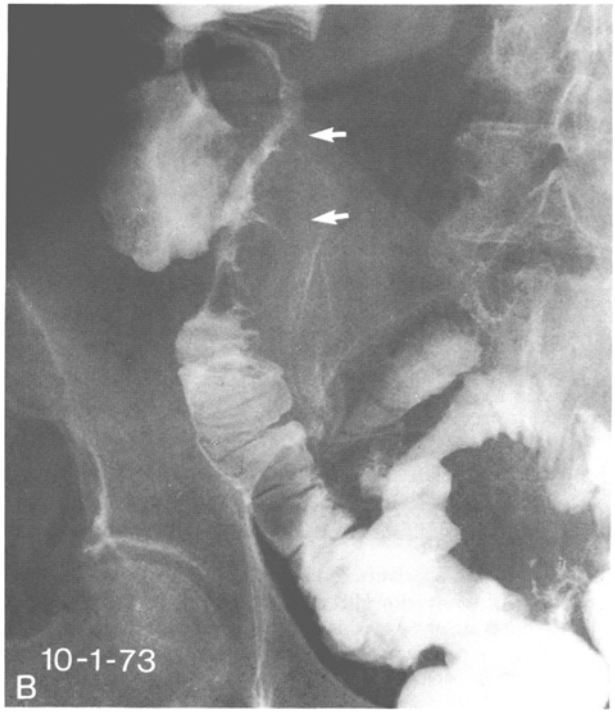
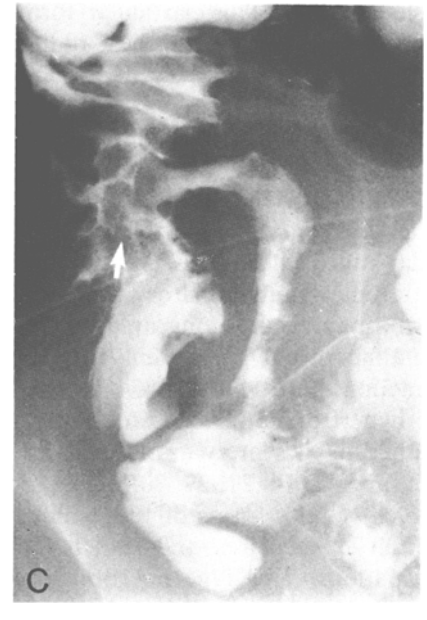


Fig. 2A–D. *Ileocolic tuberculosis.* **A** The most severe disease centers at the ileocecal valve, but there are skip areas of involvement in the distal ileum (white arrows) and transverse colon (black arrows). **B** Note the marked thickening of the lips of the ileocecal valve. The mucosa of the terminal ileum is effaced and perpendicular ulcers extend deeply into edematous mucosa and submucosa (arrows). **C** With less distension of the colon, extensive large bowel ulceration is demonstrated. Note the stellate appearance of some craters and the circumferential involvement. **D** Fluoroscopic spot film of distal ileal loops. Note the lobulated nodules in the air-contrast filled loop with only minimal luminal narrowing (arrows). The adjacent laterally positioned ileum is more involved with luminal compromise, nodularity and ulceration





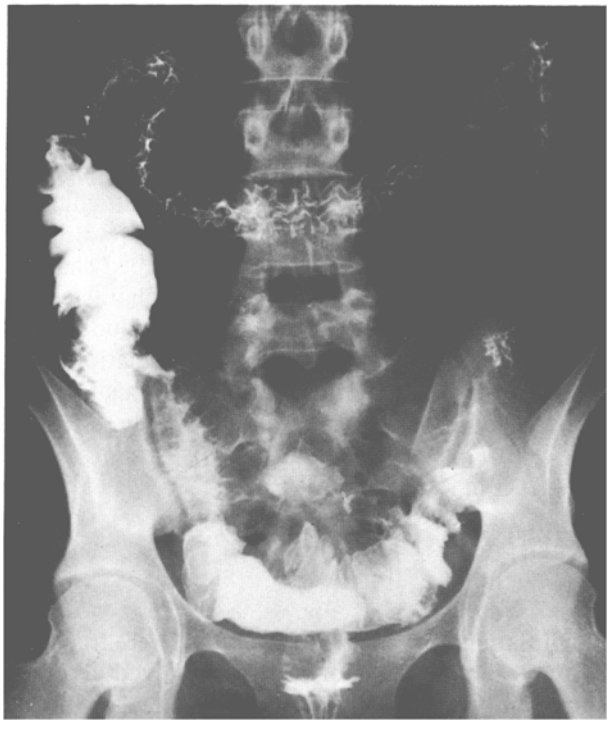


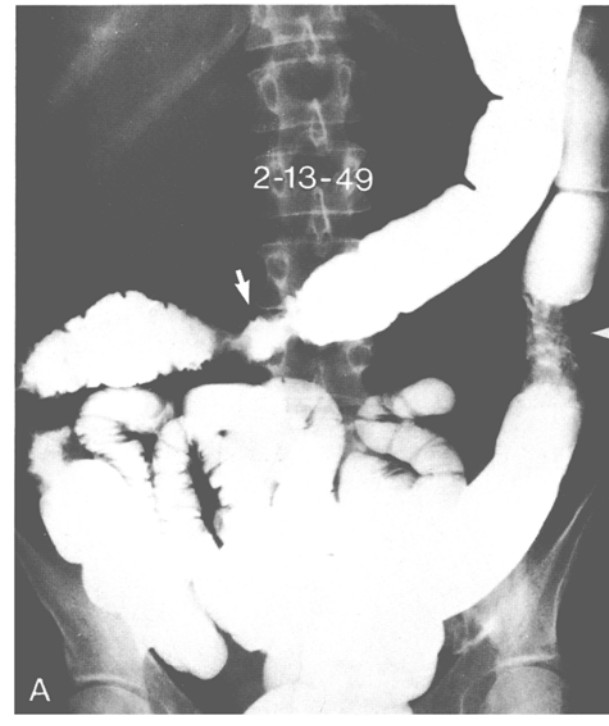
Fig. 3. On this postevacuation barium enema film, extensive ulceration of the terminal ileum is apparent. An irregularly nodular mass encases the ascending colon at the level of the ileocecal valve

Roentgenology

The roentgenologic manifestations of intestinal tuberculosis closely reflect the underlying pathologic changes but are not unique to tuberculous infection. The earliest tuberculous changes are functional and related to superficial and submucosal inflammation. Altered motility of the bowel, both accelerated transit and decreased tone, lead to separation and flocculation of the barium throughout the small bowel. Early infiltration of the mucosa leads to stiffening of the mucosal pattern and nodularity in the more distal part of the small bowel (Fig. 2 A–D). Typical ulcerations are described as linear or stellate, with converging mucosal folds a prominent feature (Figs. 2 and 3).

Hypertrophy is characterized by mass formation, rigidity, and fixation of the bowel. In the small bowel short "hourglass" stenoses are typical. Short stenotic segments in the colon (Fig. 4 A and B) are associated with stiff mucosal surfaces with scattered areas of involvement. Major involvement generally occurs on the cecal side of the ileocecal valve; thus the appearance of the area surrounding the ileocecal valve is the most important for roentgenographic diagnosis (Figs. 5 through 8).

Brombart and Massion [27] performed an extensive evaluation of patients with intestinal tuberculosis



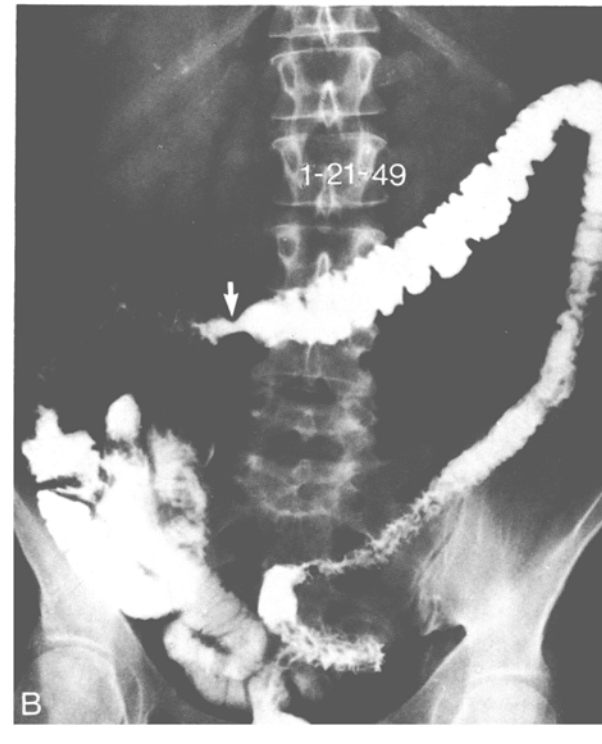
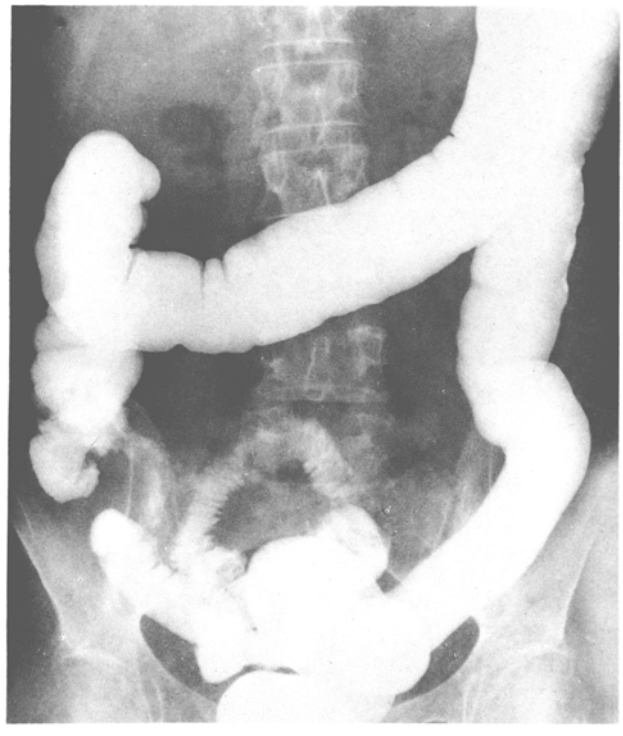


Fig. 4A and B. *Segmental colonic disease.* **A** This patient had ileocecal disease and two sharply demarcated areas of colonic involvement more distally (midtransverse and mid-descending colon) (arrows). **B** *After 5 months of streptomycin therapy* the descending colon lesion healed without scarring. The other areas of involvement remained unchanged



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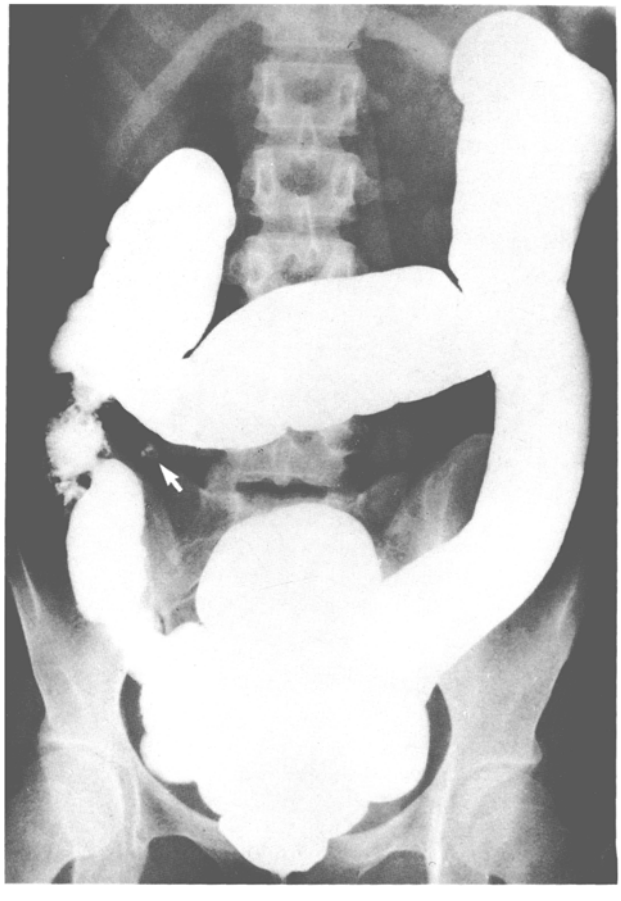

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and Crohn's disease in an effort to define differentiating radiologic changes. The radiographic appearance of enterolcolitic tuberculosis is remarkably similar to that of Crohn's disease except when massive local hypertrophy has produced a large mass with little other change. In these cases, the differential diagnosis between tumor and tuberculosis may be difficult.

Boles and Gershon-Cohen and others [6, 35], as well as Brombart and Massion [27] described changes in the ileocecal valve which are quite characteristic of tuberculous infection. These changes include thickening of the valve lips by granuloma formation (Fig. 2 A–C), wide gaping of the valve itself associated with narrowing of the immediately adjacent ileum (Fleischner's sign) (Fig. 2) and a broad-based mass lesion in the cecum caused by a closed, thickened valve. Numerous other distortions of the ileocecal area, including retraction and shortening of the cecum, localized stenosis of the cecum at the level of the valve (purse-string stenosis) (Fig. 8), and the appearance of an inflammatory mass in the cecum, were frequently found. The finding of a stiff, involved terminal ileum emptying directly through a gaping ileocecal valve into a shortened, rigid, or obliterated cecum forms the classic Stierlin's sign of intestinal tuberculosis.

Segmental colitis (Fig. 4A and B) has also been reported [1–3, 36]; with isolated colonic disease the differential diagnosis then lies between Crohn's disease, carcinoma, and tuberculosis. The pattern is one of segmental inflammation and stenosis with or without inflammatory mass formation.

In most reported cases of tuberculous enterocolitis, the diagnosis was not suspected on the basis of radiologic evaluation prior to operation. In nonendemic areas, most cases were diagnosed as Crohn's disease or carcinoma. Even when the examinations were reviewed, distinguishing characteristics were usually not defined. In areas of the world where tuberculosis comprises a substantially larger percentage of the patient population, such as South Africa [3, 15] and Ibadan [2], the same radiologic findings that suggest Crohn's disease or carcinoma in the United States

Fig. 5. *Ileocolitic tuberculosis.* On this barium enema study, barium refluxed freely through a wide ileocecal valve (Fleischner's sign). The cecum is contracted and surrounded by a mass over which the terminal ileum is draped

Fig. 6. *More advanced disease of cecum and ascending colon.* Concomitant ulcerations in the terminal ileum are masked by the overdistention from free reflux. Note the adjacent calcified mesenteric node (arrow)

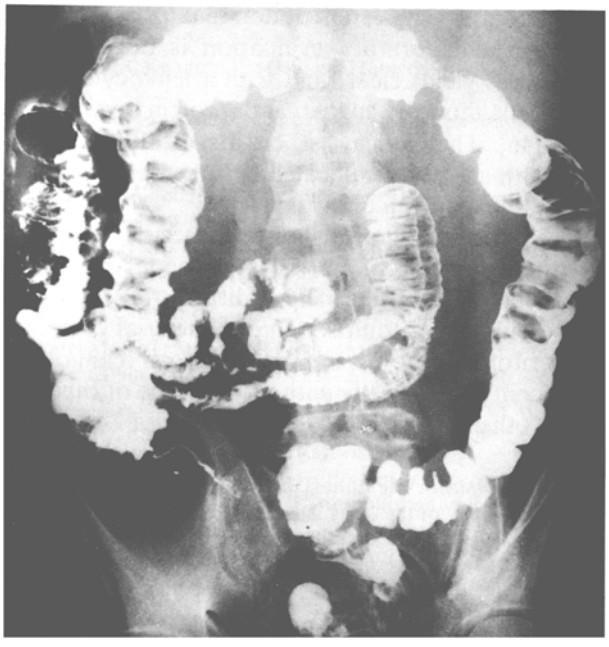

Fig. 7. *Ileocolic tuberculosis, hypertrophic type.* This 66-year-old man presented with a 6-month history of abdominal pain and a normal chest film. The cecum and ileal loops are elevated by a right lower quadrant mass. Their luminal contours are only minimally distorted. The opacified appendix retained a fixed appearance on sequential films. At laparotomy a tuberculous mass encased the cecum and appendix

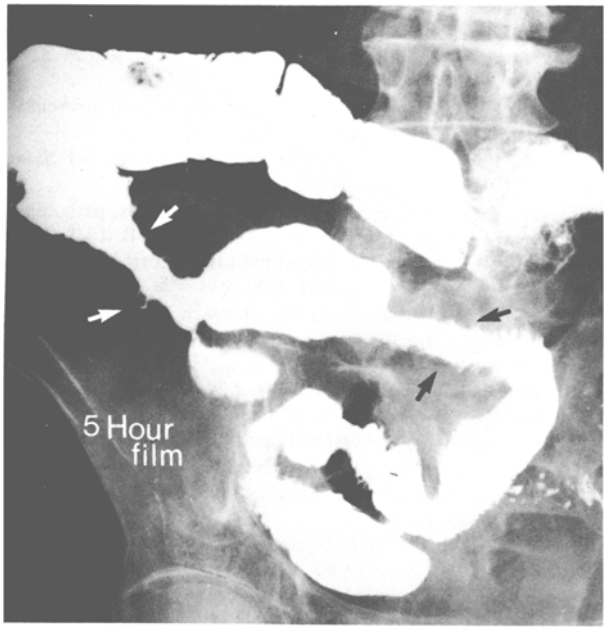

Fig. 8. *Tuberculous enterocolitis of the ulcerohypertrophic type.* This 65-year-old woman presented with a right lower quadrant mass and minimal upper lobe disease. On this 5-hour film from a small bowel series, the cecum and ascending colon are stenosed and shortened. The ileocecal valve is patulous. The ascending colon is ulcerated (white arrows) as well as the distal ileum (black arrows). The ileal loops are sharply angulated and separated indicating thickening of the bowel wall and extension of the inflammatory process into the adjacent mesentery

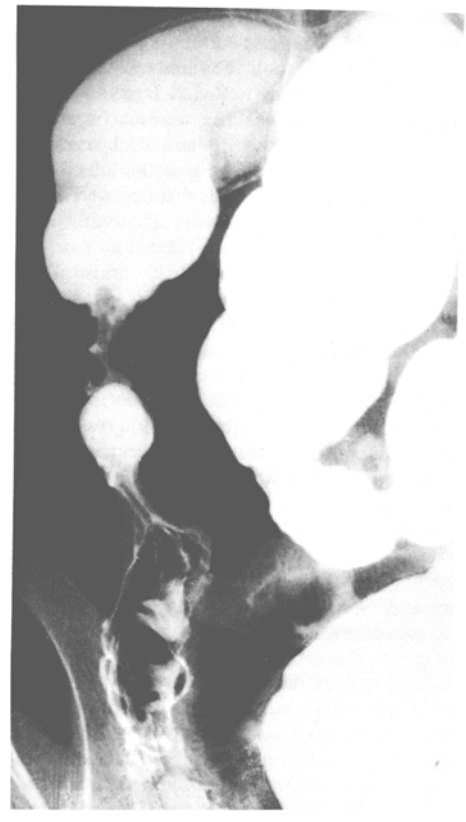

Fig. 9. *Stierlin's sign.* The terminal ileum appears to empty directly into the stenotic ascending colon with nonopacification of the fibrotic, contracted cecum

are considered quite characteristic of tuberculous infection.

Because of the infrequent preoperative diagnosis, it is difficult to establish the reversibility of roentgen changes with therapy. In all likelihood the stage of infection at the time of treatment is critical. There is evidence [24] that the appearance of the bowel treated before extensive scar formation can revert to normal. After formation of dense, collagenous strictures, however, the lesions become irreversible.

Case Report

A 68-year-old woman presented after 4 to 6 weeks of vague abdominal discomfort which had evolved into constant severe lower abdominal pain 2 weeks before admission. She had lost 10 pounds in 1 month, but had no diarrhea, fever, or rectal bleeding.

The initial physical examination was normal except for a 6-cm firm, nontender mass in the right lower quadrant of the abdomen. The admission chest x-ray was normal. Laboratory data showed a moderate iron deficiency anemia. The leukocyte count was 5,700/mm^3 with 80% neutrophils, 1% eosinophils, 10% lymphocytes, and 9% monocytes.

Shortly after admission the patient was given a barium enema (Fig. 2 A–C). The haustra of the transverse colon were asymetrically flattened along the mesenteric side and the cecum was indented medially by an adjacent mass. Barium refluxed freely into the ileum through a gaping ileocecal valve. The terminal ileum as well as two sharply demarcated, more proximal ileal segments were distinctly abnormal, with slight narrowing of the lumen and a nodular, ulcerated mucosa. The involved ileal loops were rigid and separated from adjacent intestinal segments (indicative of bowel-wall thickening). No sinuses of fistulas were identified. An area of marked spasm was observed fluoroscopically in the most proximal segment (a "string" sign). These findings were considered most consistent with regional enterocolitis (Crohn's disease).

She was discharged from the hospital on a trial of medical therapy which included small feedings, salicylazosulfapyridine, and tincture of opium for diarrhea, which had developed during hospitalization. She was readmitted 6 weeks later with progressive anorexia, abdominal pain, diarrhea, vomiting, and sweats (without fever). During this hospitalization a small bowel series was performed (Fig. 2 D). The abnormal findings were confined to the distal ileum and were similar to those of the previous barium enema study. The patient continued to deteriorate despite medical therapy and a laparotomy was performed. Forty-five centimeters of the terminal ileum and 34 cm of the right colon were resected and an ileotransverse colostomy was created.

The serosal surface of the resected specimen was dull, and showed focal areas of hemorrhage. The mesenteric fat was normal. The mucosal surfaces of the ileum, ascending colon, and transverse colon were ulcerated segmentally at several sites. The ulcers had an unusual elongated shape with a shaggy base. The ileocecal valve and adjacent terminal ileum were thickened and stiffened. The gross appearance was consistent with nonspecific inflammatory bowel disease.

On microscopic examination the muscularis was not prominently thickened. Focal ulcerations were found, but there were no sinus tracts. All sections of ileum, colon, and mesenteric nodes contained many caseating granulomas. Many acid-fast bacilli in the granulomas and on the ulcerative lesions established the diagnosis of tuberculous enterocolitis.

Tuberculous Peritonitis

The incidence of tuberculous peritonitis has been reported to be as low as 0.004% to 3.5% of tuberculous infections [37, 38]. The sex incidence is approximately equal [39]; however some series have reported slightly higher incidences for women [38, 40] and children [41]. Primary tuberculous peritonitis with no other identifiable tuberculous foci accounts for 37% to 58% of patients [38, 40, 42]. Secondary involvement of the serous cavity may occur by direct extension or by hematogenous or lymphatic routes. Direct extension comes from the gastrointestinal tract, Fallopian tubes, or a ruptured lymph node. Acute perforation of the gastrointestinal tract with acute tuberculous peritonitis is quite rare but does occur [43].

Tuberculous peritonitis usually has a chronic presentation with three forms generally recognized. There is the "wet" type with ascites or loculated fluid; a "dry" form with tuberculomas and adhesions creating a plastic abdomen; and a "fibrotic-fixed" form that appears as an abdominal mass [40, 42, 44, 45]. The laboratory evaluation is not specific; paracentesis yields clear fluid with a leukocyte count of 250 cells/mm^3 or higher and protein over 2.5 g/100 ml [46]. The clinical association of alcoholic cirrhosis with ascites complicated by tuberculous peritonitis is well known [46].

Plain roentgenograms show nonspecific ileus with or without ascites; calcified lymph nodes may also be present [41]. The principal differential diagnosis of tuberculous peritonitis includes bacterial or chemical peritonitis; however, the diagnosis cannot be made roentgenologically without evidence of tuberculosis in other sites [47].

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