Abdominal Tuberculosis: CT Evaluation

MANIFESTATIONS of tuberculosis within the abdomen are protean. The gastrointestinal tract, lymphatic system, peritoneum, and solid viscera are subject to varying degrees of involvement: alone, in combination, or in association within extraabdominal disease. Hence, clinical presentations of abdominal tuberculosis are extremely varied, and diagnosis may be obscured by lack of radiologic evidence of pulmonary tuberculosis (1, 2). Abdominal involvement is not rare, however, and complicates pulmonary tuberculosis in 6%-38% of patients (3). Computed tomography (CT) offers the advantage of examining the range of abdominal involvement in a single examination, but only two small series have been reported previously (4, 5). CT findings in 27 patients are presented here.

MATERIALS AND METHODS

The CT scans of 27 patients with proved intraabdominal tuberculosis examined between July 1981 and October 1984 at six New York City hospitals were reviewed retrospectively. No patients with disease limited to the musculoskeletal system or genitourinary tract were included. Twenty-three patients were male, and four were female. Their ages ranged from 21 to 68, with an average age of 37 years. Seventeen patients were white, eight were black, and two were oriental. Most patients had one or more risk factors for the development of tuberculosis. Ten had a history of intravenous drug abuse, eight were alcoholics, and eight suffered immunosuppression as a result of acquired immunodeficiency syndrome (AIDS) (six cases), steroid therapy (one case), or cirrhosis (one case). Infection was limited to the abdomen in five patients but was present also in the lung or other extraabdominal sites in the remainder. The causative agent was Mycobacterium tuberculosis in 23 patients and M. avium-intracellulare in four patients with AIDS. The diagnoses had been proved by microbiological or pathologic analysis of abdominal tissue (19 cases) or by improvement seen on abdominal CT studies following antituberculous chemotherapy prescribed because of the documentation of tuberculosis in extraabdominal sites (seven cases).

Scans were performed on commercially available equipment using 10-mm collimation at 10-15-mm intervals. Oral and intravenous contrast media were administered whenever possible. Clinical charts and other radiologic studies were reviewed in each case.

RESULTS

Adenopathy was the most common finding on CT study and was present in 24 patients. In 13 patients there was no associated gastrointestinal or peritoneal involvement, but generalized adenopathy or an abnormal chest radiograph evidenced the presence of tuberculosis elsewhere. In three patients, abdominal lymph nodes were the only sites involved. Enlarged nodes were found in the periaortic/pericaval retroperitoneum, mesentery, omentum, peripancreatic and porta hepatis regions, and groin (Fig. 1). Most patients had involvement of more than one compartment. Adenopathy patterns varied widely, including increased numbers of nodes of normal size, scattered mildly enlarged nodes, localized clusters of several enlarged nodes, and large conglomerate masses (Fig. 1d). Peripan-
Figure 1

Tuberculous lymphadenopathy.
a. Contrast material enhanced scan at the level of the portal vein (PV) shows low-density adenopathy with ring enhancement in the peripancreatic and retroperitoneal compartments (arrows).
b. Multiple enlarged discrete soft-tissue density mesenteric lymph nodes (N) are associated with dilated small-bowel loops with thickened folds (SB). Note relative lack of retroperitoneal involvement.
c. Contrast material enhanced scan at the kidney level demonstrates multilocular low-density retroperitoneal masses with ring enhancement (M) and soft-tissue density mesenteric adenopathy (*).
d. Heterogeneous soft-tissue density mass in left upper quadrant (M) proved to represent a mesenteric tuberculous abscess. Note lack of retroperitoneal involvement.

Celiac or porta hepatis adenopathy was demonstrable in 21 patients (Fig. 1a), mesenteric/omental adenopathy in 20 (Fig. 1b), and retroperitoneal adenopathy in 19 (Fig. 1c). A striking finding was the relative severity of the adenopathy in the mesenteric, omental, and peripancreatic locations compared with the degree of retroperitoneal involvement. The retroperitoneum was the dominant site of adenopathy in only three patients, while mesenteric/omental adenopathy was dominant in eight, and peripancreatic adenopathy was dominant in seven. The remaining six patients had diffuse abdominal adenopathy but no particular site of dominant involvement.

The centers of enlarged lymph nodes were of low density in ten patients (Fig. 1a and c). Administration of intravenous contrast material accentuated this finding by enhancing the inflammatory rim surrounding the caseous center. Discrete abscesses containing low-density material were drained surgically from the mesentery (two cases), porta hepatitis (two cases), and uterine adnexa (one case) (Fig. 2a and b). Abscesses of soft-tissue density material were found in the mesentery (one case) (Fig. 1d) and peripancreatic region (two cases) (Fig. 4c and d). Despite the considerable bulk of adenopathy in several patients, lymph-node masses were not responsible for obstruction of the urinary, biliary, or gastrointestinal tracts in any patient.

Splenomegaly, defined as a span 13 cm or greater (6), was a prevalent finding (18 cases). In only three patients were discrete intrasplenic masses identified by CT (Fig. 2c). Hepatomegaly, defined as a span greater than 20 cm (7), was a less common finding (ten cases). No patient had hepatomegaly without associated splenomegaly. In only one patient did a CT scan demonstrate intrahepatic masses (Fig. 3). Liver biopsy results were positive for caseating granulomatous disease in three patients whose CT scans showed enlarged but homogeneous livers and in one patient with a liver that appeared as nor-
Hepatic tuberculosis. Multiple low-density intrahepatic masses are associated with adenopathy (N) in the porta hepatis. The spleen was enlarged and showed similar masses.

Figure 2

Tuberculous peritonitis.

a. Contrast material enhanced scan at level of kidneys shows relatively high-density ascites (A) and marked accentuation of mesenteric vessels with mesenteric adenopathy (arrows). Retroperitoneum is relatively uninvolved.

b. In the same patient, scan through the pelvis (B = bladder, C = colon) demonstrates bilateral tubo-ovarian abscesses (A), showing partial low density on the right.

c. Scan at the renal level (K) without contrast material shows enlarged spleen with low-density mass (S) within it and voluminous ascites complicated by soft tissue standing and small irregular densities.

Figure 3

Ascites was present in seven patients, three of whom also had pleural effusion. The fluid was voluminous and diffusely distributed throughout the peritoneal cavity in five patients and was loculated in small scattered pockets in two. Tuberculous salpingitis apparently preceded diffuse peritonitis in two patients (Figs. 2a and b), and one patient had preexisting cirrhosis and ascites (Fig. 2c). Fluid-density measurements ranged from 20 to 45 Hounsfield units (HU) and averaged 30 HU for the seven patients. Peritoneal enhancement following administration of intravenous contrast material was seen in three patients. In all patients with ascites, there was abnormal thickening of soft tissue and nodularity associated with the peritoneal surfaces, mesentery, and omentum (Fig. 2c).

CT scans demonstrated ileo-cecal tuberculosis in one patient (Fig. 4a and b), diffuse small-bowel involvement in another patient (Fig. 1b), and a peripancreatic tuberculous abscess associated with a duodenal fistula in two patients (Fig. 4c and d). In each case, there was adenopathy adjacent to the involved bowel segment. In one patient with endoscopically proved colonic tuberculosis, the CT scan demonstrated diffuse abdominal adenopathy with a mesenteric predominance, but the sites of colonic in-
Gastrointestinal tuberculosis.

a. Scan through lower abdomen shows thickening of cecal wall (C) and an adjacent mass of conglomerate adenopathy (arrows).
b. In the same patient, small-bowel series demonstrates ulcerohypertrophic ileo-cecal tuberculosis.
c. A large, irregular, soft-tissue peripancreatic mass (M) deviates the stomach (S) anteriorly. A focus of extraluminal air (arrow) is seen medial to the widened duodenal sweep (D).
d. Hypotonic upper gastrointestinal study shows spiculation of the medial aspect of the second and third duodenal segments (arrows) and outlines extravasation into the peripancreatic abscess (curved arrows).

DISCUSSION

Tuberculosis within the abdomen potentially can affect the gastrointestinal tract, peritoneum, lymph nodes, liver, or spleen. Possible mechanisms in the development of abdominal disease include ingestion of infected material such as sputum or milk, or hematogenous dissemination to the abdominal viscera and lymphatic system from a distant focus, usually in the lung. Prior to the advent of CT scanning, radiologic evaluation was confined largely to studies of the gastrointestinal tract with contrast media. Extramucosal disease in lymph nodes, peritoneal involvement, or involvement of the liver and spleen could not be evaluated directly by this method. Even before the era of chemotherapy, however, it had been shown that up to two-thirds of patients with abdominal tuberculosis had lymphadenopathy or peritoneal disease without gastrointestinal involvement (8, 9). Contrast material studies alone, therefore, have been of limited utility in detecting and locating the full range of abdominal disease. Furthermore, the virtual disappearance of bovine tuberculosis in the United States and the general availability and effectiveness of antituberculous chemotherapy have resulted in a decreased incidence of intestinal tuberculosis caused by ingestion. Concurrently, tuberculosis has become less readily recognized, perhaps because of its decreased incidence or a changing pattern of clinical and radiological presentation since the advent of chemotherapy (10–12). Today, immunodeficiency associated with either alcoholism, intravenous drug abuse, diabetes, cancer, steroid therapy, and acquired states (e.g., AIDS) are prominent risk factors (13), and indeed, in our series only 20% of the patients did not have at least one of these risk factors.
Abdominal lymphadenopathy was the most common manifestation of tuberculosis on CT scans in our series and was present in nearly all (23 of 26) patients. Of course, adenopathy within the abdomen is found in a multitude of pathologic conditions, benign and malignant; but in our patients, tuberculous adenopathy exhibited several characteristic but not pathognomonic features. There was a striking tendency for the involvement of mesenteric and peripancreatic node groups to accompany and usually overshadow the extent of retroperitoneal involvement (Fig. 1a, b, and d). While a previous study (4) did describe omental involvement, the finding of mesenteric, porta hepatica, and peripancreatic adenopathy was not emphasized. Lymphangiographic studies in patients with abdominal tuberculosis have noted a gradient of retroperitoneal involvement, most severe in the upper abdomen (14), and mesenteric adenitis and peripancreatic involvements are well-recognized manifestations of abdominal disease (9, 15, 16). Perhaps this distribution reflects the lymphatic drainage of sites in the small bowel and liver that had been seeded hematogenously but may not have developed frankly clinically apparent infection. Iliac, lumbar, and lower abdominal node groups are less commonly involved because they drain the lower extremities, pelvic viscera, and distal colon—areas less likely to be affected by hematogenous tuberculous dissemination, except when infection is overwhelming.

In our series, lymph node masses, even when large, did not cause obstruction of the biliary ducts, ureters, or bowel. It should be noted, however, that obstructive jaundice secondary to adenopathy in the porta hepatis, hepatoduodenal ligament, and peripancreatic area has been reported as a rare complication of tuberculosis in North American patients (17, 18) and is apparently less rare in the Filipino population (19).

Low-density centers within tuberculous abdominal lymph nodes have been noted in previous reports (4, 5) and were identified in 40% (ten of 24) of our patients (Fig. 1a and c). This finding is not diagnostic, however, and may be found occasionally in metastatic malignancy, lymphoma (especially if treated), pyogenic infection, and Whipple disease (20). In tuberculosis, presumably the low-density nature of the involved nodes is secondary to caseation necrosis. In our series, however, there was no strong correlation between the site or extent of adenopathy and the density of the involved nodes. The largest mesenteric abscess (10 cm) was of soft-tissue density (Fig. 1d), while the smallest (2 cm) was of low density.

Gastrointestinal tuberculosis can result from ingestion, extension from the lymphatic system, or hematogenous dissemination; each of these pathophysiologic mechanisms is represented in our series. One patient whose chest radiograph was within normal limits had a history of recent travel to an area endemic for bovine tuberculosis. This patient had ulceronecrotic ileo-cecal disease, presumably caused by ingestion of infected milk (Fig. 4a and b). In two patients, massive peripancreatic tuberculous adenopathy eroded into the duodenum, resulting in fistula (Fig. 4c and d). In a patient with AIDS, diffuse bowel involvement (Fig. 1b) was associated with hematogenous dissemination to the lymph nodes, bone marrow, central nervous system, liver, and spleen. Though CT was of diagnostic importance in demonstrating ancillary findings such as adenopathy, barium studies were essential for further characterizing the nature of the bowel pathology in each patient.

Tuberculous peritonitis, as seen in seven of our patients, is a well-described manifestation of abdominal disease (8, 9, 15, 21-23) but occurs in less than 4% of patients with pulmonary tuberculosis (1, 2). Peritonitis and ascites may be associated with widespread abdominal and extraabdominal disease, as in three of our patients. Tuberculous salpingitis progressing to diffuse peritonitis, a known mechanism of spread (2), was evident in two of our patients (Fig. 2a and b). The association of cirrhosis and tuberculous peritonitis has also been described (20) and was present in one patient (Fig. 2c). In the seventh patient, tuberculous peritonitis appeared to develop primarily, as no pulmonary or other abdominal site of infection was evident.

In these seven patients, ascitic fluid density ranged from 20 to 45 HU (average, 30 HU). It has been suggested that high-density ascites may be characteristic of tuberculosis (4, 5). Theoretically this can be explained by the high protein and cellular contents in a tuberculous effusion, particularly when cell-mediated immunity has developed. We emphasize, however, that tuberculous ascites may also be near water in density, perhaps reflecting an earlier transudative stage of immune reaction. The problems inherent in using CT density measurements as absolute standards of reference should be recognized (24), and this is especially evident when “measuring” small areas such as pockets of ascites. Moreover, as in our cases, ancillary findings of tubo-ovarian abscesses, adenopathy, peritoneal enhancement, or a “dirty” appearance to the mesentery indicate a complex nature to the ascites and should suggest the diagnosis of tuberculous peritonitis. Recognition of these features proved more relevant than merely measuring ascitic fluid density or attempting to classify peritoneal involvement into the classic wet, dry, or fibrinotic-fixed forms (1, 2, 21).

Tuberculosis of the liver or spleen is reportedly uncommon, except in association with miliary dissemination (2, 9). Though hepatic involvement by tuberculosis reportedly tends to be diffuse (2), the macronodular or pseudotumor forms, as found in one of our patients (Fig. 3), is rarely seen as well (25). The high incidence of splenomegaly (18 of 27 cases) and hepatomegaly (ten of 27 cases) in our series is higher than in previous series (8, 9, 14, 15), and four of our patients did not appear to have miliary disease or even disease outside the abdomen. Our findings may in part be related to the high incidence of coexisting alcoholism and intravenous drug abuse in our patients. Interestingly, no patient had hepatomegaly without splenomegaly, and in many cases the enlargement was mild, perhaps indicating that enlargement of the liver or spleen in some cases represented a nonspecific response to infection.

Patients with AIDS presented special problems. There have been several recent reports of disseminated infection with M. avium-intracellulare complex in these patients (26-29), as was seen in four of our cases. Yet two of our patients with AIDS were infected with typical M. tuberculosis. Prior to the AIDS epidemic, disseminated infection with M. avium was exceedingly rare (30-32). In our series each patient with M. avium infection had been chronically ill with AIDS-related diseases and exhibited multisystem involvement. However, distinguishing M. tuberculosis from M. avium infection was not possible by CT criteria alone. In all AIDS patients, differential diagnosis must include the Lymph Node Syndrome, Kaposis sarcoma, lymphoma, and other opportunistic infections as well as typical and atypical tuberculosis (33); aggressive biopsy, smear, and culture studies of relevant tissue are necessary for an accurate diagnosis (26, 27, 34-37).

In any patient, diagnosis of intraabdominal tuberculosis is difficult because symptoms are vague, signs non-
specific, and clinical tests often not conclusive. Though the presence or a history of pulmonary disease may be suggestive, nearly one-fifth of our patients (five of 27) had no evidence of extraabdominal disease. Recovery of mycobacteria from the stool may not necessarily indicate gastrointestinal infection. Results of purified protein derivative skin tests may be falsely positive or negative. Results of liver biopsy testing are usually negative in the absence of miliary disease (2). The importance of reaching the correct diagnosis, however, is formidable, as untreated abdominal tuberculosis carries a 50% mortality rate (8). Laparotomy should be avoided if possible, and even those patients requiring surgery benefit from preoperative antituberculous chemotherapy (9).

Unfortunately, there are no CT findings, alone or in combination, that are pathognomonic of tuberculosis. Abdominal adenopathy, predominantly in the mesenteric and peripancreatic areas, is a suggestive finding. Low-density centers within the enlarged nodes are also a characteristic feature. Ascites—perhaps relatively high in density, loculated, or accompanied by peritoneal enhancement or adenopathy, and associated with mesenteric and omental soft-tissue changes—is suggestive of tuberculous peritonitis. Bowel abnormality associated with adjacent adenopathy must also raise the possibility of gastrointestinal tuberculosis. And most important, to make the correct diagnosis, an index of suspicion must be maintained, especially in those patients at increased risk.

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Send correspondence and reprint requests to: Donald H. Hulnick, M.D., Department of Radiology, New York University Medical Center, 560 First Avenue, New York, NY 10016.

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