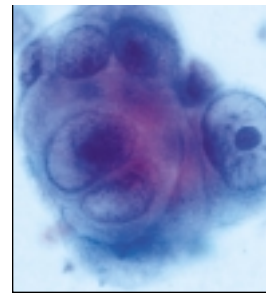


FEVER AND AN ABNORMAL ABDOMINAL COMPUTED TOMOGRAPHY SCAN

(continued from page 56)



Answer

2. Tuberculous adenitis

Percutaneous fine-needle aspiration of the soft-tissue mass was performed under CT guidance. Multiple passes with 22-gauge Greene and Westcott needles yielded samples of lymph node that were partially replaced by necrotizing granulomata. A few acid-fast bacilli (AFB) bacteria were present. These results are consistent with *Mycobacterium tuberculosis* infection. Serologic tests demonstrated a mixed population of normal B and T cells and were negative for malignant lymphoma.

Discussion

Although thrombosis of the portal vein can have a similar appearance to the rim-enhancing lesion seen on the CT scan, the location of this lesion, as well as visualization of a normally opacified portal vein more anteriorly, excludes portal vein thrombosis as the correct diagnosis. Pseudocysts from chronic pancreatitis may have a similar appearance, but no inflammatory changes of acute pancreatitis are demonstrated. Of the two remaining choices, which fall into the differential diagnosis of periportal/peripancreatic lymphadenopathy, tuberculous adenitis



Fig 2. — Axial CT scan through the abdomen demonstrates multiple enlarged lymph nodes in the periaortic and aorto-caval region due to lymphoma (arrows). This extensive lymphadenopathy demonstrates homogenous attenuation without peripheral rim enhancement that is more often seen in untreated lymphoma.

tis is the more likely diagnosis. Tuberculous lymphadenopathy commonly shows peripheral enhancement and a multilocular appearance compared with lymphomatous adenopathy, which demonstrates homogeneous attenuation and enhancement. However, this is not a pathognomonic finding (Fig 2).¹

M. tuberculosis remains a major medical issue, with global prevalence estimated at 32% (1.86 billion people).² Although the predominant form of tuberculosis is pulmonary disease, extrapulmonary involvement is common, largely due to hematogenous spread. Secondary infection can result in, but is not limited to, granulomatous disease of the meninges, eyes, bones and joints, skin, the genitourinary tract, and the abdomen. Secondary spread by ingestion or extrapulmonary primary infection is less common. Abdominal tuberculosis can affect the gastrointestinal tract, peritoneum, lymph nodes, spleen, liver, and adrenal glands. Lymphadenopathy is the most common manifestation of abdominal tuberculosis. Mesenteric and peripancreatic groups are involved most often, reflecting the lymphatic drainage of commonly affected sites in the small bowel and liver. Periportal tuberculous adenitis is a rare entity.³ Furthermore, reported complications secondary to tuberculosis lymphadenopathy such as obstructive jaundice⁴ or thrombotic portal hypertension^{5,6} are few.

Anatomic distribution and specific enhancement patterns of

lymphadenopathy seen on contrast-enhanced CT scan may be useful in differentiating between tuberculosis and untreated lymphomas of the abdominal lymph nodes. Abdominal CT scan with contrast provides a comprehensive overview of the structures involved in abdominal tuberculosis.⁷ Tuberculosis peritonitis results in either "wet" type (higher attenuation acidic fluid that is loculated or has debris) or "fibrotic-fixed" type (matted loops of bowel and large omental masses). Gastrointestinal involvement is generally found in the ileocecal region where there is an abundance of lymphoid tissue and relative stasis. With edema, typical appearance is asymmetric or circumferential thickening of the bowel wall; with lymphadenopathy, typical appearance is enlargement of the ileocecal valve with lymphadenopathy. Barium studies are more sensitive for detecting incompetence of the ileocecal valve or increased motility and for delineating luminal changes such as ulceration, stenosis, and mucosal-fold irregularity.

Involvement of the pancreas, although rare, appears as hypodense, necrotic areas that appear hypoechoic by ultrasound. Hematogenous spread to renal glomeruli manifests itself in several ways, including calculi, calcifications of part or all of the kidney, or "putty-like" homogenous calcifications. There may also be resultant scarring or focal or generalized hydronephrosis. Hepatosplenic disease results in enlargement of these organs. Multiple low-attenu-

ation lesions or the rare mass-appearing tuberculoma can be demonstrated on CT scan. The latter may have septations and irregular borders and, with progression, may enhance with contrast peripherally and eventually contain calcifications. On CT scan, these different features could represent evolving pathologic stages of the disease, with early non-caseating granulomas and subsequent caseating necrosis¹ density nodes at other sites, etc.

In contrast to the atypical radiographic findings of pulmonary tuberculosis in patients with acquired immunodeficiency syndrome (AIDS), lymphadenitis usually appears the same as in non-AIDS patients. However, 25% of cases show large, low-attenuation, nonenhancing nodes, likely due to a poor inflammatory response secondary to immune status.⁷

Although CT scans and other examinations such as barium studies are useful in assessing abdominal pathology in tuberculosis, findings are not always specific, and a histopathologic or bacteriologic confirmation is often required. Also, chest radiographs can often be within normal limits, and intradermal skin testing is negative in the anergic patient. When noninvasive studies fail to confidently characterize the lesion, fine-needle aspiration is useful in the workup of abdominal adenitis. Either CT- or ultrasound-assisted fine-needle aspiration offers a safe method of achieving a diagnosis in a patient with suspected abdominal tuberculosis who present with radiolog-

ically demonstrable but nonpalpable lesions, especially those involving lymph nodes and spleen. When this does not result in a definitive diagnosis, laparoscopic or surgical biopsy may be required.

Conclusions

This presentation of disease via an isolated, abdominal soft-tissue mass on a CT scan of a patient with fever of unknown origin but no apparent clinical findings of pulmonary tuberculosis infection emphasizes the importance of including tuberculosis in the differential diagnosis of abdominal lymphadenopathy.

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