while in the "rapid destruction" pattern, platelet life span was extremely short, resulting in a rapid early rise and a slow progressive later increase in splenic activity, as well as progressively increasing hepatic activity. In the "nonspecific" pattern, there were identical time-activity curves for the spleen and liver (both reaching a plateau early and then remaining unchanged over time) with a nearly equal distribution of counts between the spleen and liver. The results in our two patients appear to best correspond with the "rapid destruction" pattern, at least with regard to the time course of uptake in the liver.

CONCLUSION

Indium-111-platelet scanning may have a role in assessing the platelet transfusion refractory BMT patient for possible splenectomy, similar to its use in patients with ITP. While demonstration of in vivo platelet kinetics may assist in reaching a decision regarding splenectomy, consideration of the risks associated with persistent thrombocytopenia versus those of the surgical procedure itself will also be important. In addition, imaging protocols and threshold values specific for studies in BMT patients (using donor platelets) must still be defined before platelet scintigraphy can be routinely used as a standard examination for guiding therapeutic decisions.

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Indium-111-Leukocyte Imaging: A Case of Peritonitis Mimicking Inflammatory Bowel Disease

Lisa S. Gobar, Robin Graham and Katherine A. Harrison

Departments of Radiology and Surgery, Creighton University Medical Center, and Department of Radiology, University of Nebraska Medical Center, Omaha, Nebraska

Leukocytes labeled with ^{99m}Tc-HMPAO and ^{111}In have been used extensively in imaging inflammatory disorders, including inflammatory bowel disease (IBD), which has the appearance of tubular bowel activity. Peritonitis is inflammation of the serosal surfaces lining the peritoneal cavity which envelopes the bowel, giving a pattern of diffuse abdominal uptake on imaging. We present a case of an elderly man with surgically and pathologically confirmed peritonitis whose preoperative leukocyte scan mimicked the findings of IBD. Our findings suggest that diffuse peritonitis can mimic IBD on an ^{111}In-leukocyte scan.

Key Words: peritonitis; indium-111 leukocyte; inflammatory bowel disease

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Peritonitis is an acute inflammation of the serosal surfaces lining the peritoneal cavity which also envelops the bowel. Involvement is usually diffuse, a finding that has been demonstrated on leukocyte scintigraphy (*1*). Clinicians investigating inflammatory and infectious disorders rely on leukocytes labeled with ^{99m}Tc-HMPAO and ^{111}In for diagnosis and treatment decisions. Common indications include evaluation of the fever of unknown origin, search for infection in known fluid collections and inflammatory bowel disease (IBD) (*1–4,6–11*). Abscesses are generally focal areas of leukocyte accumulation, and IBD is generally seen as tubular activity corresponding to segments of the small or large intestine. We present a case of pathologically confirmed peritonitis with the scintigraphic appearance of IBD.

CASE REPORT

A 73-yr-old man presented with malaise, weight loss, back pain and watery diarrhea for several weeks duration. The patient had a past medical history of steroid-dependent asthma, stroke, hypertension and arthritis. On presentation, he had mild abdominal tenderness, an elevated white blood cell count of 15.6 with 50% bands and metabolic acidosis. Iron deficiency anemia (hematocrit

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For correspondence or reprints contact: Lisa Suzanne Gobar, MD, Department of Radiology, Creighton University School of Medicine, 601 N. 30th St., Omaha, NE 68131.

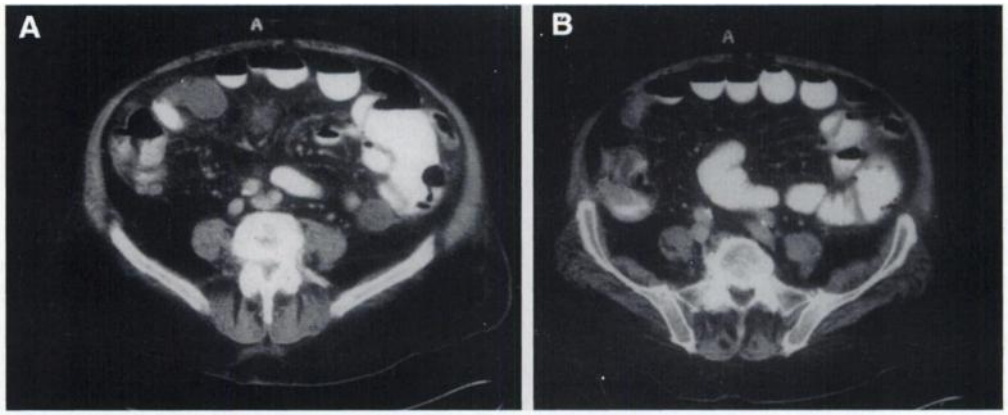

FIGURE 1. Two CT images at different levels of the pelvis demonstrate LLQ fluid collection/abscess (A) and absence of small intestine wall thickening (B).

29) and renal insufficiency (creatinine 3.5) were also present. The following day he developed a fever of 100.2° F and abdominal pain. There were no peritoneal signs.

Stool (fecal leukocytes, C. difficile toxin, giardia antigen, anaerobic and aerobic cultures and guaiac) and urine studies were negative. Amylase was normal; an erythrocyte sedimentation rate was not performed. A radiograph of the abdomen demonstrated dilated loops of small bowel consistent with an adynamic ileus or enteritis. Sonography found an enlarged gallbladder, atrophic kidneys and loculated fluid collections. A CT scan of the abdomen revealed multiple intra-abdominal fluid collections, particularly in the LLQ, mid-abdomen and perirectal regions. There was no intestinal mucosal thickening (Fig. 1) or obstruction, though an ileus was seen. The abnormalities seen on ultrasound were confirmed. There was no aneurysm, though aortic calcifications were present. At this point, the fluid collections were assumed to be infected; the abscesses were felt to be localized without peritonitis. Hydration and intravenous antibiotics were administered. The patient remained stable and free of peritoneal signs, but his white blood cell count continued to rise; consequently, 4 days later an ^{111}In-leukocyte study was performed.

Sixty milliliters of the patient's blood were used to label the white cells with 19.6 Mbq of ^{111}In; the patient's white cell count on the day of the labeling was 18.8×10^3/ml. Eighty and three tenths percent of the indium used for labeling was bound to the leukocytes. Twenty-four hours after reinjection, planar anterior and posterior views of the chest, abdomen and pelvis were performed. Localized foci of intense activity were seen in the perirectal region (not shown), mid-abdomen and LLQ, corresponding to the fluid collections seen on CT, consistent with abscesses. Focally increased activity was seen in the left mid-abdomen, which did not correlate to fluid collections on the CT of 2 days previous. Abscesses had either developed in the interim, or the activity on the leukocyte scan could be due to overlapping intestine. Very little activity could be identified in the liver, spleen or bone marrow, as the preponderance of leukocytes were localizing in the infection sites in the abdomen. No potential sources of swallowed leukocytes were identified, such as sinusitis. Of particular interest, markedly increased activity in a broad curvilinear of tubular pattern suggestive of small intestine was seen throughout the abdomen (Fig. 2). This activity appeared to be within the mucosa diffusely or in the lumen. The differential diagnosis for the diffuse small intestinal activity was felt to be ischemic bowel disease versus inflammatory bowel disease (most likely Crohn's). Images were very similar at 48 hr. The patient was still free of peritoneal signs and had only mild abdominal discomfort.

The following day, the patient's condition deteriorated, and he was taken to surgery. There were dense adhesions throughout the small intestine. An inflammatory mass in the left upper quadrant was resected with a simultaneous hemicolectomy. Pathologic examination of the colon demonstrated diverticulosis without diverticulitis (acute inflammation was not identified in the diverticulum which were examined microscopically), as well as focal acute peritoneal inflammation. Cultures of the abscesses and peritoneal fluid grew bacteroides fragilis and anaerobic diptheroids. There was no evidence of necrotic bowel or inflammatory bowel disease in the colon. Subsequently, the patient required re-exploration, with resection of 75 cm of small intestine. Pathologic examination revealed a normal mucosal surface with inflammation of the serosal surface and mesentery, consistent with peritonitis. Again, there was no evidence of necrotic bowel or inflammatory bowel disease.

DISCUSSION

Labeled leukocyte studies are efficacious in the diagnosis, staging and follow-up of patients with IBD (*1,3,8–11*), which is diagnosed on leukocyte scans by tubular activity conforming to the bowel. In the evaluation of diverticulitis, diagnosis is made

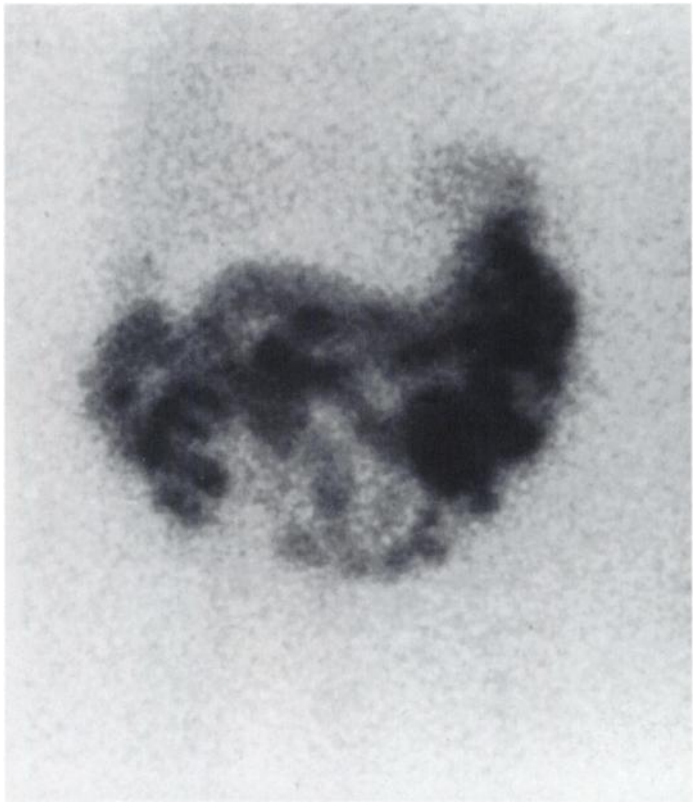

FIGURE 2. Anterior planar ^{111}In-white blood cell image of the abdomen at 24 hr demonstrates more focal abscesses of the LLQ and mid-abdomen surrounded by intense tubular small intestine activity, later proven to be diffuse peritonitis.

by visualization of a small focus of activity, usually in the LLQ (*1,8*), representing a single inflamed diverticulum or frank abscess formation. Diverticulosis without diverticulitis, a condition present in our case, has no leukocyte accumulation. The leukocyte scintigraphic findings of acute peritonitis have been described as diffuse activity throughout the abdomen (*1*). Chandramouly et al. (*5*) described a case of loculated purulent peritoneal fluid collections, which were seen as foci of intense activity in a patient with peritonitis. Our case is unique in that the pattern mimics the findings of ischemic or IBD.

This case report is classic for acute diverticulitis with abscess formation, in both the clinical manifestations and the radiographic and scintigraphic findings of the abscesses. Initially the abscesses appeared to be well-contained and free of peritonitis. Subsequently, the patient developed peritonitis clinically, which was documented during surgical and pathological examination. There is no pathologic evidence of ischemic bowel, or of Crohn's disease, ulcerative colitis or other inflammatory processes of the bowel wall. Multiple diverticuli were seen in the colon specimen, but none of those examined microscopically were acutely inflamed. The only pathologic findings that would explain the tubular small intestine activity seen on the leukocyte scan is peritonitis.

Although causes of false-positive leukocyte scintigraphy are numerous and well documented (*2,3,9,10*), the tubular activity on the leukocyte scan in our case is unlikely to represent a false-positive. False-positives with the tubular pattern are associated most frequently with technetium-labeled leukocytes, and appear as mild uptake throughout the lumen of the intestine. It is generally accepted that this activity is intraluminal radiopharmaceutical secreted through the hepatobiliary system (*3*). For this reason, leukocytes labeled with ^{111}In have been recommended over technetium-labeled for identifying abdominal abscesses, as was done in the case presented. (Alternatively, very early imaging at 30–60 min after injection has been recommended.) Intraluminal isotope has not been documented in ^{111}In studies; false-positives in ^{111}In studies in a tubular pattern are usually caused by leukocytes from another source of infection, such as sinusitis, which are then swallowed. This activity is usually faint and it should move between the 24 and 48 hr images; neither is true of our study. Nor was there any evidence for a gastrointestinal bleed that may have resulted in either unbound indium or labeled leukocytes in the intestine. Fecal leukocyte studies were negative. Finally, there was no pathologic, laboratory or surgical evidence that the abscess communicated with the bowel—if it had it would have communicated to the colon, whereas the tubular activity was in the small intestine.

CONCLUSION

A leukocyte scan obtained preoperatively demonstrated a left lower quadrant inflammatory mass/abscess confirmed at surgery. The dramatic leukocyte accumulation in a pattern that follows the contours of the small intestine is explained by the surgical and pathologic findings of peritonitis. Our findings suggest that a diffuse peritonitis may present as tubular bowel activity on an ^{111}In-labeled leukocyte study.

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