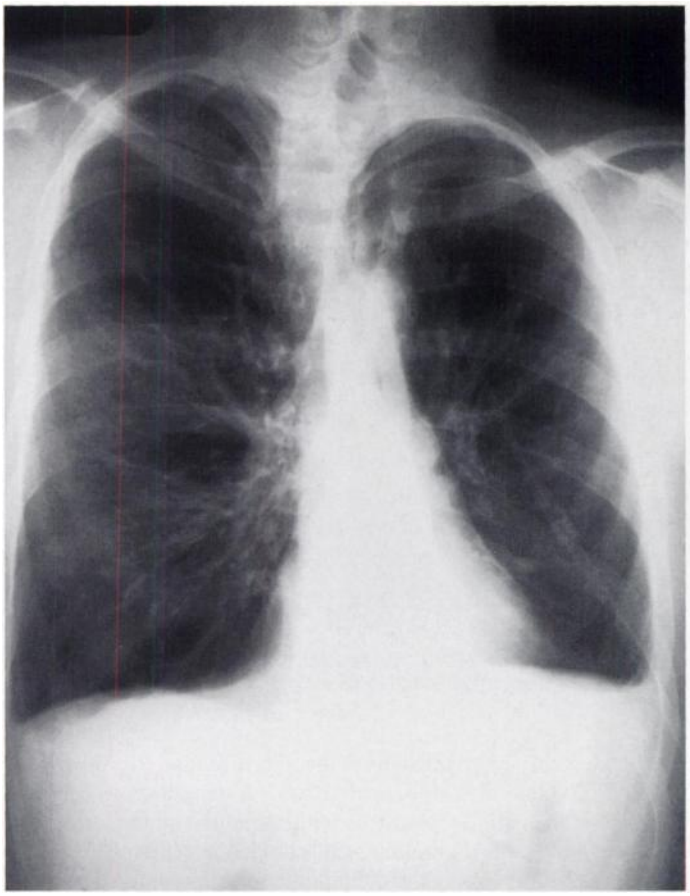

FIGURE 2. Posterioanterior chest radiograph demonstrates left lower lobe collapse with compensatory overinflation of the upper lobe causing the appearance of generalized oligemia.

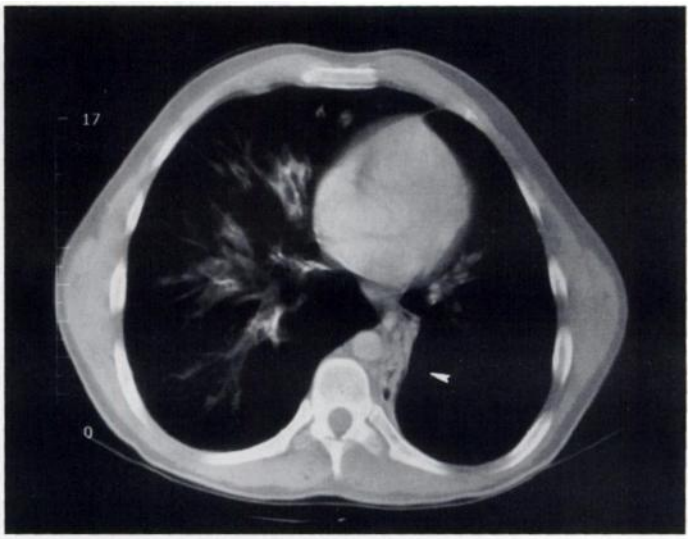

FIGURE 3. Chest CT confirms left lower lobe collapse (arrow) with oligemia of the overinflated left upper lobe.

We propose that the pseudostripe sign due to lobar collapse be included in a differential of the stripe sign and that it too should indicate the absence of pulmonary embolism in the involved lobe.

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Abnormal Colonic Accumulation of Fluorine-18-FDG in Pseudomembranous Colitis

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A 51-yr-old man with a history of pancreatic carcinoma was studied with [^{18}F]fluorodeoxyglucose ([^{18}F]FDG) and PET as part of staging for residual disease after chemotherapy. The PET study was performed during a clostridium difficile-associated diarrheal illness. Striking [^{18}F]FDG uptake was demonstrated in the wall of the colon over its entire length. Clostridium difficile associated diarrhea and mechanisms of [^{18}F]FDG uptake in normal and abnormal tissues are briefly reviewed and a mechanism for FDG uptake in this patient is postulated.

Key Words: fluorine-18-FDG; pseudomembranous colitis

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CASE REPORT

A 51-yr-old man originally presented in January 1994 with left loin pain. CT scan demonstrated a mass in the body and tail of the pancreas and additional low-density lesions within the liver suggestive of metastatic malignancy. A biopsy of the pancreatic mass was performed and histological examination depicted undifferentiated carcinoma with some elements suggestive of choriocarcinoma. There was no evidence of another primary site.

Chemotherapy was started later that month with carboplatin, ifosfamide with mesna and etoposide. After the third cycle of this treatment, the patient's β-HCG levels had fallen but remained elevated. The pancreatic mass had reduced in size. A change in treatment to a combination of procarbazine, vincristine and bleomycin (PVB) was begun at this time.

After four cycles of this combination chemotherapy regimen, repeat CT showed persistent lesions in the liver suggestive of residual tumor, but the pancreatic mass had continued to shrink.

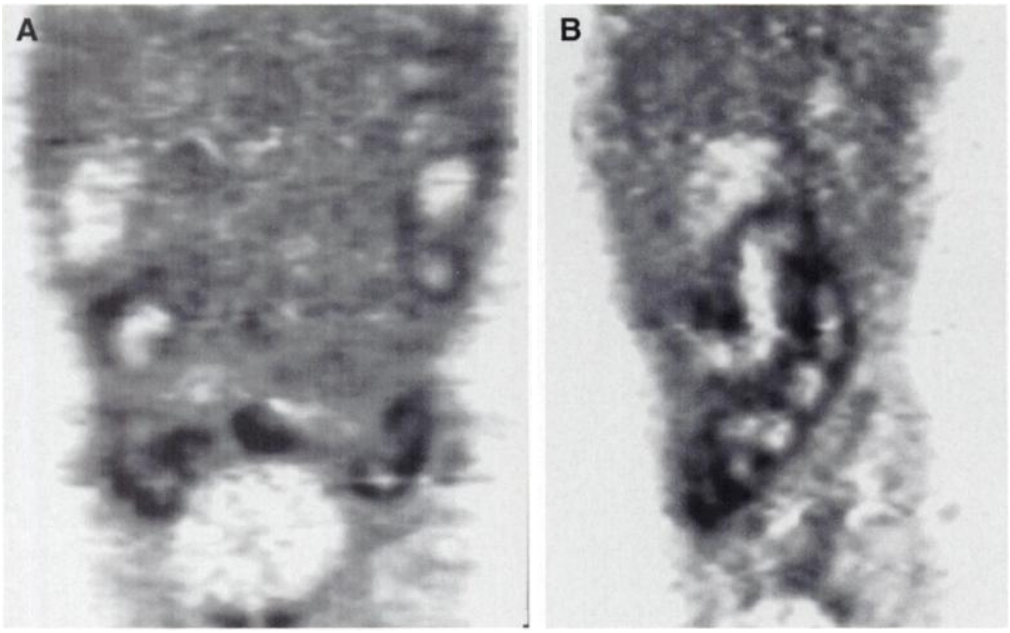

FIGURE 1. Coronal (A) and sagittal slice (B) of [^{18}F]FDG PET images. Colon is dilated and there is marked [^{18}F]FDG uptake in the colon wall (SUV = 2.1). Other slices showed that most of the length of the colon had this uptake. Background abdominal activity (SUV = 1.2) approximates that in the liver (SUV = 1.3). Patient had been diagnosed as having clostridium difficile toxin in the stool.

His β-HCG level remained elevated. He continued to exhibit thrombocytopenia with hypocellular bone marrow. He developed central venous catheter site infection at this time and was treated with intravenous and oral flucloxacillin.

Nine days after the fifth PVB cycle, he was admitted to the hospital with pyrexia, diarrhea without evidence of abdominal tenderness and neutropenia. Blood cultures were sterile, but the fecal specimen was positive for clostridium difficile toxin A. He was treated with oral metronidazole and intravenous ceftazidime.

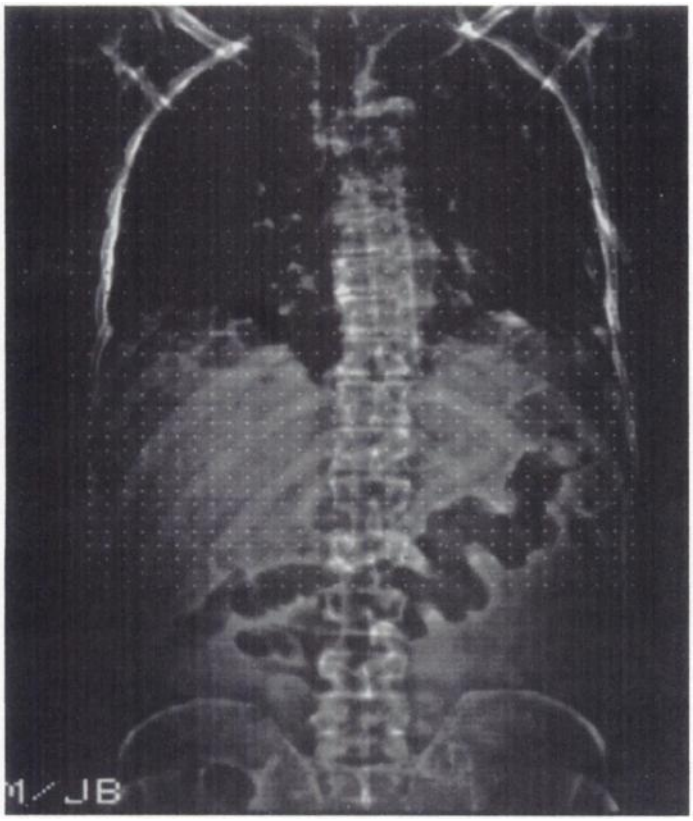

FIGURE 2. X-ray CT scout image obtained shortly after [^{18}F]FDG PET study. A dilated length of colon is evident.

There was no improvement in the diarrhea and he developed bloody diarrhea over the next 2 wk. Sigmoidoscopy showed proctitis with changes consistent with pseudomembranous colitis.

Since his stool clostridium difficile toxin remained positive during this time (implying continuing infection), he began oral vancomycin therapy. At this time, his tumor marker levels had returned to normal, although CT scan showed residual masses in the liver and the pancreas. FDG-PET was performed 3 days after the diagnosis of clostridium difficile toxin in the stool to assess residual malignancy.

A regional body scan was performed, which included the abdomen and pelvis in the area surveyed. The patient fasted for 4 hr before the FDG injection. Transmission images were obtained, and emission scans were performed 45 min after administration of 390 MBq [^{18}F]FDG intravenously. Four bed positions were required to cover the area of interest. Each transmission acquisition took 7 min duration (at 50–70 kcts/ sec). During the uptake phase the patient lay quietly on the scanner table. For the emission images, counts were acquired over each bed position for 20 min (at 9–13 kcts/sec). They moved only slightly during the entire imaging sequence. Both sets of images were reconstructed using a 128 × 128 pixel matrix, Hanning filter with a cutoff frequency of 0.4 cycles per pixel and a zoom factor of 1.5. All images were corrected for decay. Final images were reconstructed using transmission data for attenuation correction of the emission scan.

The PET image (Fig. 1) showed striking [^{18}F]FDG uptake in the wall of the colon over almost its entire length. The colon was dilated (also see x-ray CT scout image, Fig. 2). The amount of background activity within the abdomen was increased, approximating that of the liver. There was no evidence of abnormal liver uptake to suggest residual tumor deposits.

Standardized uptake values (SUV) for several areas of involved colon, liver and background abdomen were calculated using the following formula:

$$\text{SUV} = \frac{\text{Maximum activity in region of interest}}{\text{Corrected dose of [}^{18}\text{F]FDG } (\mu\text{Ci})/\text{Body weight (g)}}.$$

All values were (mean ± s.d.): involved colon: 2.1 ± 0.4, liver: 1.3 ± 0.2, background abdomen: 1.2 ± 0.2.

DISCUSSION

Pseudomembranous colitis was first described by Finney in 1893. Over time, it has best been classified as part of a spectrum of antibiotic-associated diarrheal illnesses related to clostridium difficile bowel infection. This spectrum ranges from diarrhea alone (although clostridium difficile is implicated in only 15%-20% of these infections, the rest have undetermined etiology) to diarrhea with colitis, to pseudomembranous colitis (PMC) (*1*).

The causative organism in pseudomembranous colitis, clostridium difficile, is an obligate anaerobic, spore-forming, gram-positive bacillus, which occurs ubiquitously in the natural environment. It elaborates two toxins, an enterotoxin (toxin A, mol wt 308 kDa) and a cytotoxin (toxin B, mol wt 270 kDa) that are involved in the induction of enterocolitis by this organism. Toxin detection is the most specific diagnostic test in clostridium difficile-associated disease.

In hospitalized patients, it is one of the most common causes of diarrheal illness, almost always after either parenteral or oral antibiotic therapy. The most frequently implicated antiobiotics are clindamycin, ampicillin and the cephalosporins. Symptoms usually develop after 1–2 wk of therapy. In addition to patients infected after antibiotic therapy, there is another group of patients who develop diarrhea in association with clostridium difficile infection after antineoplastic chemotherapy (*2*). Furthermore, PMC has been described in patients with diabetes mellitus, renal failure, intestinal obstruction and after abdominal surgery.

The clinical presentation of pseudomembranous colitis is diverse. Most patients develop brown or clear, watery diarrhea with or without mucus. A few patients have bloody diarrhea. Fever (up to 38°C) in just over half of patients and leukocytosis is common. Pseudomembranes may in fact be found through the entire length of the gut, from the esophagus to the rectum. The mucosa may show ulceration and petechial lesions. There is marked polymorphonuclear neutrophil (PMN) infiltration, mostly of the superficial lamina propria. The pseudomembranes overlying patchily inflamed mucosa appear gray or yellow and are composed of sloughed epithelial cells, PMNs and fibrin. At the most severe end of the clinical spectrum is the patient with toxic megacolon and bowel perforation with secondary hemodynamic problems.

The mechanism of abnormal [^{18}F]FDG uptake in our patient's colon is most likely related to the active inflammation in the bowel wall. Studies have demonstrated that uptake in inflammatory tissue localized to highly metabolic white cells such as neutrophils and macrophages (*3–5*). In these cells, glycolysis is increased and the hexose monophosphate shunt may be 20–30 times more active than baseline levels (*3*). Autoradiographic studies in mice have shown that although most of the activity within tumor represents actively metabolic malignant tissue, approximately 24% of this activity is in fact present in tumor-associated macrophages. The rate of uptake by macrophages in these circumstances is more rapid initially than that of the tumor, granulation tissue or necrotic tissue contained (*7*).

It is our experience, and that of other clinicians (*8*), that normal gut has unpredictable and patchy FDG uptake. The uptake tends to be low-grade and slightly less than that in normal liver, except in occasional instances of marked cecal uptake. The reason for this uptake is unknown and may be related to bacterial overgrowth. Bischof Delaloye et al. (*8*) have examined SUVs for normal intra-abdominal organs. Their values reflect the pattern mentioned above for liver and gut. They reported values of 3.2 ± 0.79 for liver, 2.95 ± 1.25 for gut, 2.84 ± 1.06 for stomach and 2.58 ± 0.58 for spleen, in their study (i.e. liver > gut > stomach > spleen). Although SUVs calculated at different institutions cannot be directly compared, the trend of uptake in different organs is a consistent one. There is one published report of [^{18}F]FDG uptake in colitis (*9*), in which there was no evidence of ova, parasites or clostridium difficile toxin and x-ray CT showed diffusely edematous colon.

The degree of background activity demonstrated in this abdominal study is closer to that of liver. Normally, abdominal background activity is substantially lower. It is tempting to postulate that this may indicate peritoneal inflammation, although clinical signs do not suggest this pathology. Drug-induced and other forms of colitis have been described as mimicking acute peritonitis (*10–12*) and excessive free fluid in the abdomen has been seen during laparotomy in some patients, suggesting the possible presence of subclinical peritonitis in our patient.

CONCLUSION

The diagnosis of colitis as a cause of abnormal [^{18}F]FDG uptake should be considered whenever a similar pattern of uptake is observed in the colon.

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