Radionuclide Detection of Duodenal Ulcer Perforation

Gregory R. Applegate, Philip Braunstein, and Felix Wang

Division of Nuclear Medicine, Department of Radiological Sciences, University of California, Irvine, Medical Center, Orange, California

An elderly obese male with a lengthy history of melanotic stools was admitted and was shown to have a posterior duodenal ulcer by endoscopy. He became obtunded and developed infected ascites. Because of his obesity, ascites, and inability to cooperate, the GI radiologist felt that a Gastrografin upper GI series would not be helpful. We therefore gave the patient ^{99m}Tc-labeled sulfur colloid and tap water through his nasogastric tube. We were able to clearly image a site of perforation at the duodenal bulb communicating with the lesser sac.

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There have been, to our knowledge, no reports of peptic ulcer perforation detected scintigraphically with orally administered radionuclides. There have been some reports of duodenal ulcer perforation (*1,2*) and altered biliary dynamics in a patient with an ulcer (*3*) identified on hepatobiliary studies.

CASE REPORT

A 74-yr-old white male was admitted with several weeks of melanotic stools. The patient had been taking four to five aspirin tablets a day for abdominal pain. He had a history of alcohol abuse, however, the last report of alcohol intake was 2 wk prior to admission. Physical examination on admission revealed an obese white male who was lethargic but oriented. Blood pressure pulse, and respiratory rate were 110/70 mmHg, 104/min and 25/min. Spider angiomata were identified over the anterior chest. There was abdominal distention with shifting dullness and the stools were melanotic and heme positive. Laboratory abnormalities included an albumin of 2.3 g/dl, with normal liver function tests. Hemoglobin was 5.9 g/dl with a hematocrit of 18.2%. Chest and abdominal x-rays revealed only mild elevation of the left hemidiaphragm and suggested splenomegaly without free air.

Two days following admission, endoscopy revealed a 1.5-cm large necrotic ulcer of the posterior duodenal bulb, that was not actively bleeding at the time of endoscopy. A paracentesis performed several days following endoscopy revealed yellow, cloudy peritoneal fluid with an elevated amylase of 2,304 U/l, LDH of 451 U/l, and a protein of 1.2 g/dl. There were 10,900 white cells per mm^3, with 96% polymorphonuclear cells, 1% lymphocytes, and 3% monocytes. Cultures of the peritoneal fluid grew out Clostridium perfringens. The patient was treated with ceftizoxime, ampicillin, and Flagyl for bacterial peritonitis felt to be secondary to gastrointestinal perforation, most likely from the large duodenal ulcer seen at endoscopy. Abdominal computed tomography (CT) examination at that time revealed cirrhosis with ascites, splenomegaly, with no evidence of free intraperitoneal gas. As a result of the patient's deteriorating condition, further evaluation with fluoroscopy or repeat endoscopy was felt to be impractical. At this time this obese patient with ascites was so obtunded that although there was no problem in getting him to lie still, he could not at all move cooperatively for fluoroscopic examination. As discussed below, the ability to localize GI perforation with water-soluble contrast agents is less than adequate (*4*). The GI radiologist felt that in this patient this inadequacy would be compounded by his obesity, ascites (which further decreases contrast), and his inability to cooperate. The GI radiologist was therefore most reluctant to perform an upper GI (with Gastrografin). Consequently, a radionuclide upper GI examination was performed on the patient without difficulty.

The patient was given 100 ml of tap water with 500 μCi of technetium 99m labeled sulfur colloid through his nasogastric tube. Anterior gamma camera acquisitions were obtained with a large field-of-view camera with the patient supine at five-min intervals up to 1 hr.

The initial image showed an area of increased radionuclide activity superior to the duodenal bulb and gastric antrum which, on subsequent images at 5, 10, 15, 20, and 25 min (Fig. 1A-D), increases in intensity and spreads superiorly to the lesser curvature of the stomach in the area of the lesser sac. There was no evidence of gastric outlet obstruction,

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For reprints contact: Philip Braunstein, MD, Div. of Nuclear Medicine, Dept. of Radiological Sciences, University of California, Irvine, Medical Center, 101 City Dr., Orange, CA 92668.

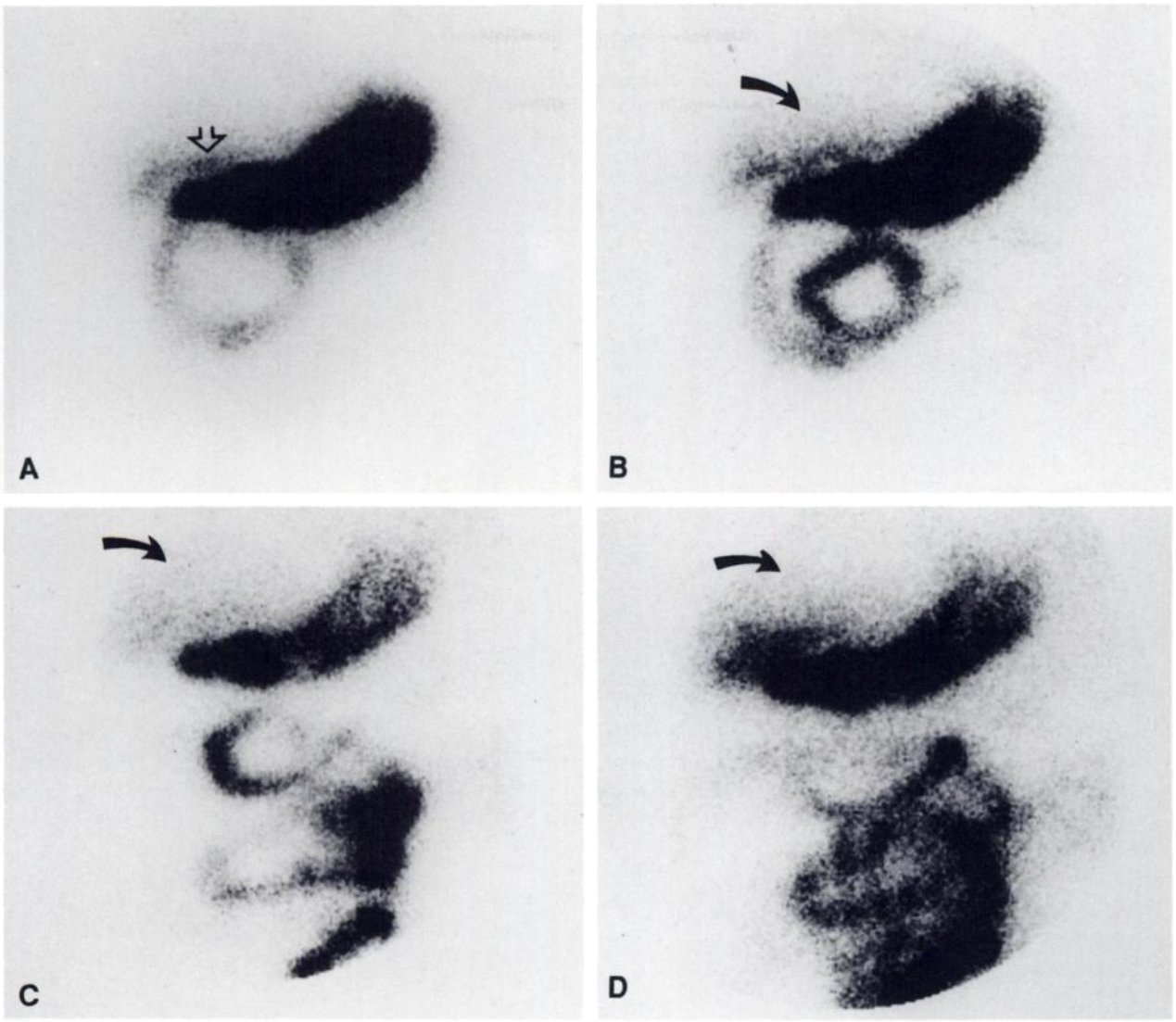

FIGURE 1
A: Initial anterior image over the upper abdomen. Note abnormal area of activity superior to the duodenal bulb (open arrow). B-D: Sequential images at 5, 15, and 20 min additionally demonstrate a progressive area of increased activity superior to the lesser curvature of the stomach in the area of the lesser sac (curved arrows).

however, there may have been minimal delay in the emptying of activity from the stomach. Given these scintigraphic findings, it appeared that the perforation was arising from the region of the posterior duodenal bulb and entering the lesser sac.

Unfortunately, several days following this study the patient expired and, at the request of the family, no autopsy was performed.

DISCUSSION

In a series of 65 patients with proven perforated duodenal ulcer, only 50% had leakage of contrast material that could be demonstrated fluoroscopically or using conventional radiography (*4*). Although the majority (93%) (*4*) of these patients will have demonstrated free intraperitoneal air, the ability to localize the site of perforation has obvious clinical application.

In conclusion, this appears to be a safe, noninvasive, simple study to perform for the detection of gastric perforation which may be done portably in an ICU setting. This study may be an alternative to contrast fluoroscopic examinations in some critically ill patients. These fluoroscopic examinations may at times be difficult to perform and have been reported to have low sensitivity (*4*), in part a result of poor contrast with water-soluble contrast agents. Further investigation regarding the specificity and sensitivity of nuclear imaging in detection of perforation will be required before this study can be recommended for routine clinical use.

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