# Indium-111 WBC Detection of Emphysematous Gastritis in Pancreatitis

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We present a case of emphysematous gastritis initially detected with $^{111}$In oxine-labeled white blood cell scintigraphy and subsequently confirmed by computed tomography. Early aggressive antibiotic and supportive therapy resulted in a successful clinical outcome.

**J Nucl Med 1990; 31:112–114**

Emphysematous gastritis is a rare entity, with a recent literature review reporting only 31 cases to date (*1*). Of the hollow viscera, the stomach is the least often reported site of intramural gas (*2*). Patients are invariably toxic and there is an associated 60–80% mortality (*1*). We report an early case of emphysematous gastritis, which was initially detected with indium-111- ($^{111}$In) labeled white blood cells and successfully treated with aggressive antibiotic therapy.

## CASE REPORT

A 54-yr-old black female with chronic renal failure on peritoneal dialysis was admitted with acute sepsis of unknown etiology. Three months prior to admission, she had presented with acute pancreatitis and an infected pseudocyst in the lesser sac which had been treated via an elective percutaneous transgastric drainage procedure (*3,4*). Physical examination during the current admission revealed a febrile (39.2°C) obese female with epigastric tenderness without guarding or rebound. Pertinent admission laboratory data revealed a sed rate of 26 mm/hr, hemoglobin of 6.8/mm$^3$, hematocrit of 21%, white blood cell of 13,800/mm$^3$, and peritoneal white blood cell count of 2,295/mm$^3$ (38% neutrophils). Blood cultures were consistently negative.

The patient remained febrile, her white blood cell count increased to 37,800/mm$^3$, and a culture of the peritoneal fluid grew Torulopsis glabrate which was treated with i.v. amphotericin B. Subsequently, an [$^{111}$In]oxine-labeled autologous white blood cell scan (Fig. 1A and B) was ordered to rule out a catheter tunnel infection. A focus of increased activity was identified in the pelvis, compatible with infection of the peritoneal dialysis catheter. In addition, the scan demonstrated increased radionuclide activity in the pancreatic bed and a broad curvilinear band of increased activity with a sharply demarcated outer margin and a less distinct inner margin, suggesting localization within a hollow viscus, such as the stomach. A prior upper gastrointestinal series, demonstrating findings compatible with chronic uremic gastropathy, is shown for comparison in Figure 2. A computed tomographic (CT) scan of the abdomen (Fig. 3A and B) confirmed the presence of intramural gas in the posterior gastric wall and revealed a fluid collection in the tail of the pancreas with gas in the pancreatic bed.

Gastroscopy with biopsy was performed and demonstrated extensive inflammation and necrosis of the posterior gastric wall, compatible with emphysematous gastritis. Gram stain of the biopsy specimen revealed mixed gram-negative organisms. The patient was started on an empiric regimen of gentamicin and the peritoneal dialysis catheter was removed and hemodialysis initiated. Following a 6-wk course of gentamicin, parenteral nutrition, and supportive therapy, the patient survived despite several complications including: congestive heart failure, uremic pericarditis, pneumonia with exudative effusions, and abdominal wound dehiscence. Serial CT scans during the convalescent period demonstrated a gradual decrease of the intramural air and follow-up endoscopy showed marked resolution of the inflammation.

## DISCUSSION

Historically, gas within the gastric wall has been categorized under two major headings; gastric emphysema and emphysematous gastritis (*1,2*). Gastric emphysema implies a situation with intramural gastric gas without associated gas-forming organisms. Typically, the intramural gas is linearly oriented, without gastric wall thickening. Etiologies for gastric emphysema include: instrumentation, trauma, obstruction, and primary pulmonary disease (*2,5,6*). Emphysematous or phlegmonous gastritis defines pathology caused by gas-forming bacteria. The stomach often appears irregular and mottled with gastric wall thickening, although this appearance is variable (*5,7*). Frequently there is a his-

Received May 16, 1989; revision accepted Sept. 11, 1989.

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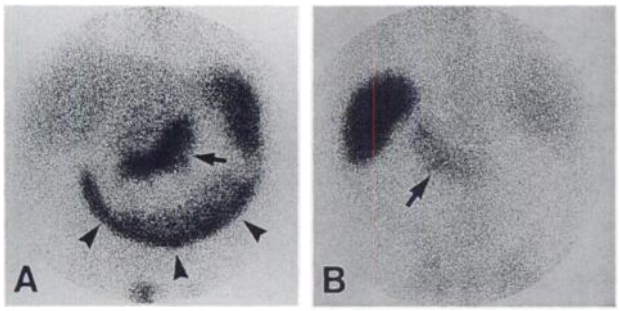


**FIGURE 1**
Anterior (A) and posterior (B) views from an $^{111}$In white blood cell scan demonstrate activity in the gastric wall (arrowheads) inferiorly, the pancreatic bed (arrows) superiorly and posteriorly, and the peritoneal dialysis catheter site in the pelvis.

tory of a prior insult such as alcohol abuse, gastritis, gastric infarction, and trauma (*1,6,7*). A case of emphysematous gastritis in association with pancreatitis and renal failure, similar to our patient, has been previously reported (*8*). Common organisms include: *Escherichia coli, Clostridium welchii, and Staphylococcus aureus* (*2*). The entire gastric mucosa may separate as a necrotic cast, invariably causing cicatricial scarring in survivors (*5*). The treatment is aggressive antibiotic therapy and vigorous supportive measures with nasogastric suction, intravenous fluids, and electrolytes. Surgery in the acute situation is recommended only in cases of perforation (*1,2*).

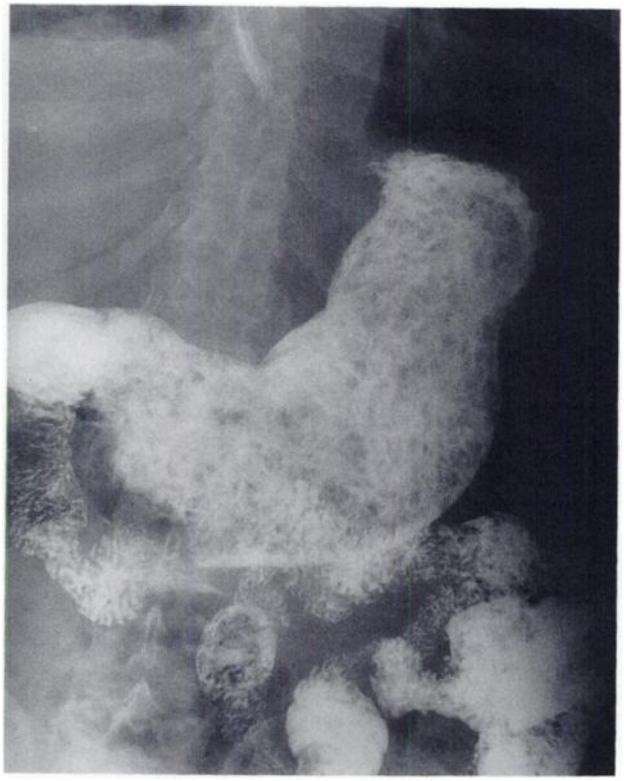

**FIGURE 2**
Anterior view from a prior upper gastrointestinal series demonstrating marked gastric dilatation with retained food particles, compatible with chronic uremic gastropathy.

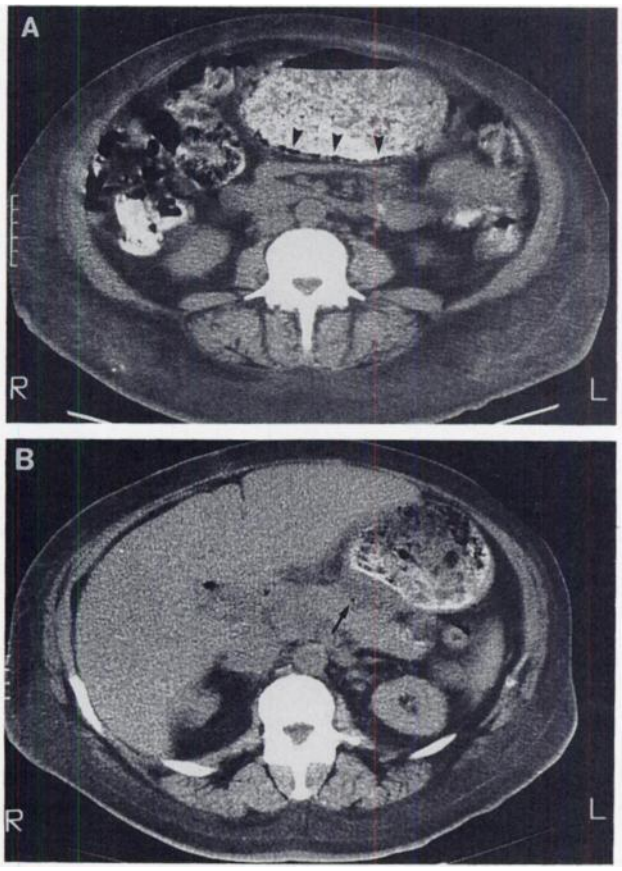


**FIGURE 3**
CT scans of the abdomen (A–B) demonstrate gas within the posterior gastric wall (arrowheads) and inflammed pancreatic bed (arrows).

In our case, the most likely etiology was extension of gas-forming organisms along the catheter-induced gastropancreatic fistula. The diagnosis was initially suggested by an $^{111}$In white blood cell scan which demonstrated an inflammatory process in the gastric wall with apparent diffusion of the radionuclide activity toward the gastric lumen. Computed tomography clearly confirmed the presence of intramural gastric gas and gram-negative organisms were subsequently cultured from endoscopic biopsies. Computed tomographic demonstration of intramural gastric gas has been described (*1*). Unfortunately, since gastric wall thickening is variable in emphysematous gastritis (*5,7*), its absence on CT scans does not exclude the diagnosis. Previous studies have shown a high sensitivity and specificity of $^{111}$In leuckocyte scanning for the detection and localization of inflammatory bowel disease (*9,10*). The present case suggests that scintigraphy may be particularly helpful in these patients in distinguishing gastric emphysema from emphysematous gastritis.

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