

The radiology corner*

Regenerated Splenosis Masquerading as Gastric Fundic Mass

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A case of regenerated splenosis 20 years after splenectomy masquerading as a gastric fundic mass is reported. Several endoscopic examinations revealed an extramucosal extrinsic mass in the gastric fundus and a chain of small ulcers high along the lesser curvature. Radionuclide technetium scan and CT scan accurately detected the regenerated splenic tissue indenting the gastric fundus and thus obviated the need for angiography and exploratory surgery.

INTRODUCTION

After splenectomy, splenules may regenerate or inactive accessory splenic tissue may become active and proliferate. Regenerated splenules may develop in any part of the abdomen. Since the spleen develops from the dorsal mesogastrium, some residual tissue may remain there after the full development of the spleen. After splenectomy, splenules may even grow in the wall of the stomach and simulate a gastric mass lesion.

We report a case presenting with upper gastrointestinal hemorrhage due to regenerated splenosis and gastric ulceration.

CASE REPORT

A 52-year-old white man was in excellent health until 8 days before admission to the University Hospital, Ann Arbor in June 1983, when he began to experience melanic stools. He complained of no bright red blood per rectum. There was no previous history of gastrointestinal complaints, peptic ulcer disease, or gastrointestinal hemorrhage. Twenty years previously in 1952 he underwent splenectomy for trauma sustained during a motor vehicle accident. There was no significant family or social history except that he is a nonsmoker and drinks socially. On the day of admission he felt weak and dizzy while walking home from work and came to the outpatient "walk-in clinic" at the University Hospital.

Physical examination revealed a pale slightly overweight white man in no acute distress. Blood pressure was 110/74, pulse 84/min, and respiration rate 12/min. The remaining physical examination was unremarkable. Laboratory data revealed hematocrit 25.3 but otherwise no abnormality. Urine analysis was normal. Electrocardiogram showed nonspecific S-T segment changes. Chest and abdominal radiographs were normal.

He was transfused with 5 μ of packed red blood cells. The hematocrit level rose to 36.9 and he was hemodynamically stable. The endoscopic examination of the upper gastrointestinal tract revealed a mass in the gastric fundus with two to three superficial ulcers high on the lesser curvature. A small blood clot and slight oozing of blood was noted.

An upper gastrointestinal series revealed a mass distorting the fundus around the gastroesophageal junction (Fig. 1A). The mass had sharp margins and showed two ulcers. The radiological diagnosis suggested ulcerating neoplasm of the gastric fundus.

He was reendoscoped three times again during the next week and each time a submucosal mass at the gastroesophageal junction protruding on to the high lesser curvature was noted. A chain of 3-mm ulcers were noted high on the lesser curvature just distal to the gastroesophageal junction. Since the bleeding had stopped and tiny ulcers were healing, no biopsy was taken. A CT scan in July 1983 revealed a regenerated spleen indenting the gastric fundus posteriorly (Fig. 1B). No other abnormalities were seen on the CT scan. A radionuclide Technetium sulfur colloid liver/spleen scan confirmed (Fig. 1C) the presence of regenerated splenic tissue adjacent to the fundus of the stomach close to the gastroesophageal junction. The patient was discharged without any further work-up. At follow-up clinic visit in August 1983 he had no complaints and was feeling fine. A repeat endoscopic examination again revealed a 2 \times 3 cm extramucosal mass at the gastroesophageal junction region protruding on half of the

* Edited by Daniel Maklansky, M.D., F.A.C.G.

circumference of gastroesophageal junction medially and posteriorly. All ulcers in the stomach had healed. At 6 months follow-up visit he is doing fine. Although

no angiogram was performed the findings of CT and radionuclide liver/spleen scan are convincing evidence of regenerated splenosis indenting the gastric fundus.

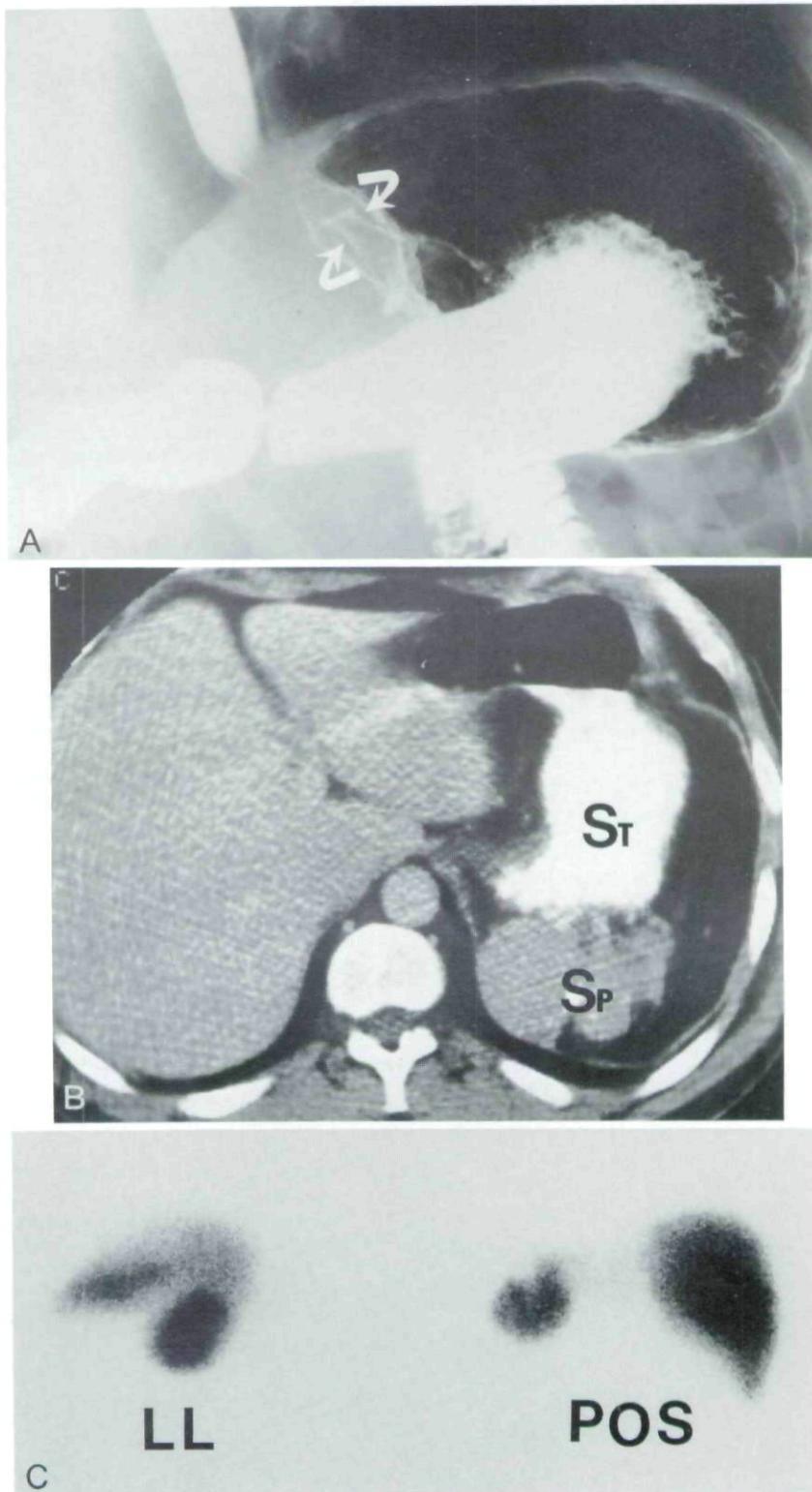


FIG. 1. *A*, an upper gastrointestinal examination shows distortion of gastric fundus by a mass lesion with overlying linear ulceration (*arrows*). The sharp margins of the mass lesion suggest intramural-extramucosal location. *B*, CT scan at the level of the fundus of the stomach showing a lobulated homogenous mass indenting the gastric fundus from behind and medially. Note absence of spleen because of previous splenectomy. The mass is regenerated splenosis. (*ST*, stomach; *Sp*, regenerated spleen). *C*, a radionuclide liver-spleen scan using 99m Technetium sulfur colloid confirms that the mass indenting the gastric fundus indeed represents splenic tissue (*LL*, left lateral; *POS*, posterior).

DISCUSSION

The ability of the splenic tissue to autotransplant and grow in ectopic sites after traumatic or surgical injury to the spleen has been recognized since 1883 (1). The term "splenosis" coined by Buchbinder and Lippkoff (2) in 1939 typically describes multiple small splenic implants scattered throughout the peritoneal cavity and abdominal viscera. However, splenic implants have been known to occur in the thorax (3) and subcutaneous tissue (4). It has been estimated that accessory splenic tissue may occur in as many as 10–16% of the population (5, 6). The regenerated splenosis is often incidentally found at surgery, autopsy, or diagnostic imaging tests in patients who had previous splenectomy (7–12). Eighty percent of the accessory spleens have been found to be located near the splenic hilum and 15–20% near the tail of the pancreas. The splenic tissue may occasionally regenerate in close proximity to other viscera and simulate primary disease of these organs. When regenerated splenosis is located near the stomach, a contour abnormality simulating an intramural mass lesion is usually seen on barium upper gastrointestinal series and differentiation from a primary neoplasms of the stomach may be very difficult. The implants of regenerated splenosis are usually asymptomatic and are incidentally discovered at laparotomy, autopsy, or diagnostic imaging tests in patients who had a previous splenectomy.

Review of reported cases of splenosis masquerading as gastric mass lesion (13–20) showed that gastric fundus was the area of involvement in all cases. The presentation as acute upper gastrointestinal hemorrhage due to erosive gastritis or overlying gastric ulcer was present in three of the eight previously reported cases.

Various methods used to detect accessory splenic tissues have been angiography and radionuclide liver spleen scans using Technetium 99m-sulfur colloid and chromium-51 labeled heat-treated erythrocytes. Recently, CT scanning has been extremely useful in detecting accessory splenic tissue. Our patient showed intramural extramucosal mass at several endoscopic examinations; the CT scan clearly demonstrated an extragastric mass which on Technetium 99m sulfur colloid scan was confirmed to be reticuloendothelial tissue and most likely spleen in this postsplenectomy setting.

This case emphasizes the important diagnostic role of CT and radionuclide spleen scans in symptomatic postsplenectomy patients to detect regenerated splenosis and to thus obviate the need for invasive procedures such as arteriography or exploratory laparotomy.

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