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Splenic abscess secondary to perforation of gastric ulcer

Introduction

Despite the availability of over the counter antihistaminic H2 receptor blockers and proton pump inhibitors, severe

complications of peptic ulcer disease are common and the incidence of potentially life-threatening ulcer complications has not declined. Peptic ulcer disease complications are on the rise in older patients because of an increased use of aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs). A gastric ulcer can penetrate to adjacent structures but in contrast to duodenal ulcer free perforation is rare.

Case Report

A 63 years old male presented to our emergency department with history of epigastric pain and fever of 3 weeks duration. On examination, there were diminished breath sounds in the left infrascapular area and abdominal examination revealed epigastric and left hypochondrial tenderness. On evaluation he had hemoglobin of 13.2 gm/dl, WBC count of $15.9 \times 10^9/L$ (normal, 4-11). His liver function tests revealed total serum bilirubin of 20.3 umol/L (normal, 0-17); AST 35 U/L (normal, 15-37); ALT 31 U/L (normal 30-65); gamaglutamyl transferase 498 U/L (normal, 7-32) and ALP was 408 U/L (normal, 50-136). His serum amylase was 26 U/L (normal, 25-115); and plasma lipase, was 93 U/L (normal, 14-286). Urea, creatinine and electrolytes were normal. X-ray chest (**Figure 1**) showed left-sided pleural effusion with underlying atelectasis and heterogeneous opacity in the left upper abdomen. Also air and fluid was seen in left upper quadrant because of leakage of gastric contents. CT scan after intravenous and oral contrast (**Figure 2**) showed

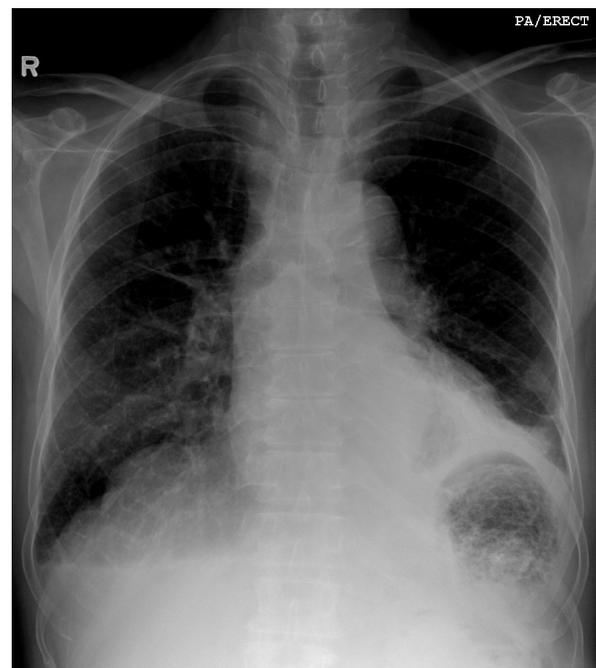


Figure 1: Chest X-ray showing left side pleural effusion and atelectasis

the splenic artery was thrombosed. A cavitory lesion, 18 cms x 12cms x 5 cms in size, with a thick irregular enhancing wall containing air and fluid was noted replacing the spleen. The capsule of the spleen was ruptured with extension of air anterior to the left hepatic lobe creating a pnemoperitonium, with an extension of the air pocket to the adjacent anterior abdominal wall. A free perforation in the body of stomach was present. Upper GI endoscopy (**Figure 3**) revealed a deep ulcer along the greater curvature in the proximal gastric body, covered with very thick exudate which was difficult to aspirate. Biopsy from the margins of the ulcer showed chronic active gastritis with ulceration, no *H. pylori* infection was seen on special stain. The patient underwent ultrasound guided pig tail drainage and 350 cc of purulent material was drained. The culture from the drainage fluid grew *E. coli* sensitive to piperacillin and tazobactam. After receiving a 2 week course of intravenous antibiotics the patient was doing fine, and was discharged on proton pump inhibitors.

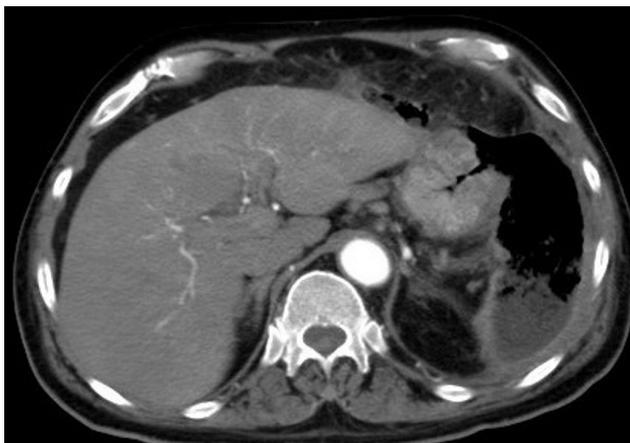


Figure 2: CT abdomen showing splenic abscess with air fluid level and free perforation of the stomach wall.

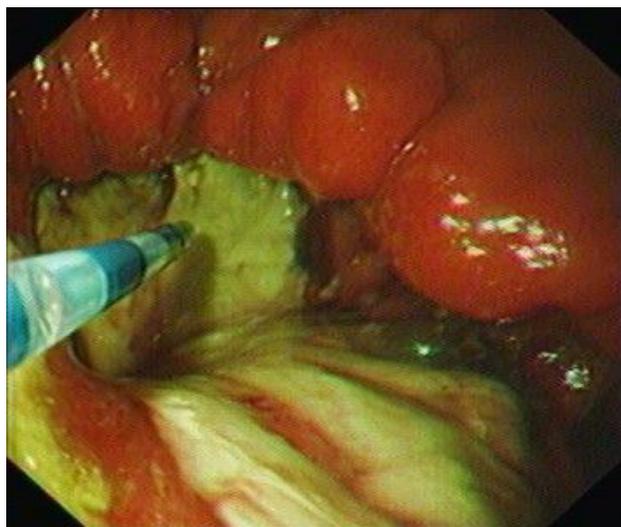


Figure 3: Endoscopy picture showing a deep gastric ulcer along the greater curvature in proximal gastric body

Discussion

In the era of proton pump inhibitors serious complication of peptic ulcer disease are still seen. Symptoms of gastric ulcer range from immediate pain after eating and non-specific epigastric discomfort to a complete absence of symptoms.¹ Gastric ulcers can occur throughout the stomach. Eighty percent are located on the lesser curvature, usually in the antrum or the angulus. The fundus, body, and greater curvature are less commonly affected.² The four major complications of peptic ulcer disease are bleeding, perforation, penetration, and gastric outlet obstruction. Upper gastrointestinal (UGI) bleeding is the most common complication among these. Comorbid disease increases mortality.^{3,4} One-third to one-half of perforated ulcers are associated with NSAID use.^{5,6} Gastric ulcers can penetrate to adjacent organs if left untreated. Unlike duodenal ulcer the free perforation of gastric ulcer is very rare. There are reports of penetration of gastric ulcer into the aorta, pancreas, liver, spleen, pericardium and other adjacent organs. Nishiyama et al reported penetration of spleen by a benign gastric ulcer with out free perforation in to the peritoneum.⁷ Another report has described a large gastric ulcer located in a giant hiatus hernia perforating into the heart leading to pneumo and pyo pericardium and cardiac temponade.⁸ Spontaneous pneumoperitonium secondary to penetrating benign gastric ulcer is rarely seen.⁹ In our patient there was free perforation of gastric ulcer in to the lesser sac resulting in thrombosis of the splenic artery and splenic abscess. The left sided pleural effusion was due to inflammatory reaction adjacent to left hemi diaphragm.

HAMID ULLAH WANI¹

BILALAHMED²

NADEEM HASSAN¹

ADELALQUTUB¹

Correspondence: Dr. Hamid Ullah Wani

Department of Medicine, Division of Gastroenterology,1

King Fahad Medical City, Riyadh, 11525,

Kingdom of Saudi Arabia.

Department of Emergency Medicine,2

Max Healthcare, New Delhi, India.

Email: drhamidwani@gmail.com

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True inflammatory polyps mimicking polyposis syndrome with intussusception and colo-duodenal fistula

Introduction

Polyps are common in the colon. There are different types of polyps which can occur in the colon. Different polyps have different criteria for their diagnosis, depending on the number and site of occurrence in the colon. However, occurrence of multiple true inflammatory polyps mimicking polyposis syndrome has neither been described in the literature nor does it have any criteria to diagnose it.

Case Report

A 38 yr old male patient presented with history of recurrent bilious vomiting and abdominal distention for a period of 3 yrs. At the onset of the disease, patient underwent investigations and was found to have a lesion in the illeocecal area, biopsy of

which was inconclusive. Following this, the patient received a trial course of anti-tubercular drugs (RNTCP) for 6 months. However, there was no improvement in symptoms.

On examination his vitals were stable. He had lost 17 kg of weight over a period of 3 yrs. He had no cutaneous markers of any disease. Abdomen was soft and did not reveal any mass lesion. Per rectal examination was normal. Routine blood investigations were normal. He underwent a barium examination (Barium meal follow through (BMFT) + barium enema). In the BMFT as the barium was not entering into the colon, a barium enema was also taken. BMFT showed (**Figure 1a**) a lesion compressing and protruding into the lumen of the duodenum. Barium enema (**Figure 1b**) showed a lesion in the region of hepatic flexure, with a polypoidal appearance. Colonoscopy (**Figure 1c**) showed multiple polypoidal lesion at 70 cm from the anal verge. No lumen could be visualized from that point. Biopsy showed only non specific lymphoid infiltrates.

The patient underwent surgery, which revealed a pulled up caecum. There was an inflammatory mass in the region of right upper quadrant. When the colon was opened by a small incision there was a polypoidal mass which was compressing and protruding into the region of duodenum. The lesion had perforated the colon and the surgeons were not able to pull back the lesion from the duodenum. Hence the duodenum was opened, lumen of which showed the polypoidal lesion (**Figure 2a**). The lesion was pulled back and the rent in the duodenum was repaired. A right hemicolectomy was also done.

Gross specimen showed multiple small elongated polyps (**Figure 2b**) and an area of thinning and perforation in the colon. Histology was suggestive of inflammatory polyps with dense chronic inflammation in the stroma of the polyps (**Figure 2c**). Patient became asymptomatic following surgery and has put on 15 kgs weight.

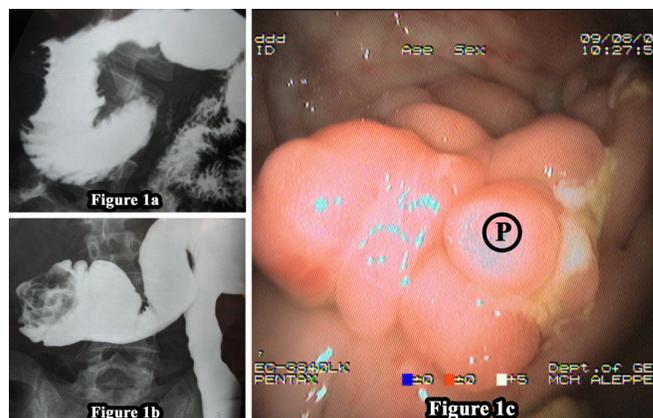


Figure 1: (a) Polyps projecting into the duodenum (BMFT), (b) polyps seen in barium enema, (c) polyps seen intussuscepting in colonoscopy. (P, polyp).