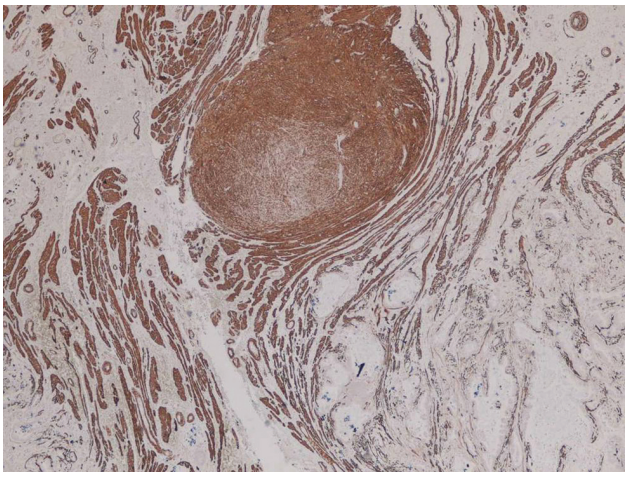

Figure 5. SMA expression of spindle cell tumor (H&E x200)

nancies, both solid tumors and hematologic malignancies, have been described in patients with CLL/SLL. The greatest risk is for skin cancer (7). Other solid tumors such as renal cell carcinoma, head and neck cancers, lung cancers, and colon cancer have been reported in association with CLL/SLL (3, 6). The increased cancer risk remained relatively constant over time since diagnosis of CLL/SLL, and was observed in both men and women, although men were at significantly higher relative risk than women (6). This is the first reported case in English literature of simultaneous gastric cardiac adenocarcinoma and leiomyoma in association with CLL/SLL infiltration in the perigastric lymph nodes and spleen.

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Acute gastric volvulus presenting with gastric outlet obstruction and upper gastrointestinal bleeding

Mide çıkışı tıkanıklığı ve üst gastrointestinal kanama ile başvuran akut gastrik volvulus

To the Editor,

Acute gastric volvulus is a rare disease that requires a high index of suspicion for diagnosis and rapid treatment (1,2). Many cases occur with a paraesophageal hernia (3). It is potentially life threatening because delayed diagnosis and treatment may lead to infarction, perforation, and death

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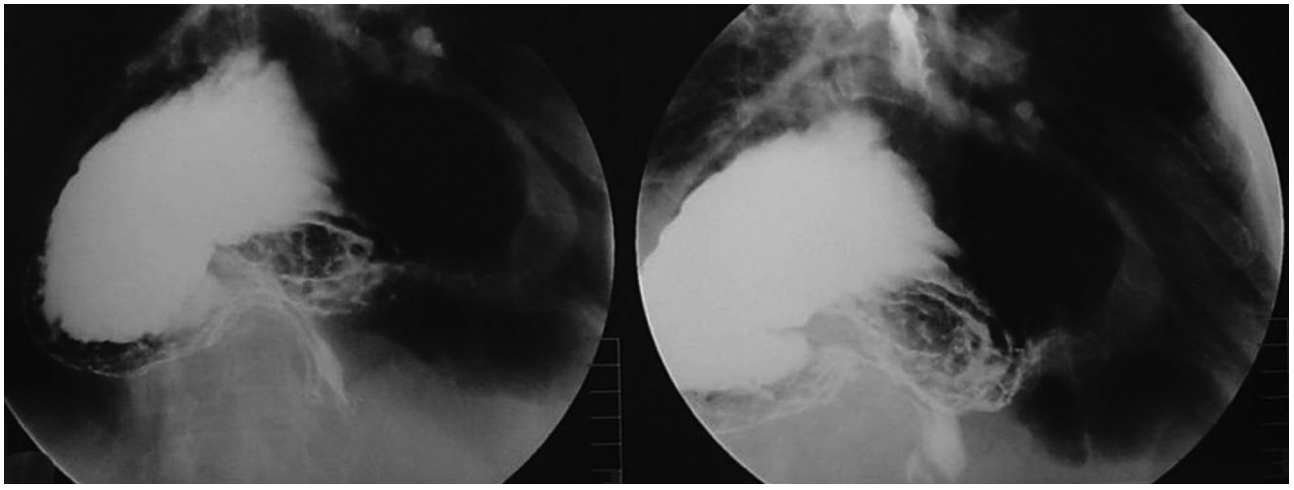

Figure 1. Upper GI series: Herniation of the stomach into the lower thorax. The greater curvature is located up and the lesser curvature is located down. Large paraesophageal hernia with organoaxial rotation of the stomach resulting in a gastric volvulus is seen.

(4,5). Acute gastric volvulus is regarded as a surgical emergency (5). We report a case of acute gastric volvulus secondary to a traumatic diaphragmatic injury that presented with gastric outlet obstruction and upper gastrointestinal (GI) bleeding.

A 75-year-old sailor presented to the casualty room with a two-day history of epigastric pain, nausea, and hematemesis. His medical history was significant for a hospital admission due to multiple trauma secondary to a motorcycle accident approximately 10 months prior. Physical examination revealed an elderly man who appeared ill and dehydrated. The initial clinical impression was upper GI bleeding, and appropriate management was performed.

Upper endoscopy was performed to verify the cause of upper GI bleeding. Diffuse esophagitis with ulceration was evident on examination of his esophagus. We observed a huge and deep, wide-mouthed outpouching of proximal gastric mucosa likely due to a large diaphragmatic defect. Examination of the cardia and proximal stomach revealed severe congestion and ulceration with multiple, discrete, various-sized ulcers. Upon retroflexion, the endoscopic picture was compatible with a huge mixed paraesophageal and sliding hiatal hernia. In spite of our repeated attempts at full endoscopic evaluation, the endoscope could not be advanced to the distal body of the stomach and antrum due to a twisted distal stomach.

The upper GI series showed herniation of the stomach into the lower thorax. The greater curvature was located cranially, and the lesser curvature was located caudally. A large paraesophageal hernia with organoaxial rotation of the stomach was demonstrated (Figure 1). The chest, upright and supine abdominal radiographs also confirmed the diagnosis.

We offered the transabdominal open surgical approach. At surgery, his stomach was completely herniated into the thorax with organoaxial volvulus; only the pylorus and distal antrum were located in the abdomen, with twisting of the distal stomach. After successful surgery, he was released from the hospital.

Gastric volvulus is a rare cause of both upper GI bleeding and gastric outlet obstruction. Endoscopic abnormalities such as ischemic ulceration and difficulty in negotiating the pylorus should raise the suspicion for gastric volvulus.

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Thrombocytopenia as a side effect of pantoprazole

Pantoprozolün yan etkisi olarak trombositopeni

To the Editor,

Proton-pump inhibitors (PPIs) are prescribed for treatment of peptic ulcer disease, gastroesophageal reflux disease, acute gastrointestinal bleeding, Zollinger–Ellison syndrome, and *Helicobacter pylori* eradication. These agents are generally well tolerated, with few commonly reported adverse effects (1). A rarely reported adverse effect consists of thrombocytopenia associated with the use of PPIs (2-5). We describe a case in which the patient developed thrombocytopenia while taking intravenous pantoprazole.

A 45-year-old man was admitted to the emergency room with upper gastrointestinal hemorrhage. The patient had a past medical history of a duodenal ulcer. The patient had taken nonsteroidal anti-inflammatory drugs. Upper gastrointestinal endoscopy had revealed a duodenal bulb deformity with a deep, penetrating ulcer over the anterior wall five months ago. Laboratory tests indicated hemoglobin 11 g/dl (13–15 g/dl), total leucocyte count 12.000 cells/cc (4.000–11.000 cells/cc) and platelet count 350x10^3/cc. Prothrombin time was normal. He was resuscitated with intravenous fluids and started on intravenous pantoprazole, given as a bolus intravenous injection of 80 mg followed by an infusion of 8 mg per hour for 72 hours. After resuscitation he underwent an upper gastrointestinal endoscopy, which showed a normal esophagus and stomach, and a non-bleeding, deep, duodenal ulcer approximately 1 cm in diameter on the anterior wall of the duodenal bulb. He was kept nil by mouth and given parenteral crystalloids and intravenous pantoprazole on hospital day 3. The hemoglobin level remained stable during the hospitalization. During the hospital course, the patient did not receive any blood products or fresh frozen plasma transfusions. He was then started on oral feeds on hospital day 4. The patient's platelet count decreased daily during the hospital course for 3 days, until it reached a nadir of 70 x 10^3/cc. Peripheral smear showed a reduced platelet count with normal morphology. Pantoprazole was discontinued on hospital day 4, and by days 6 and 8, the platelet count had risen to 100x 10^3/cc and 220 x 10^3/cc, respectively. Pantoprazole was switched to oral rabeprazole on hospital day 8. The patient's platelet count remained stable during the hospitalization. The patient was given rabeprazole 40 mg/day, amoxicillin 2000 mg/day, and levofloxacin 500 mg/day for 2 weeks for treatment of *Helicobacter*

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