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Authors

Jean, Michael C

Bencharit, Sittiporn

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CLINICAL VIGNETTE

Splenic Infarction after Nissen Fundoplication

Michael C. Jean, MD and Sittiporn Bencharit, MD

Case

A 77-year-old female presented to the outpatient gastroenterology for evaluation of severe gastroesophageal reflux disease (GERD), nausea, and epigastric pressure. Past surgical history included a laparoscopic Nissen fundoplication, performed 11 years ago at an outside hospital. Prior to the Nissen fundoplication, she reported severe GERD symptoms, which resolved for 2 years after surgery. Over time, her GERD symptoms gradually returned, followed by progressive nausea and epigastric pressure. Proton pump inhibitors, which she had taken for years, only partially improved these symptoms. She requested a surgical referral for redo of the Nissen fundoplication. Preoperative computed tomography (CT) imaging of the abdomen revealed a large 5cm hiatal hernia. Past medical history included hypertension. Preoperative cardiac clearance was obtained and preoperative blood work was normal, and she was referred for surgical evaluation.

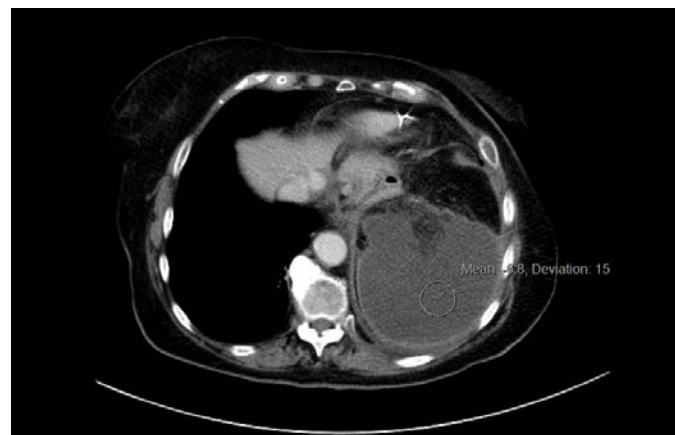
Open laparotomy was performed by a local surgeon, described as difficult due to dense adhesions extending from the stomach to the left lobe of the liver and diaphragm. These dense adhesions were carefully lysed, and it was noted that the previous fundoplication had broken down. A repeat hiatal Nissen fundoplication was performed, which also involved removal of previously placed surgical mesh. A ventral abdominal wall hernia was also repaired in the mid upper abdomen during this same surgery.

After the surgery, the patient reported significant postoperative pain in the epigastrium and lower chest. This pain radiated posteriorly into the back. On postoperative day number one, the white blood cell (WBC) count rose to 18,700. A gastrografin esophagram did not show any signs of leak or extravasation. After discussion with the surgeon, the postoperative pain was attributed to the difficult surgery, and her symptoms and pain slowly improved during the hospital course. The patient was discharged on postoperative day six, with her pain controlled with oral opioid analgesics. She remained afebrile during the hospital course and her WBC decreased to 11,100.

Four weeks later, the patient presented to the emergency room with an acute onset of right mid-abdominal pain. Her previous described postoperative epigastric and chest pain had since migrated to the left-upper quadrant. She reported a 25-pound weight loss since her surgery, despite significant recent improvement in her oral intake. Repeat CT imaging in the emergency room revealed an incarcerated right inguinal hernia. In addition, a 13.1cm x 11.2cm x 11.3cm fluid collection was

seen in the left-upper quadrant, fully replacing the spleen (Fig. 1). This liquefaction of the spleen was attributed to a perioperative splenic infarct. Repair of the right inguinal hernia was uneventful, but the patient continued to have elevated WBC, as well as fever to 39.2°C. A pigtail drain was placed into the splenic fluid collection, which grew out *Enterobacter aerogenes*. Intravenous imipenem was initiated, which was converted to oral ciprofloxacin for two weeks upon discharge. The patient's left-upper quadrant pain and early satiety symptoms resolved eventually after multiple pigtail drain changes over several months. Complete resolution of the fluid collection was documented on repeat CT imaging at the 7-month mark.

Figure 1



Discussion

The splenic artery is a branch off of the celiac trunk, which follows a path posterior to the stomach toward the spleen. As the splenic artery approaches the splenic hilum, it typically divides into multiple branches. These peripheral splenic arterial branches have poor collateral circulation. Thus injury or manipulation during surgery could result in downstream infarction of the spleen, typically in a wedge shaped pattern.¹ The short gastric vessels, which supply the gastric fundus, typically arise from these peripheral branches. Ligation of the short gastric vessels during mobilization of the gastric fundus is performed with certain methods of performing Nissen fundoplication.^{2,3} Given the proximity of the short gastric vessels to the peripheral splenic arterial branches, inadvertent injury to these branches can occur during gastric surgery. Signs and symptoms of

splenic infarction include left-upper quadrant pain, early satiety, nausea, fever/chills, and an elevated WBC count.^{4,5} The majority of patients can be managed conservatively.⁶ Splenic infarction can lead to the development of abscesses, splenic hemorrhage or rupture. If persistent or worsening symptoms occur, occasionally pigtail drainage is performed⁷ and rarely splenectomy is needed.⁴

Splenic Infarction is a known, rare complication after Nissen fundoplication and is estimated to be less than 1%.⁸ The true incidence may be higher given the difficulty of diagnosis and given that some patients have minimal or no symptoms.

The diagnosis should be considered following other types of gastric surgeries as well. In one series involving radical gastrectomy for gastric cancer patients, thirty-six (4.10%) of 877 patients developed splenic infarction.⁷ There is also a case report following laparoscopic sleeve gastrectomy.⁹

Conclusion

Splenic infarction is a known, rare complication following Nissen fundoplication. Symptoms are often difficult to differentiate between routine post-operative symptoms and splenic infarction. A high index of suspicion is needed in order to make the diagnosis, which is usually found on intravenous contrast CT imaging.

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