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Asymptomatic Partial Splenic Infarction In Laparoscopic Floppy Nissen Fundoplication And Brief Literature Review

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Short gastric vessels are divided during the laparoscopic Nissen fundoplication resulting in splenic infarct in some cases. We report a case of laparoscopic floppy Nissen fundoplication with splenic infarct that was recognized during the procedure and provide a brief literature review. The patient underwent a laparoscopic floppy Nissen fundoplication. We observed a partial infarction of the spleen. She reported no pain. A follow-up computed tomography scan showed an infarct, and a 3-month abdominal ultrasound showed complete resolution. Peripheral splenic arterial branches have very little collateral circulation. When these vessels are occluded or injured, an area of infarction will occur immediately. Management strategies included a trial of conservative management and splenectomy for persistent symptoms or complications resulting from splenic infarct. In conclusion, we believe that the real incidence is probably much higher because many cases of SI may have gone undiagnosed during or following an operation, because some patients are asymptomatic. We propose to check spleen carefully for the possibility of splenic infarct.

Key words: Laparoscopic Nissen fundoplication – Short gastric vessels – Splenic infarction

The laparoscopic Nissen fundoplication (LNF) is widely accepted as the gold standard surgical therapy for gastroesophageal reflux disease (GERD) since Dallemagne reported the first cases in 1991.[1] The division of the short gastric vessels for mobilizing the greater curvature and fundus is controversial.[2,3] Although the occlusion of the main splenic artery rarely causes splenic infarction (SI), the peripheral splenic arterial branches have very little collateral circulation and the occlusion or injury of peripheral splenic arterial branches results in an area of infarction distal to the involved

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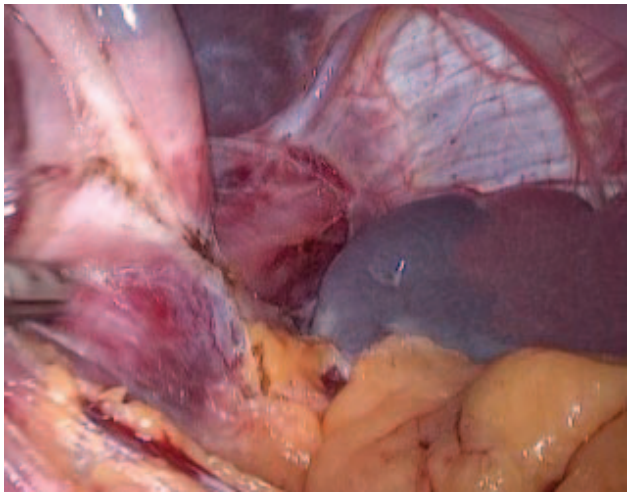

Fig. 1 Splenic infarction of the upper pole recognized during the procedure.

branches.[4] Irrespective of whether uncontrolled bleeding occurs, the terminal branches of the splenic vessels can be injured or ligated during division of the short gastric vessels (SGV). This risk increases in case of bleeding. Unfortunately, when SI results from vessel ligation, the injury to the spleen may go unrecognized.[5]

We report a case of laparoscopic floppy Nissen fundoplication with SI that was recognized during the procedure and provide a brief literature review.

Case Report

A 32-year-old woman presented with GERD symptoms that were not fully controlled with medical management. She underwent standard preoperative testing including an esophagogastroduodenoscopy (EGD) with a biopsy, upper gastrointestinal examination (UGI), and esophageal manometry. She underwent a floppy LNF, with division of the SGV performed by using the thermal sealing device. There was no bleeding during the procedure. We observed a partial infarction of the spleen (Fig. 1). The patient made a rapid postoperative recovery and was discharged on postoperative day 2 with a recommendation of close follow-ups weekly.

On follow-up visits, the patient reported good reflux control and no pain. A computed tomography (CT) scan obtained 2 weeks postoperatively to investigate the SI showed a segmental infarction of the upper pole of her spleen (Fig. 2A). The patient was managed conservatively. A follow-up CT scan 6 weeks later showed a much smaller area of infarction (Fig. 2B), and a 3-month abdominal ultrasound showed complete resolution.

Discussion

Splenic artery occlusion or injury rarely causes SI. However, peripheral splenic arterial branches have very little collateral circulation. When these vessels are occluded or injured, an area of infarction will occur immediately distal to the involved branch or branches.[4] Small areas of ischemia, infarction, or both may go unrecognized, mainly in the operating room.[5]

There is limited information about SI in the literature. Henry *et al*[6] reported a case of partial SI in a series of 169 laparoscopic adrenalectomies, and this case was managed with observation alone. Another 5 cases of SI occurred as a complication of LNF.

SI as a complication of LNF is rare. It is estimated to be less than 1.0%.[5,7,8] The real incidence is

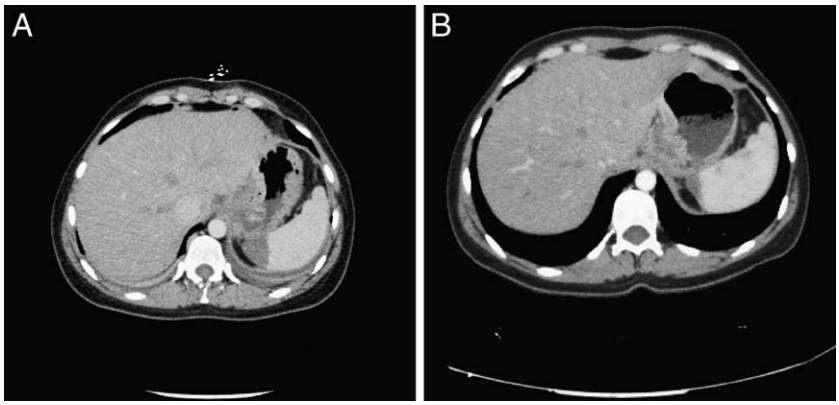

Fig. 2 (A) CT scan demonstrating an area of splenic infarction of the upper pole. (B) A repeat CT scan at 6 weeks demonstrating nearly complete resolution and atrophy.

probably much higher because many cases of SI may have gone undiagnosed during or following an operation.

An unrecognized SI following an LNF may be difficult to diagnose. Left-upper quadrant abdominal pain, changes in appetite, and early satiety are common after LNF. Fever and an elevated white blood cell count developing after esophageal mobilization would raise suspicion of an esophageal or gastric injury. The diagnosis can be confirmed with an intravenous (IV) contrast CT scan, liver-spleen scan, ultrasound, or angiography. The CT scan demonstrates a low attenuation wedge of tissue surrounded by normal-appearing, contrast-enhanced splenic parenchyma.[5]

Wilkinson *et al*[5] reported about 2 patients referred to their institution with left-upper quadrant pain and radiographic evidence of SI that resulted from a peripheral splenic artery branch injury during LNF. In the first case, one superior vessel near the splenic hilum retracted and continued to bleed during the SGV dissection. Direct application of the harmonic scalpel controlled the bleeding. The patient was discharged on postoperative day 3, a day later than planned because she was experiencing somewhat more pain than usual, although she remained afebrile and diet was well-tolerated. On follow-up visits, she reported severe left low back and flank pain. A CT scan obtained 19 days postoperatively showed a segmental infarction of the upper pole of the spleen. The patient was managed conservatively. A follow-up CT scan 1 month later showed a much smaller area of infarction, and a 3-month scan showed complete resolution. The second patient underwent a standard LNF. Bleeding from 1 SGV was controlled with the application of multiple surgical clips. The patient was discharged on postoperative day 2. The patient reported persistent fever and left-upper quadrant pain and was readmitted to the hospital 1 week following surgery. The CT scan demonstrated a large segmental infarction. The patient was initially managed conservatively, but his pain and fever worsened. Two months after surgery, a repeat CT scan showed persistent superior pole infarction with intense surrounding inflammation. The indication for surgery was persistent pain. The spleen was approached laparoscopically, but efforts to mobilize the spleen from the adjacent diaphragm and colon were unsuccessful. Early conversion to a midline laparotomy facilitated the procedure.

Martínez *et al*[7] reported intraoperative, discrete color changes suggestive of ischemia of the upper pole of the spleen after SGV division. One month later, the patient presented with a splenic abscess, which required open splenectomy.

Watson *et al*,[9] in a multicenter, prospective, double-blind, randomized trial of laparoscopic Nissen versus anterior 90° partial fundoplication, reported SI that entailed the loss of blood supply to approximately 75% of the spleen following clipping of a bleeding vessel during division of the SGV. Further surgical intervention was not required.

Ipek *et al*[8] reported 20 cases of SI in 2100 patients who underwent laparoscopic floppy Nissen fundoplication. The classic pattern, in all cases, was a small area of infarction, less than 10–15% of the total splenic volume, localized mainly in the upper pole. There were no conversions. The mean length of hospital stay was 1.2 days (range, 1–2 days). During the 3-month follow-up period, only 2 patients (10%) had persistent abdominal pain, in which CT demonstrated the infarcted areas involving less than 15% of the splenic parenchyma. There was no significant bleeding during SGV division in the 20 cases presented. The occurrence of SI can be explained by possible variations in the vascular anatomy of the spleen. All cases were managed successfully with observation alone.

Dijkman *et al*[10] reported a patient with Down syndrome with a vanishing spleen after a Nissen fundoplication, who died of overwhelming pneumococcal septic shock 7 months after the operation. Vascular anomaly in Down syndrome, inadvertent ligation of the splenic artery, or volvulus of the spleen may have caused a compromised splenic arterial circulation.

Driessen *et al*[11] reported 11 pediatric patients with discoloration of the upper pole of the spleen during surgery, with a median estimated splenic surface of 20% (range, 5–50%). Postoperatively, there were no patients with left-upper abdominal pain, fever, or chill as symptoms of splenic infarction. Pre- and postoperative ultrasound measurements of the spleen were performed in the children undergoing Nissen fundoplication, and it was concluded that the discoloration of the spleen found after SGV division is not associated with a significant postoperative decrease in splenic size.

Diagnosis and follow-up are carried out by performing CT scans. A CT scan usually demonstrates a well-defined hypodense area surrounded by normal-appearing, contrast-enhanced splenic parenchyma[5,12] Ultrasonography can also be used to differentiate between a hypoechoic acute infarction and a hyperechoic healed lesion and to

demonstrate the evolution of the SI into an abscess.[12] When the patient's clinical signs are normal and radiologic studies, if necessary, do not merit any further intervention, conservative therapy, aimed at pain relief alone, is adequate.[8]

The infarction is usually silent, in autopsy series of SI, 10 to 50% of patients had no clinical symptoms that could be attributed to SI.[13,14] In our case, there were no symptoms related to SI; it was recognized during the procedure.

If the infarction is secondary to the focal ischemia, it may progress to splenic abscess or massive subcapsular hemorrhage with eventual rupture if it is secondary to the occlusion of a major splenic vessel.[7,12]

Ipek *et al*. reported that no patients experienced persistent postoperative symptoms, except in 2 cases, where moderate left-upper quadrant abdominal pain lasting 6 to 10 days was reported on follow-up visits. No further attention was given to these events because of the regression of the infarcted areas demonstrated on control CT scans and the gradual resolution of the symptoms.

After postoperative SI was diagnosed, a trial of conservative management is recommended. If an SI-related complication is to be managed conservatively, the patient and surgeon should be educated regarding the risks of observation. When surgery is indicated, semielective splenectomy with preoperative immunization and full bowel preparation can be performed in most cases.[5,8]

Conclusion

LNF-related SI is fairly common pathology. We believe that the real incidence is probably much higher, because many cases of SI may have gone undiagnosed during or following an operation because some of the patients are asymptomatic. Most of the cases are diagnosed with symptoms following surgical intervention, but can be recognized during the procedure. We propose checking the spleen carefully for SI. Conservative treatment and close follow-up is the favorable management strategy.

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