Splenic Infarction Following Laparoscopic Nissen Fundoplication: Management Strategies

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ABSTRACT

Techniques for mobilizing the greater curve of the stomach during laparoscopic Nissen fundoplication (LNF) include division of the short gastric vessels (SGV). The splenic artery and vein lie directly posterior to the proper plane of dissection. Uncontrolled bleeding during SGV division places the splenic vessels at risk for inadvertent injury or ligation. We report herein on 2 patients referred to our institution who had left upper quadrant pain and radiographic evidence of segmental splenic infarction (SI) that resulted from a peripheral splenic artery branch injury during LNF. Management strategies included a trial of conservative management and splenectomy for persistent symptoms or complications resulting from SI. Intense inflammation and adhesion formation making laparoscopic splenectomy difficult should be anticipated when operating on the infarcted spleen.

Key Words: Splenic infarction, Laparoscopic Nissen fundoplication, Laparoscopic splenectomy, Short gastric arteries, Partial splenic embolization.

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INTRODUCTION

The laparoscopic Nissen fundoplication (LNF) procedure has revolutionized the surgical management of gastroesophageal reflux disease (GERD). Numerous surgical series have demonstrated good symptom relief and minimal operative morbidity. The necessity for mobilizing the greater curve of the stomach via division of the short gastric vessels (SGV) has been debated.[1,2,3] At present, division of the SGV is commonly performed.[4,5] If uncontrolled bleeding occurs during SGV mobilization, the splenic vessels are at risk of inadvertent injury or ligation. When bleeding results, these injuries are most often recognized and managed at the time of surgery. Unfortunately, when splenic infarction (SI) results from vessel ligation, the injury to the spleen may go unrecognized. We report on 2 patients referred to our institution with left upper quadrant pain and radiographic evidence of splenic infarction that resulted from a peripheral splenic artery branch injury during LNF. We have outlined a diagnostic algorithm and management strategy for this complication **(Figure 1)**.
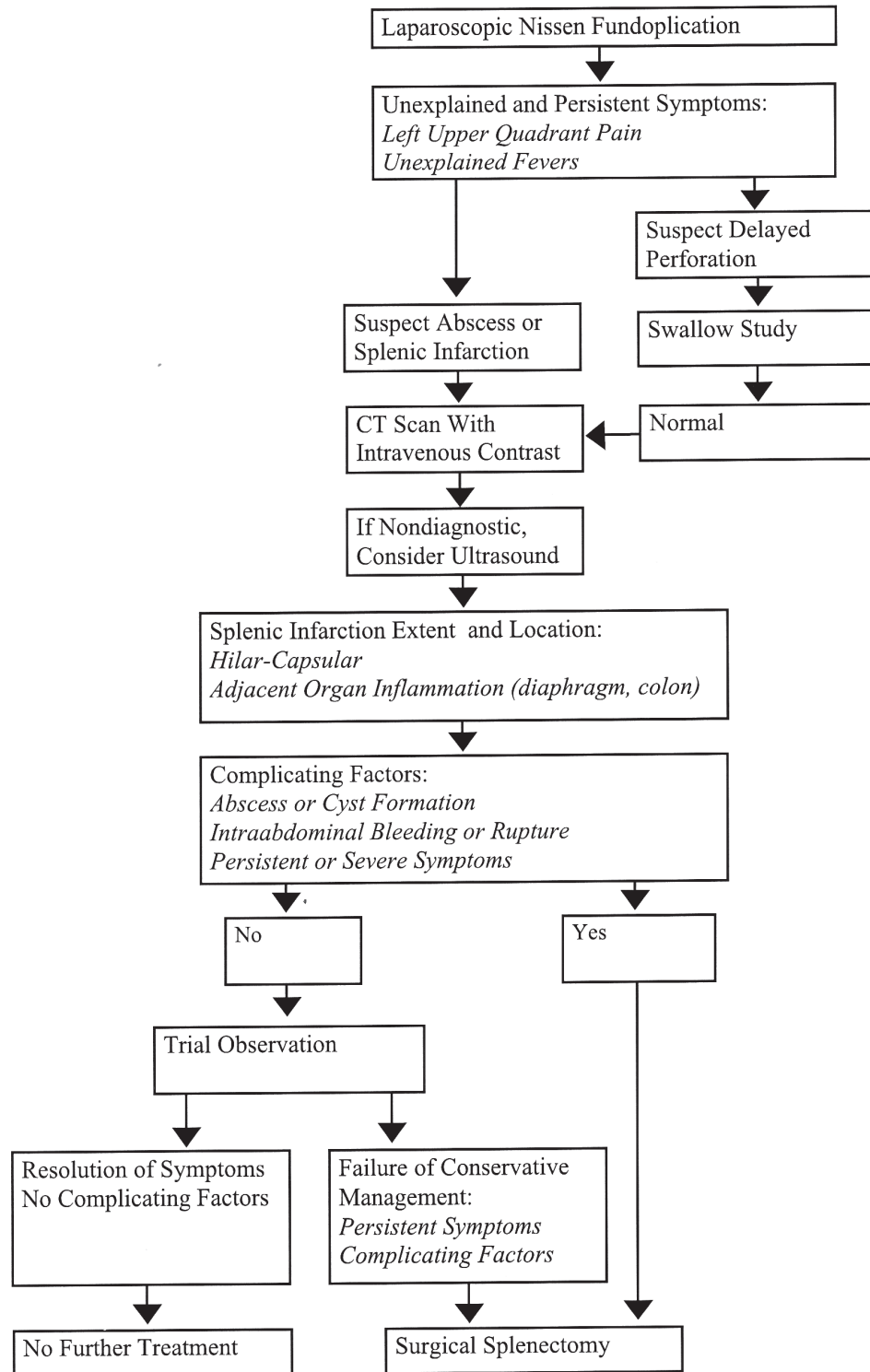
CASE REPORTS

Case 1

A 45-year-old female presented with classic symptoms of GERD, which 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 standard LNF with division of the SGV performed using the Harmonic scalpel. During the SGV division, 1 superior vessel near the splenic hilum retracted and continued to bleed. Directed application of the Harmonic scalpel controlled the bleeding with only minor blood loss of approximately 50 cc. The case was completed without further incident. The patient was discharged on postoperative day 3, one day later than planned because of somewhat more pain than usual though she remained afebrile and was tolerating a diet well.

On follow-up visits, she reported good reflux control but persistent, severe left low back and flank pain. A com-
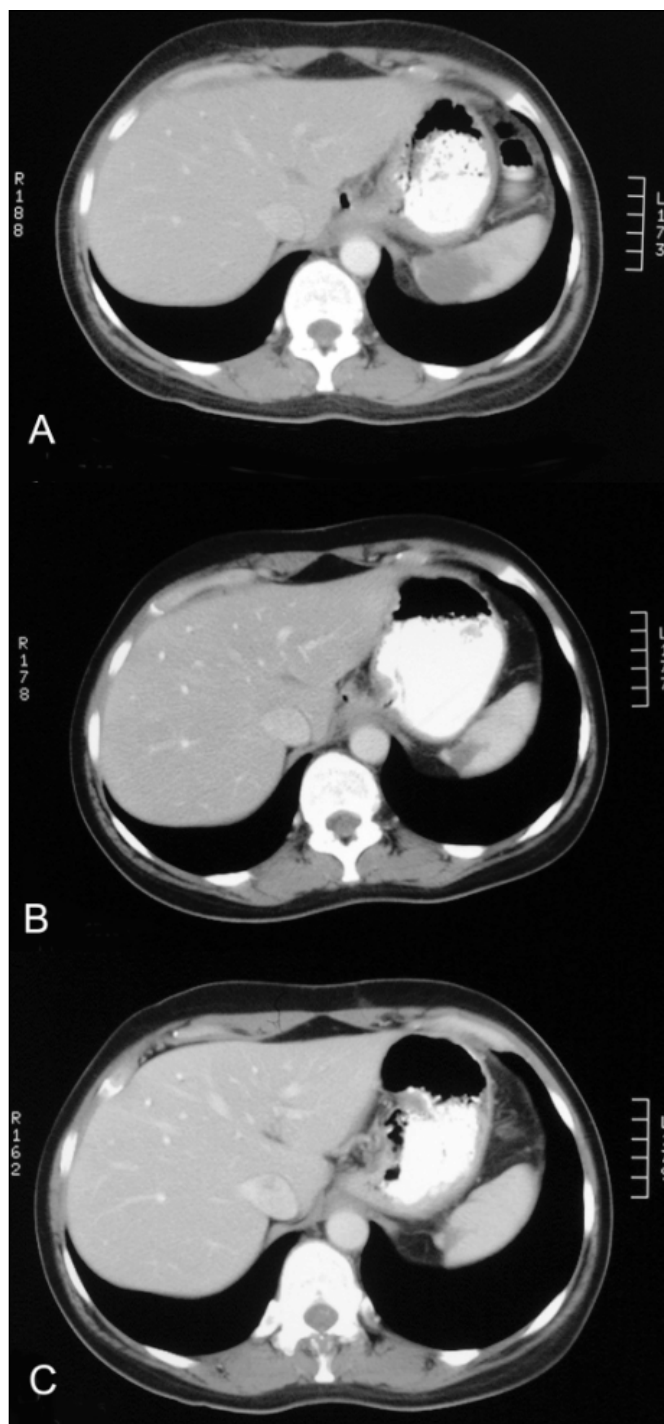
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│     Laparoscopic Nissen Fundoplication   │
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│  Unexplained and Persistent Symptoms:    │
│  Left Upper Quadrant Pain                 │
│  Unexplained Fevers                       │
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│  Splenic Infarction │  └─────────────────────┘
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│  If Nondiagnostic,   │
│  Consider Ultrasound │
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│  Splenic Infarction Extent and Location:       │
│  Hilar-Capsular                                │
│  Adjacent Organ Inflammation (diaphragm, colon)│
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│  Complicating Factors:                         │
│  Abscess or Cyst Formation                     │
│  Intraabdominal Bleeding or Rupture            │
│  Persistent or Severe Symptoms                 │
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│ No Further   │ │   Surgical Splenectomy    │
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Figure 1. Management strategy following laparoscopic Nissen fundoplication.

Figure 2. CT scan demonstrating a small area of intracapsular infarction with minimal inflammation (A). Repeat CT scan at 6 weeks (B) and 3 months (C) demonstrating complete resolution. This case was successfully managed conservatively.

puted tomography (CT) scan obtained 19 days postoperatively to investigate these complaints showed a segmental infarction of the upper pole of her spleen **(Figure 2A)**. The patient was managed conservatively with pain medication, and her symptoms largely subsided over the next month. A follow-up CT scan 1 month later showed a much smaller area of infarct **(Figure 2B)**, and a 3-month scan showed complete resolution **(Figure 2C)**. The patient subsequently underwent a laparoscopic cholecystectomy 1 year later. Laparoscopic examination of the spleen demonstrated a single adhesion over a normal capsule.

Case 2

A 33-year-old male suffered from worsening GERD symptoms that were only controlled with escalating doses of proton pump inhibitors. He opted for elective surgical correction over chronic medication dependence. Preoperative evaluation included EGD with a biopsy and manometry. The patient underwent a standard LNF with division of the SGV performed using the Harmonic scalpel. Bleeding from one SGV was controlled with the application of multiple surgical clips. The patient made a rapid postoperative recovery and was discharged on postoperative day 2.

The patient reported persistent fevers and left upper quadrant pain and was readmitted 1 week following surgery. A directed search for esophageal injury was performed to include a swallow study and a CT scan. The CT scan demonstrated a large segmental infarction **(Figure 3)**. The patient was initially managed conservatively, but his pain and fevers worsened. A CT scan obtained 6 weeks after surgery demonstrated a persistent area of infarction and new pleural effusions. The pleural effusions were aspirated and cultured. Three sets of blood cultures and the pleural fluid cultures were negative. Two months following surgery, he was transferred to our institution. On the first hospital day, he had a temperature to 101.4°F, an elevation in his white blood cell count (WBC) to 17.4, and an increase in his granulocytes to 82%. A repeat CT scan showed persistent superior pole infarction with intense surrounding inflammation. An arteriogram was performed that confirmed our clinical suspicion of SI resulting from a peripheral splenic artery branch ligation **(Figure 4)**.

The patient underwent preoperative immunization and bowel preparation. He was scheduled for a laparoscopic

Figure 3. CT scan demonstrating an infarction involving the superior pole of the spleen with intense inflammation surrounding the capsule. The indication for splenectomy was persistent pain and fevers.

Figure 4. Arteriogram demonstrating the occluded peripheral splenic arterial branch and several clips. Note the absence of vascular contrast to the superior pole of the spleen. Splenic infarction occurs with peripheral branch occlusion due to limited collateral circulation.

splenectomy with clear counseling about the high conversion rate. Surgically, the abdomen was adhesion free and initial splenic mobilization went uneventfully. As the superior pole of the spleen was approached, intense fibrous, inflammatory adhesions involving the diaphragm, stomach, and left lobe of the liver were encountered **(Figure 5)**. After converting to an open procedure, the spleen was removed. Pathologic examination showed focal necrosis with surrounding fibrosis. Gram-positive organisms were seen in the specimen and cultures grew both streptococcus intermedius and coagulase negative staphylococcus. The patient did well following surgery and was discharged on postoperative day 7.

DISCUSSION

The spleen is an organ with good proximal collateral circulation via the gastric, omental, and pancreatic vessels. Occlusion of the main splenic artery seldom produces splenic infarction. On the other hand, 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.[6] Most spontaneous cases of SI are caused by hematologic or thromboembolic events. Three recent reviews describe the clinical presentation and management of this form of SI.[7,8,9] A growing body of lit-

erature discusses therapeutic partial splenic embolization (PSE). PSE produces SI to treat various diseases including hematologic splenic disorders, splenic trauma, and portal hypertension. The experience gained from PSE can be applied to iatrogenic SI.

Splenic injuries have been reported in the laparoscopic literature. Most of these injuries involve parenchymal tears or vascular injuries that are recognized and managed intraoperatively. Management tenets involve standard surgical teachings: obtain hemostasis by primary repair, direct pressure, or splenorrhaphy/splenectomy as indicated. Splenic bleeding presents as an acute surgical event; by comparison, infarction is silent and may go unrecognized at the time of surgery. Working at the splenic hilum provides limited exposure because fluid and blood can obscure the surgical field. Mobilizing the stomach away from the spleen helps visualize the SGV for safe division. If bleeding occurs near the hilar area, blind clip application or the use of the Harmonic scalpel
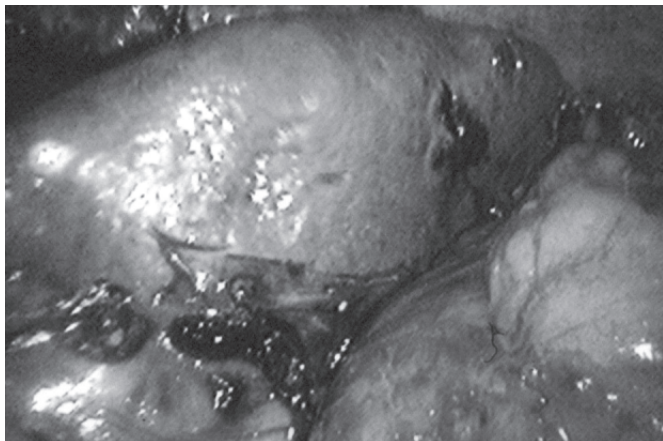
Figure 5. Laparoscopic splenectomy was attempted, but intense inflammation involving the diaphragm, liver, and colon required converting to an open procedure.

places the splenic vessels in danger because the SGV arise from the splenic artery. Occlusion of a peripheral splenic artery will produce an infarct due to the limited collateral circulation in this area. Small areas of ischemia, infarction, or both, may go unrecognized in the operating room. In the postoperative setting, either splenic ischemia, infarction, or both of these, will produce unexpected symptoms as described below. How often and whether these subjective complaints are evaluated for SI or simply managed with pain control is unknown.

Very little has been reported about SI as a complication of surgery. A single case of partial SI was reported in a series of 169 laparoscopic adrenalectomies.[10] This case was managed successfully with observation alone. The incidence of SI as a complication of LNF is unknown. Because the military medical system is defined by region and each region has a designated tertiary referral center, we can estimate the incidence of this entity. Within the southeastern region, complications are referred to only 1 tertiary military hospital. This hospital, Eisenhower Army Medical Center, received only 2 cases of SI following LNF during the past 3 years, 1998 to 2002, out of a total of 231 LNF completed within the region. Note that this does not include procedures that were converted to open Nissen fundoplication or those initiated using an open technique. We calculate the minimum incidence to be 2/231 or <1.0%. In actuality, the incidence is probably much

higher because many cases of SI may have gone undiagnosed.

Little information exists about the diagnosis and treatment of iatrogenic SI in the surgical literature. The best information available comes from experience with spontaneous (hematologic or thromboembolic) events and therapeutic (PSE) SI. Nonspecific complaints are present in the majority of cases of hematologic and thromboembolic SI. Left upper quadrant pain (67%), fever-chills (70%) and nausea-vomiting are common symptoms. But abdominal symptoms may be absent in 33% of embolic cases.[9] In autopsy series of SI, 10% to 50% of patients had no clinical symptoms that could be attributed to SI.[7,8] An unrecognized infarction that results following a laparoscopic procedure may be difficult to diagnose based on clinical presentation alone. Left upper quadrant abdominal pain, subjective changes in appetite and early satiety are common after LNF. Fever and an elevated WBC developing after esophageal mobilization would raise suspicion of an esophageal injury. Unless SI is suspected and properly investigated, the diagnosis would be easy to overlook.

The diagnosis of SI can be confirmed with an intravenous (IV) contrast CT scan, liver-spleen scan, ultrasound, or angiography. The classic pattern on CT scan demonstrates a low attenuation wedge of tissue surrounded by normal-appearing contrast enhanced splenic parenchyma. A word of caution, when the entire spleen is involved, the CT scan may fail to demonstrate this classic pattern. Other CT scan findings include cysts, abscesses, and gas formation within the splenic parenchyma.[6] In 1 series, the false-negative rate of CT scans was 25%.[7] Clinical judgment and use of alternate radiographic modalities may be necessary.

Diagnosing SI is important because patients deserve to know why they are having persistent symptoms, and complications that arise from SI should be recognized, anticipated, and treated. The natural history of SI can be extrapolated from the experience gained from cases of therapeutic PSE. In the 1980s, elective PSE was performed as an adjunct to renal transplantation, benign splenic pathology, and portal hypertension. Mozes et al[11] reported the results of elective PSE in a series of over 100 patients. They reported severe abdominal pain lasting between 2 to 5 days requiring epidural anesthesia to manage. Fevers (>100°F) and elevated white blood counts occurred following the procedure and lasted

between 2 and 4 days. In their experience, PSE in excess of 60% to 70% of the spleen volume resulted in a higher complication rate. Overall, they reported a 17% (17/101) complication rate to include splenic cysts (n=2), splenic abscesses (n=2), pancreatitis (n=3), and pleural effusions (n=10). Splenic cysts were associated with delayed mortality in 2 cases. They concluded that therapeutic PSE had a low complication rate (17%) but that when complications did develope prompt treatment with splenectomy was indicated.[11,12] PSE is presently used to treat hypersplenism in patients suffering from cirrhosis and portal hypertension. In 1 series of 17 patients, abdominal pain lasting 1 to 2 weeks and fever lasting up to 15 days was reported. Two severe complications (12%) were reported to include liver failure and splenic abscess.[13] In the trauma literature, PSE is used to manage blunt splenic injuries. Therapeutic embolization has been shown to improve nonoperative splenic salvage rates for patients suffering high-grade injuries.[14] Following PSE, suspicious radiographic findings are seen in 9% of cases (7/80) and include subcapsular fluid collections, cysts, abscesses, and intraparenchymal gas. Interestingly, these radiographic findings have been documented to occur up to 10 weeks after embolization.[6] Splenic artery embolization is also used as an adjunct to elective laparoscopic splenectomy. In these cases, the spleen is removed surgically after infarction and provides little useful clinical information about the management of chronic SI. From the PSE experience, we know that the initial procedure can be done safely, and the expected signs and symptoms can be managed. Unfortunately, an unpredictable delayed complication rate exists that may result in significant morbidity and even mortality.

After diagnosing postoperative SI, a trial of conservative management is recommended. Duration of symptoms is reported to be 2 days to 2 weeks in some historical series. In the series by Mozes et al,[11] involving over 100 patients, splenectomy was never required for pain control. In the series by Nores et al,[9] 34% of SI cases required splenectomy for persistent pain. These represent 2 different populations, the former being therapeutic PSE and the latter spontaneous SI. When to apply a surgical approach in cases of iatrogenic SI has yet to be clearly outlined. For severe and persistent pain lasting several weeks, a more aggressive surgical approach is warranted. These patients may benefit from early splenectomy.

Complications arising from SI occur in 17% to 20% of cases, and patients with large infarction volume (excess of 60% to 70%) have a higher complication rate.[7,9,11] The management of a recognized SI complication remains controversial. Splenic cysts and abscesses are the most commonly reported complications. Unfortunately, it is difficult to differentiate between these 2 conditions radiographically, and the natural history of these conditions may be unpredictable. Limited series report that percutaneous drainage can differentiate and treat these SI-related complications.[6,14] Splenic rupture, hemorrhage, and death are the most feared events arising from an SI-related complication. The incidence of these dramatic events ranges from 17% (2/12)[9] to 47% (14/30)[7] in retrospective series. The conservative approach is to perform splenectomy for any SI-related complication to include cyst or abscess formation, rupture, or hemorrhage. Others have questioned the need for splenectomy for asymptotic splenic cysts resulting from SI, stating that these lesions can be safely observed.[12] If an SI-related complication is to be managed conservatively, the patient and surgeon should be educated as to the risks of observation. When surgery is indicated, semi-elective splenectomy with preoperative immunization and full bowel preparation can be performed in the majority of cases.

In the cases presented here, SI caused symptoms that were severe enough to merit further investigation. Because symptoms had been present for over 6 weeks, these patients had already been observed and failed a trial of observation for "unrecognized" SI. One patient had persistent symptoms that were mild. This patient was managed conservatively and followed clinically without sequela. No SI-related complications have been identified to date. The second case presented with persistent symptoms and multiple admissions for pain and fever. CT scan and arteriography confirmed the diagnosis of SI. No radiographic evidence existed of cyst or abscess formation, and the clinical presentations (pain and fever) were nonspecific findings commonly associated with uncomplicated SI. 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. On final pathological review, a splenic abscess was present. Katkhouda et al[15] reported successfully removing an infarcted spleen with abscess formation by using laparoscopic techniques. The intense adhesion formation and inflammatory process may be variable, and the location of the infarction-abscess with respect to the adjoining

structures must be taken into account. At the present time, we recommend that the laparoscopic approach be used cautiously when performing elective splenectomy for iatrogenic SI.

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