CASE REPORT JSLS

Retraction-Related Liver Lobe Necrosis After Laparoscopic Gastric Surgery

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ABSTRACT

Background: Liver retraction is necessary for optimal exposure during laparoscopic gastric surgery. Though transient venous congestion of the retracted lobe of the liver is invariably seen during operations, major parenchymal injury is rare. We describe a case of Nathanson liver retractor-induced left lobe liver necrosis and review the pertinent literature.

Case Report: A 78-year-old man underwent a laparoscopic-assisted total gastrectomy for gastric cancer. A Nathanson liver retractor was used to retract a large fatty left liver lobe. The operation was prolonged due to splenic bleeding requiring splenectomy. On the second postoperative day, the patient deteriorated rapidly and developed multi-organ failure. A computerized tomogram confirmed necrosis of the left lobe of the liver with gas in the liver parenchyma. The necrotic liver lobe was excised at reoperation. The patient died from a postoperative myocardial infarction.

Discussion: Though minor liver injuries, in the form of intraoperative trauma and congestion, are common with laparoscopic liver retraction, major lacerations and necrosis are rare. Prolonged surgery and enlarged fatty liver lobe increases the risks of major injury. In our report, we discuss various types of retractor-related liver injuries and their management and highlight the importance of intermittent release of retraction during prolonged surgery.

Key Words: Laparoscopic surgery, Liver, Injury, Necrosis.

INTRODUCTION

Laparoscopic gastric surgery requires adequate visualization of the diaphragmatic hiatus and upper stomach, which is usually concealed under the left lobe of the liver. Retraction of this lobe is paramount for satisfactory access to this region. Such retraction is achieved by different techniques, which can vary from simple grasping of the parietal peritoneum anterior to the hiatus with a toothed grasper traversing underneath the left lobe of the liver to various flexible or fan-shaped purpose-built retractors. Another retractor, which is commonly used for this purpose is the Nathanson retractor. The Nathanson retractor is a curved metal blade that lifts the left lobe of the liver and opposes it against the diaphragm, whilst it is externally fixed to a supporting metal arm. Such retraction can cause significant pressure on the liver parenchyma, because it is compressed between the metal blade of the retractor and diaphragm, particularly when the patient is positioned in the reverse Trendelenburg for a prolonged period. Compression-related injury and parenchymal fractures are more likely if the liver lobe is enlarged, fatty, and fragile.[1]

Minor liver injury in the form of venous congestion is invariably seen during such laparoscopic procedures, because the liver parenchyma is trapped by the retractor. Fortunately, such minor pressure-related injuries are usually temporary and without any major clinical significance. Clinically, they are reflected as a transient elevation of liver enzymes, in particular, aspartate aminotransferase (AST). This benign phenomenon has been described previously as retraction transaminitis.[2] Evidence of such minor pressure-related retractor injury has also been found incidentally on serial postoperative CT scans.[3]

Major lacerations and parenchymal bleeding occur rarely and are identified during procedures. Like most traumatic liver injuries, these are usually amenable to conservative treatment.

Delayed necrosis of the left lobe of the liver due to venous infarction is a rare complication of laparoscopic liver retraction and can have serious implications on the outcome. We present a case of such liver necrosis related to Nathanson retractor used during a prolonged and difficult

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laparoscopic gastrectomy along with a review of the pertinent literature, highlighting the safety issues with the use of laparoscopic liver retraction.

CASE REPORT

A 78-year-old man with gastric cancer underwent a planned laparoscopic-assisted total gastrectomy. Pre-existing comorbidites included type II diabetes mellitus, ischemic heart disease (previous myocardial infarction 12 years earlier, and occasional ongoing angina), and benign prostatic hypertrophy. He had also had an open cholecystectomy several years earlier.

The operation was performed with the patient under general anesthesia with epidural analgesia. Initial laparoscopic findings confirmed no evidence of peritoneal or occult spread of cancer, but revealed some adhesions around the cholecystectomy site and dense perisplenic capsular adhesions. The liver was fatty with a bulky left lobe. The left lobe of the liver was retracted using the Nathanson retractor (Cook Ireland Ltd, Limerick, Ireland), secured externally through a Murdoch mechanical arm (Cook Ireland Ltd, Limerick, Ireland). The Nathanson retractor was inserted just below the xiphisternum through a 5-mm trocar puncture and placed under the left lobe of the liver, retracting it antero-superiorly to expose the hiatus. With the patient in the reverse Trendelenburg position, laparoscopic mobilization of the stomach and omentum was then carried out, and the first part of the duodenum was divided by using a stapling device and oversewn. Splenic capsular bleeding was encountered during mobilization of the short gastrics. This could not be controlled with conservative techniques and necessitated laparoscopic division of the splenic vessels and subsequent splenectomy. A small epigastric transverse incision was then made to remove the stomach and spleen. A Roux-en-Y esophagojejunostomy was created and a feeding jejunostomy tube sited. The total laparoscopic time of the operation was 3.5 hours.

The patient was cared for in the intensive care unit postoperatively where he remained hypotensive for the first 18 hours, requiring increasing inotropic support. By 24 hours after the operation, his blood pressure stabilized. Serum AST levels had shot up rapidly when checked 5 hours after the operation and then gradually came down over the next 2 days **(Figure 1)**. In contrast, serum levels of alkaline phosphatase, gamma-glutamyltransferase, total bilirubin, and C-reactive protein were slow to rise and fell only gradually **(Figure 2)**.

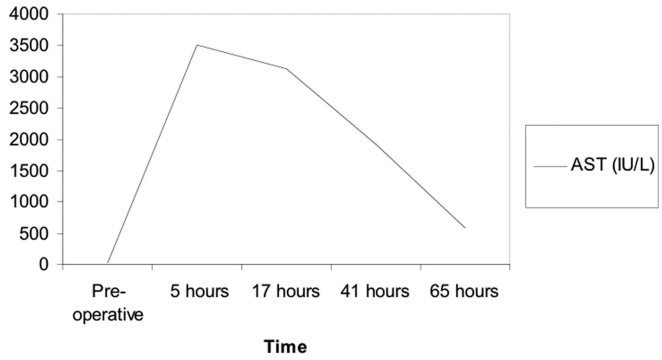

Figure 1. Temporal changes in serum aspartate transaminase (AST). Greater than 20 times elevation of AST level was reached at 5 hours after the operation (AST normal level = 5-55 IU/L).

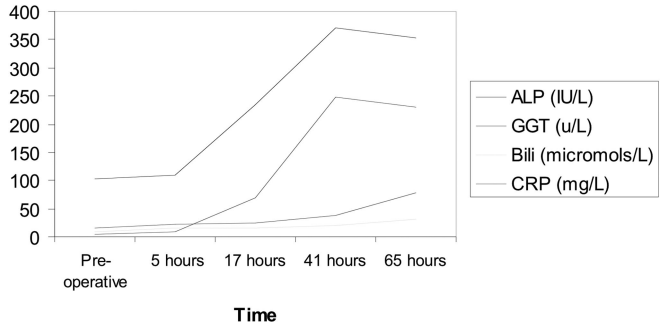

Figure 2. Postoperative changes in the serum levels of liver and inflammatory markers. (ALP: alkaline phosphatase; GGT: gamma glutamyltransferase; Bili: total bilirubin; CRP: C-reactive protein).

On the first postoperative day, there were no remarkable abdominal findings, and the patient was started on jejunostomy feed. However, by 48 hours after the operation, the patient had developed tachycardia and fever (temperature 38°C). Over the next 6 hours, he rapidly deteriorated, requiring large doses of inotropes and ventilatory assistance.

Because no obvious cause was evident clinically, the suspicion of anastomotic dehiscence was raised, and the patient went on to have an urgent noncontrast abdominal computerized tomogram (CT). The CT scan did not show any obvious evidence of anastomotic leak, but demonstrated massive destruction in the left lobe of the liver with large loculations of intraparenchymal gas as well as gas in the portal radicals **(Figure 3)**.

In view of the CT finding of gas-filled liver necrosis and the presence of septic shock, a decision was made to perform an exploratory laparotomy. At reoperation, the

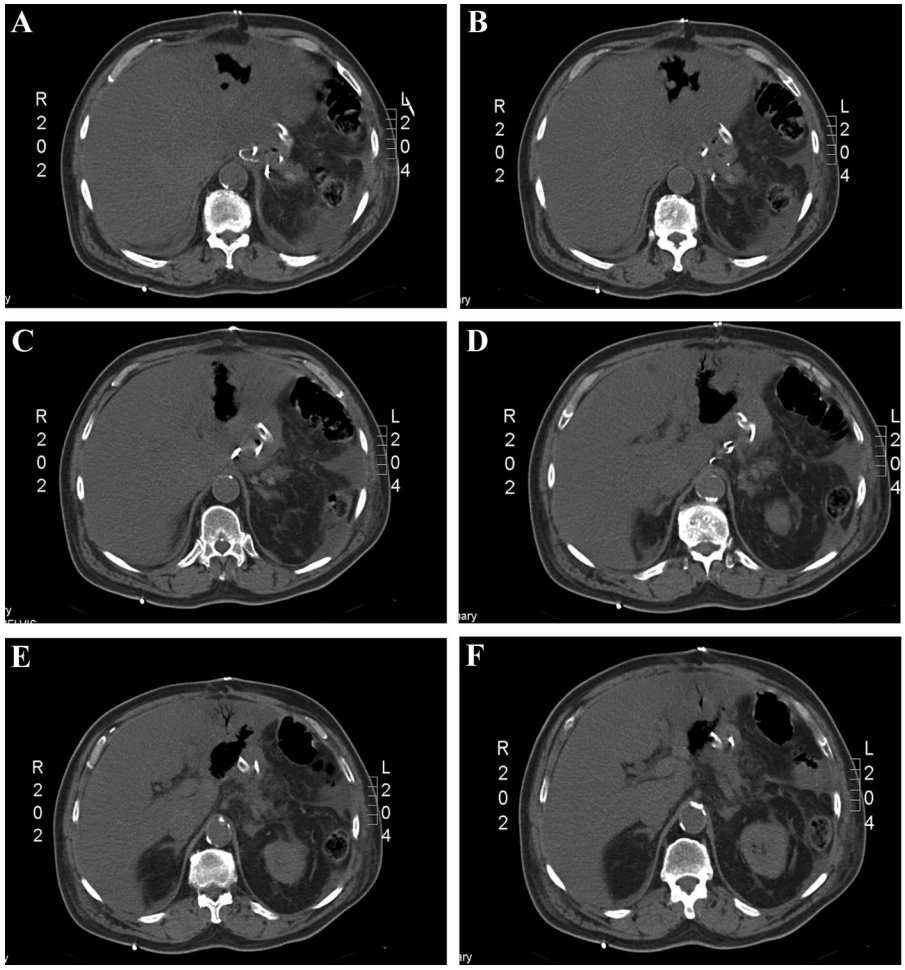

Figure 3. Sequential cephalad (a) to caudad (f) noncontrast CT scan images demonstrating destruction of the left lobe of the liver with free intraparenchymal gas. In addition to a large loculus of gas, a branching linear pattern of gas with the portal radicals is seen clearly (e). Also note the absence of spleen, absence of any localized collections, inflammatory stranding in left subhepatic space along the drain placed towards the hiatus.

left lobe of the liver was necrotic and turgid. A left lateral sectionectomy (segments 2,3) was performed.

The patient remained on inotropic support postoperatively and died from a myocardial infarction 14 hours later.

DISCUSSION

Pertinent literature review performed by using an unrestricted PubMed search on the subject confirmed that retraction-related liver injuries are rare. From our experience and review of the available literature, we can classify liver injuries secondary to mechanical retraction of the liver during laparoscopic surgery into 3 types. These are in the form of minor congestion injury, traumatic parenchymal rupture, and delayed liver necrosis.

The commonest form of retraction-related injury is secondary to parenchymal congestion and is evident during the procedure by the appearance of bluish discolored swollen liver tissue trapped lateral to the retractor blade. Usually this injury is reversible, depending on the duration of retraction and the amount of liver tissue trapped, and may not have any postoperative clinical significance. Usually it causes the asymptomatic rise in the serum levels of aspartate aminotransferase.[2] If the congestion is severe enough to cause small parenchymal venous infarctions, it may still remain asymptomatic, but may be picked up as a radiological abnormality incidentally on postoperative CT scans.[3]

The second type of retraction injury is uncommon and is a result of retraction-related parenchymal fracture or tear.

These are more likely when the liver is tense and fragile due to fatty enlargement. These can occur as a direct injury by the blade of the retractor or indirectly due to retraction of adjacent structures.[4-6] Most of such parenchymal trauma is identified on its occurrence and only causes bleeding, which is often self-limiting. Infrequently, such direct retraction-related parenchymal injury presents postoperatively due to slowly developing subcapsular hematoma.[7,8]

The third type of injury occurs from substantial parenchymal necrosis and usually presents late after the initial procedure. Such injuries are typically identified on a postoperative CT scan and may have variable morphological features.[9-11] These infarcts vary from wedge-shaped lesions, to rounded lesions or to irregular geographic lesions. Finding of gas in the area of infarction is usually considered a sign of infection. A chronologically sequential review of CT scans of 21 liver infarctions ranging over a period of 2 days to 53 days by Stewart and colleagues[11] suggests that liver infarcts primarily do not have any gas pockets in them, but only acquire them once infection sets in. Such infected infarctions can have gas pockets in the parenchyma, in well-formed abscess cavities or in the biliary and portal radicals in the nondependent part of the involved segment.[11] Portal venous gas can also occur secondary to severe portal pyemia or due to bowel ischemia and occasionally due to certain benign conditions.[12]

Liver infarcts can be treated conservatively but may get complicated by bile leak and secondary infection. Both of these complications can be treated by percutaneous drainage but surgical intervention may be necessary.[11,10,13]

Retraction-related liver infarction has been described during open gastric surgery as well.[13] In their study, Kitagawa and Iriyama[13] describe 2 cases of left lobe infarctions after open gastrectomy related to infolding and medial retraction of the left lobe of the liver and compare them with postoperative hepatic infarctions related to direct injury to the hepatic artery. Similarly, liver necrosis can occur postoperatively after liver transplantation or Whipple's surgery due to vascular insufficiency related to interference near the porta or hepatoduodenal ligament.[11] These cases of liver necrosis due to operative vascular injury should be considered distinctly from retractor-related necrosis. Anesthetic agents can also cause massive liver necrosis and can complicate laparoscopic surgery but need to be identified as a distinct entity.[5]

In our review of the published literature for retractor-related major liver necrosis, we found only 2 confirmed cases during open surgery, and both involved the left lobe of the liver.[13] We picked up 3 other cases involving necrosis of the left lobe of the liver after laparoscopic gastric bypass in an article[10] describing normal and abnormal CT scan findings after laparoscopic gastric bypass surgery. This report by Yu et al[10] focuses on the CT scan findings in consecutive patients who had a CT scan after gastric bypass and does not explore the causes of the CT-detected abnormalities. It has to be assumed that these 3 cases after laparoscopic bariatric surgery, developing postoperative liver necrosis in the lateral segment of left lobe are retractor-related injury. Our case is the first formally reported case of massive liver necrosis secondary to Nathanson retractor injury after laparoscopic liver retraction.

Our patient had prolonged retraction with a Nathanson retractor while in the reverse Trendelenburg position, which has the effect of compression of the left lobe of the liver between the diaphragm and the retractor blade against the pressures generated by the weight of the patient. The patient developed a remarkable rise in the serum level of AST **(Figure 1)**. The peak level was >20 times the upper limit of normal, which is considered diagnostic for ischemic hepatitis in the absence of other causes of hepatitis.[14] This level of AST is much higher than found in cases of transient transaminitidis, where the levels remained <6 times the upper limit of normal.[2]

Though liver necrosis can be treated conservatively, the CT scan confirmed free gas in the liver parenchyma, which is usually considered an indication for intervention. Because the patient was catastrophically unwell by the time of his CT scan, and we could not exclude any other intraabdominal source of sepsis or anastomotic ischemia, we had to subject our patient to emergency laparotomy. We were able to excise the necrotic liver; however, the patient died from a postoperative myocardial infarction.

CONCLUSION

Massive liver necrosis following laparoscopic liver retraction is rare and can be catastrophic. In suitable patients, it may be amenable to conservative treatment or percutaneous drainage with others requiring segmental hepatic resection. Prolonged surgery, fatty liver, and the reverse Trendelenburg position increase the risk of such injury. We recommend that the Nathanson liver retractor should be removed intermittently during prolonged surgery in a similar situation to re-establish hepatic lobe circulation.

Our article is the first reported case of such massive liver necrosis due to sustained pressure injury from Nathanson

liver retractor used in laparoscopic gastric surgery. Laparoscopic gastric surgery is being increasingly performed using such liver retractors, and simple steps necessary to prevent such injury are often overlooked. This report increases the awareness of these injuries, which we believe are underreported and can be catastrophic in nature.

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