ABSTRACT

The accumulation of chylous fluid in the abdominal cavity is an infrequent, yet alarming, complication in abdominal surgery. Laparoscopic fundoplication has assumed a central role in the surgical treatment of gastroesophageal reflux disease and is significantly altering the balance of therapy toward more common and earlier surgical intervention. We report the case of a 67-year-old woman with gastroesophageal reflux disease and intense esophagitis who underwent a laparoscopic Nissen fundoplication in February 2000. The procedure was performed without apparent complications. Twenty days later, the patient complained of abdominal pain and distension. Ultrasonography showed ascites, whereas endoscopic and radiological exploration of the fundoplication demonstrated no abnormalities. A paracentesis was performed, which showed a milky fluid with high concentrations of triglycerides (1024 ng/dL) and cholesterol (241 ng/dL). The patient was treated successfully with total parenteral nutrition for 3 weeks, followed by a low-fat diet. To our knowledge, this is the third reported case of chylous ascites after a Nissen fundoplication and the second case after laparoscopic fundoplication. The development of chylous ascites seems to be related to the injury of lymphatic vessels, including the thoracic duct, during the retroesophageal window dissection. The careful dissection and judicious use of diathermy is proposed to prevent this rare complication.

Key Words: Chylous ascites, Complications of Nissen fundoplication, Laparoscopy.

INTRODUCTION

Chylous ascites is a rare clinical condition that occurs as a consequence of disruption of the abdominal lymphatics. It is a challenging clinical entity from a therapeutic standpoint. Multiple causes have been described in the literature, including abdominal surgery, malignant neoplasms (the most common cause in adults), blunt abdominal trauma, spontaneous bacterial peritonitis, cirrhosis, pelvic irradiation, peritoneal dialysis, abdominal tuberculosis, and congenital defects in lacteal formation.1,2

Laparoscopic surgery for gastroesophageal reflux is now second only to biliary tract surgery in the frequency of minimally invasive procedures performed in everyday surgical practice.3 This procedure has been successfully performed laparoscopically since 1991. The main benefits of this approach are the reduction of wound-related morbidity, the short hospital stay, and the quicker return to full activity.4,5 The most common complications after laparoscopic fundoplication (LF) are pneumothorax, pneumomediastinum, esophageal or gastric perforation, bleeding, and even mesenteric thrombosis. Herein, we discuss an uncommon complication that has been reported only twice in the literature.

CASE REPORT

A 67-year-old woman with intense esophagitis (grade III of Savary) and a mild hiatal hernia underwent a laparoscopic Nissen fundoplication in February 2000. Her most important clinical symptoms were retrosternal pain, acid and food regurgitation, and heartburn. She was also dependent on proton-pump inhibitors for symptom relief.

No problems or difficulties occurred during the procedure. At laparoscopy, no abnormality was found. Her immediate postoperative period was unremarkable. A hook electrocautery was used for dissecting the diaphragmatic crura and opening the retroesophageal window.

Twenty days after the surgery, the patient complained of abdominal pain and distension. Physical examination...
revealed an increased abdominal girth, ascites, and exaggerated tympanism. Diffuse ascites was also confirmed by an ultrasound examination. A paracentesis was performed and showed a white-milky odorless fluid with a specific gravity of 1.030, sodium 135 mEq/L, potassium 3.6 mEq/L, total protein 4.4 g/L, amylase 25 U/L, triglycerides 1024 ng/dL, cholesterol 241 ng/dL, red blood cell count of 3450, and white blood cell count of 2150 (70% lymphocytes). Electrophoresis of the fluid showed that lipids were mainly in the form of chylomicrons. Gram stain and bacterial cultures were negative.

A central line was placed and parenteral nutrition started. Over the next 3 weeks, the abdominal girth improved with decreased firmness and increased bowel sounds. The central line was removed on day 20 and oral fat-free diet restarted. One-month later, abdominal ultrasound demonstrated no ascites.

**DISCUSSION**

The anatomic relationship between the thoracic duct and esophagus shows that although lymphatic injury is a possible complication of a Nissen-type fundoplication, it should be rare. The lumbar, intestinal, liver, and inferior intercostal lymphatics drain into the cisterna chyli (anterior to the second lumbar vertebra), which is located between the origin of the abdominal aorta and azygos vein and is also located posterior to the right crus of the diaphragm. This collectively forms the thoracic duct, which passes through the aortic hiatus and courses in the right posterior mediastinum cephalad to the junction of the left subclavian and left internal jugular veins, where it empties.\(^1\)\(^2\) Lymphatic channels in the abdomen follow the course of the blood vessels, and after passing through numerous lymph nodes, come together to form the thoracic duct. Recent studies have shown that a true cisterna chyli is absent from most dissections and the thoracic duct is formed directly by the collecting lymphatic channels.\(^6\)

The thoracic duct carries lymphatic drainage from all of the body except the right side of the head and neck, right arm, and right side of the thorax. Fifty to 90% of the thoracic duct lymph is derived from intestinal and hepatic lymphatics.\(^1\)\(^2\) Blalock\(^7\) performed thoracic duct occlusion experiments and concluded that thoracic duct obstruction alone was not sufficient to cause chylous ascites. Patients with limited-reserve anastomotic channels are more at risk of developing persistent effusions/ascites when obstruction or division of lymphatic channels occurs.

Lymph is usually colorless, but when it leaves the small bowel it is saturated with minute particles of fat (chylomicrons). Paracentesis and fluid examination are essential in the diagnosis of chylous ascites. Specific gravity greater than 1.012, total protein greater than 30 g/L, alkalinity, and sterility are characteristic of chylous fluid. Normal lymph flow through the thoracic duct averages 1.0 mL/kg per hour and may be as high as 200 mL/kg per hour after a fatty meal. Long-chain triglycerides are carried in the lymphatics to the venous system. Medium-chain triglycerides are absorbed directly into the portal venous system, bypassing the lymph system and reducing the amount and flow of chyle. Elemental diet reduces the chyle flow with this mechanism. Total parenteral nutrition diminishes chyle production and flow by allowing the bowel to rest. Aggressive nutritional repletion is essential in massive chylous ascites. Significant protein loss occurs.\(^1\)

Postoperative chylous ascites is a rare complication after several retroperitoneal operations, such as lymphadenectomy, pancreaticoduodenectomy, distal spleen-renal shunt, and aortic surgery.\(^4\) The prognosis of chylous ascites is mainly dependent on the cause. Patients with malignant neoplasms as the cause and infants with congenital lesions do poorly. These patients need aggressive treatment, and peritoneovenous shunting may be needed to maintain nutritional integrity. Postoperative chylous ascites has a favorable prognosis.\(^1\)

Parys and Hart\(^2\) reported a case of a 22-month-old girl who developed chylous ascites 3 weeks after an open Nissen fundoplication. She was successfully treated with parenteral nutrition and a fat-free diet.

Al-Mousawi and Abu-Nema\(^6\) reported a case of chylous ascites after truncal vagotomy. They concluded that chylous ascites occurred because of an injury to an aberrant lymphatic trunk at the lower end of the esophagus. The patient was also treated with conservative therapy.

Slim et al\(^4\) reported the unique case of chylous ascites after laparoscopic Nissen fundoplication. They describe the mechanism of lymphatic leakage as an injury of the cisterna chyli or thoracic duct during the retroesophageal dissection (with scissors or monopolar dissection). Opening the retroesophageal window too far posteriorly exposes the lymphatics to injury. The use of electro-
cautery in this area is another possible mechanism of lymphatic damage. Alternatively, obstruction/injury of the thoracic duct may have happened when the posterior crura were sutured together. Careful dissection in a correct plane behind the esophagus and minimal use of monopolar coagulation are the 2 most important measures for preventing this complication.

Treatment of chylous ascites includes both surgical and dietary manipulations. Surgical treatment is indicated in a patient when a surgically correctable lesion is present, persistence of symptoms are unresponsive to a low-fat, long-chain triglyceride diet after several months of parenteral nutrition has been instituted, and deterioration of clinical status has occurred in patients with undetermined causes. Some authors advocate the use of isosulfan blue dye injected through foot lymphatics to identify the site of a leak during surgery. Peritoneovenous shunts have been performed with variable reported success, and complications include occlusion, sepsis, and disseminated intravascular coagulation.

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