CHYLOUS ASCITES FOLLOWING OPEN ABDOMINAL AORTIC ANEURYSM REPAIR: AN UNUSUAL COMPLICATION

Georgios Galanopoulos, M.D., Ph.D.[a,b]; Theofanis Konstantopoulos, M.D.[a]; Stavros Theodorou, M.D.[a]; Ioannis Tsoutsas, M.D.[a]; Dimitrios Xanthopoulos, M.D., Ph.D.[a]; Elias Kaperonis, M.D., Ph.D.[a]; Vassilios Papavassiliou, M.D., Ph.D.[a]

[a]Sismanogleio-Amalia Fleming General Hospital of Athens, Athens, Greece; [b]Medical School of Athens, Athens, Greece

Abstract

Chylous ascites is a rare complication after abdominal aortic aneurysm repair. Accumulation of chyle within the close space of the peritoneal cavity may cause severe discomfort to the patient, complicating the postoperative course. Prompt diagnosis is needed to adopt measures for reducing lymph leakage and contributing to lymphatic fistula closure. Fortunately, conservative treatment is successful in the majority of cases. In the rare cases that do not respond to conservative treatment, surgery becomes mandatory. Accurate preoperative localization of lymph leakage is a prerequisite for a successful outcome. Postoperative chyloperitoneum has a benign course and an excellent prognosis.

Introduction

Chylous ascites, or chyloperitoneum, is defined as the accumulation of chyle within the peritoneal cavity. Chyle is a characteristic milky fluid with a high triglyceride concentration. Chylous ascites has numerous causes, including infectious, neoplastic, inflammatory, cardiac, gastrointestinal, or cirrhotic. It may also be congenital in the context of a syndrome in cases that involve children. On rare occasions, chylous ascites develops secondarily after surgical procedures such as radical nephrectomy, pancreaticoduodenectomy, retroperitoneal lymphadenectomy, and gastrectomy. Open repair of an abdominal aortic aneurysm tends to conceal such a complication even though it is rare.

Anatomic and Physiologic Remarks

Lymph is a fluid containing proteins, immunoglobulins, lymphocytes, and products of the digestive process, such as lipids. Triglycerides create a characteristic milky appearance in the lymph. Chylous leakage is accompanied by loss of lipids, proteins, and electrolytes, resulting in nutritional defects and disturbance of the electrolytic equilibrium. Loss of immunoglobulins and lymphocytes renders the patient more susceptible to infections. This immunologic deficit coupled with immunosuppression induced by surgery could expose the patient to severe infectious complications.

Lymph flows from lymphatic vessels into lymphatic trunks and then into the thoracic duct. Lymph nodes are dotted through the course of the lymphatic channels, draining the lymph. The lumbar lymphatic vessels derive from the common iliac lymphatics, draining the lymph from the lower extremities, pelvis, and genitalia.[1] These vessels are located between the aorta and the inferior vena cava and are joined by the lymphatic glands of the abdominal and retroperitoneal organs, so that the lymph from the lower part of the body drains into the thoracic duct. The thoracic duct, which is the largest lymphatic channel in the body, originates from the cisterna chyli (cistern of Pecquet), which is a sacciform structure located anterior to the bodies of the first and second lumbar vertebrae. About half of all individuals lack a true cisterna chyli. Instead of this anatomic structure, there is a confluence of lymphatics from which the thoracic duct originates.[2] The thoracic duct crosses the aortic hiatus of the diaphragm to enter the right posterior mediastinum.[1] At the level of the fifth thoracic vertebra, it crosses to the left, entering the superior mediastinum behind the aortic arch. It terminates at the junction of the left jugular and left subclavian veins, communicating with the venous system. It is important to emphasize that the major part of the lymph transported by the thoracic duct derives from the gut and liver and contains lipids in the form of chylomicrons.

Aortic Repair and Chylous Ascites

Abdominal aortic aneurysm (AAA) repair is considered to be the most frequent cause of postoperative chyloperitoneum, accounting for about 80% of cases. It is expected that an open AAA repair endangers the lymphatic vessels of this area. High proximal aortic clamping may damage the cisterna chyli or the lumbar lymphatics, while surgical preparation of common iliac arteries may do the same. Up to 50% of patients undergoing high aortic dissection present lymph leakage. Things become worse in the case of abdominal aneurysm rupture since exposure of the aorta must be rapid in order to control bleeding. The risk of chylous ascites seems to be significantly increased in this case. Resection of an inflammatory aortic aneurysm also increases the risk of chylous ascites. Inflammatory reaction in the surrounding (periaortic and retroperitoneal) tissues may involve the lymphatics, which can be easily resected in the course of the operation resulting in lymph leakage. Since inflammatory AAAs account for about 5% of all AAAs, chylous ascites becomes even more rare.

The advent of the endovascular approach to treating AAA drastically reduced the incidence of iatrogenic chyloperitoneum. It is interesting to note that some authors believe that the retroperitoneal approach in the treatment of AAA is less traumatic to the lymphatics and results in a lower incidence of postoperative chyloperitoneum.[3]

Epidemiology

As expected, epidemiological data are almost nonexistent due to the rarity of this complication. Only 9 cases of chylous

ascites after AAA repair were reported in the literature up until 1987, with a total of 38 cases reported up to 2008.[4] It is likely that this complication is underestimated since lymph channel damage is sometimes unavoidable during AAA repair. However, spontaneous healing of these lymphatics in the early postoperative period keeps this complication clinically silent. Mortality rates of chylous ascites following abdominal aortic surgery vary between 11% and 18%.[5]

Clinical Presentation and Diagnosis

Symptoms of chyloperitoneum usually occur within 1 or 2 weeks after surgery, with patients usually complaining of weight gain and abdominal distension. In addition, the progressive increase of intra-abdominal pressure causes patients to complain of dyspnea and discomfort. Abdominal distention is commonly painless. Other nonspecific symptoms include nausea, vomiting, and early satiety. A high index of suspicion is required in order to promptly diagnose this nosological entity.

Paracentesis of the intra-abdominal fluid is of paramount importance in the diagnosis of chyloperitoneum. Analysis of macroscopic and microscopic fluid characteristics could confirm clinical suspicion. A milky appearance with an elevated content of triglycerides and chylomicrons is typical and diagnostic. Triglyceride levels are greater than 200 mg/dL.[6] Protein content varies between 2.5 and 7 g/dL, with a mean of 3.7 g/dL,[7] while the serum/chylous albumin gradient is less than 1.1.[6] Leucocytes are present with lymphocytic predominance. Cholesterol level is typically low,[7] and glucose content is less than 100 mg/dL.

Computerized tomography (CT) is an important diagnostic tool, although it has low specificity.[1] However, in the case of strong clinical suspicion, it offers useful diagnostic elements. First, it confirms the presence of ascites. It also reveals extra- and intraperitoneal collection of fluid. Chyle can be distinguished from blood on CT because of the difference in density; chyle has a low attenuation on CT. On the other hand, its density has the appearance of water, rendering it indistinguishable from other biological fluids such as bile or simple ascites. Other findings on CT include pleural effusion.

Lymphoscintigraphy or lymphangiography are not easily available in the everyday clinical practice, but in cases of persistent chylous leakage in the abdomen, accurate localization of the source of chylous leak is extremely important for subsequent surgical repair. Lymphoscintigraphy allows functional assessment of lymph transport and is performed by injecting a tracer (^{99m}Tc) intradermically at the dorsal surface of the feet. Lymphoscintigraphy can be used in cases of lymphangiography contraindication.[2]

Management

Treatment of chyloperitoneum can be conservative or invasive. Every effort should be made to treat all cases conservatively before resorting to invasive modalities to interrupt lymph leakage.

In the majority of the published cases, conservative measures led to complete resolution. The first target of treatment is lymph flow decrease into the cisterna chyli to permit healing of the defect. This measure reduces the leakage of lymph into the peritoneal cavity. It has been observed that postprandial flow of chyle can reach values greater than 200 mL/min, especially after a fatty meal. Consequently, accumulation of a large quantity of chyle into the peritoneal cavity may be rapid. To reduce lymph flow, patients are placed on a low-fat diet with medium-chain triglycerides and high protein content.[6] During digestion, long-chain triglycerides

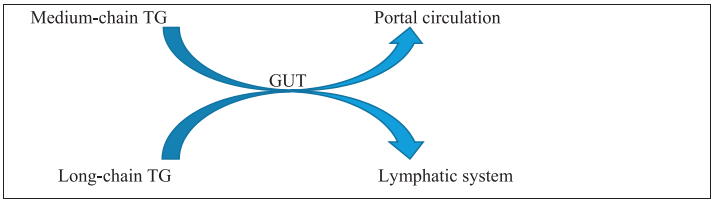

Figure 1. Destination of triglycerides (TG) accordingly to their molecular weight.

are converted into monoglycerides and free fatty acids. Passing through the epithelial cells of the gut, these molecules are combined again to form triglycerides, which are transported as chylomicrons to the intestinal lymphatics. Due to their lower molecular weight, medium- and short-chain triglycerides (such as fats derived from butter) can be absorbed by mucosal cells of the gut and transported as free fatty acids and glycerol to the liver through the portal circulation (Figure 1).[8] Medium- and short-chain triglycerides are more hydrosoluble than long-chain triglycerides. This property permits their direct diffusion from the epithelial cells to the blood in the capillaries of the intestinal villi, so the intestinal lymphatic flow is bypassed.

Patients who are nonresponsive to alimentary restriction should receive total parenteral nutrition (TPN). TPN corrects all nutritional deficits and significantly reduces lymph flow, thus permitting the closure of the lymphatic defect. It is believed that combined treatment with TPN and medium-chain triglycerides may be of benefit in more severe cases. TPN alone or in combination with medium-chain triglycerides or paracentesis could have a success rate of 60% to 100% after 2 to 6 weeks of treatment.[1] According to some authors, TPN should start immediately as a first-line treatment after diagnosis of chyloperitoneum[5] and continue for 2 to 3 weeks. Subsequently, a low-fat diet with medium-chain triglycerides should be continued for at least 3 months.

Hormonal therapy with somatostatin or its analogue, octreotide, with a longer half-life, is useful in cases nonresponsive to dietary restriction or TPN. Somatostatin or octreotide could also be administered in combination with TPN. The goal of somatostatin/octreotide administration is lymph flow reduction to achieve lymph fistula closure. The use of these hormones is based on the fact that intestinal lymphatics have somatostatin receptors. Octreotide is used at a dose of 100 mcg three times a day subcutaneously.[9-11]

Apart from its diagnostic value, paracentesis may also have a therapeutic role. Decompression of symptomatic chylous ascites, repeated several times, may provide relief of symptoms and have a transitory palliative effect.

It is generally accepted that 6 to 8 weeks of conservative treatment should be undertaken,[12] although the literature reports that the mean time to heal with conservative treatment is 90 days. If symptoms persist despite the above-mentioned conservative measures, surgical treatment may be necessary. In this case, laparotomy would be the favored approach, although a repeat surgery in these patients increases the mortality and morbidity rates. Precise identification of the source of lymph leakage is important in order to guide surgical repair. To this end, a lymphangiogram or lymphoscintigraphy prior to surgery are useful tools. Administration of 60 g of butter 4 hours before surgery may also be useful since it increases flow of the milky fluid, making it easier to localize the leakage[13]; failure to localize the site of injury leads to treatment failure. Surgical treatment includes suture ligation of all leaking lymph channels.

In patients with deteriorated general condition who are not considered fit for surgery, the use of peritoneovenous shunting could be useful. Long catheters are used to drain lymph from the peritoneal cavity into the venous system. However, frequent complications with this approach, including infection, air embolism, and obstruction of the shunt, increase morbidity and mortality and thus limit its use.[2] Therefore, peritoneovenous shunts should be used only as a last resort.

Another technique that has been used to treat chylous ascites secondary to laparoscopic gastric surgery is percutaneous lymphatic embolization with cyanoacrylate glue mixed with ethiodol. Lymphography is performed to locate the lymph leakage, and closure of the lymph fistula is achieved through percutaneous glue embolization.[14]

Conclusion

Chylous ascites is a rare complication following AAA repair. This disorder should be taken into consideration in patients who experience persistent abdominal distension, which often clinically manifests 2 weeks after aortic surgery. In an old paper, Gaylis stated that high abdominal aortic dissection is almost always accompanied by lymphatic vessel damage leading to limited lymph leakage.[15] This leakage is clinically insignificant. Spontaneous healing in the early postoperative period is frequent and due primarily to abstinence from oral intake. On the rare occasion that this does not occur, a chyloperitoneum develops. The outcome is usually benign, and resolution with conservative treatment is the rule. Surgery plays a role in severe cases that are nonresponsive to conservative therapy.

Conflict of Interest Disclosure: The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords: chylous ascites, chyle, abdominal aortic aneurysm, open repair, complication

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