Diagnosis and Management of Chylous Ascites

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ABSTRACT

Chylous ascites (CA) is a rare form of ascites that results from the leakage of lipid-rich lymph into the peritoneal cavity. This usually occurs due to trauma and rupture of the lymphatics or increased peritoneal lymphatic pressure secondary to obstruction. The underlying etiologies for CA have been classified as traumatic, congenital, infectious, neoplastic, postoperative, cirrhotic, or cardiogenic. Since malignancy and cirrhosis account for about two-thirds of all the cases of CA in Western countries, CA is classified according to portal and non-portal etiology. The diagnosis of CA is based on the distinct characteristic of the ascitic fluid which includes a milky appearance and a triglyceride level of > 200 mg/dL. The management consists of identifying and treating the underlying disease process, dietary modification, and diuretics. Treatment with nutritional optimization and management of the underlying etiology are the cornerstones of therapy. When conservative measures fail, other interventions such as octreotide/somatostatin analogues, surgical ligation, embolization, and transjugular intrahepatic portosystemic shunt in patients with cirrhosis can be considered.

Keywords: *chylous ascites, portal hypertension, ascitic fluid, lymphatic system*

ABSTRAK

Chylous ascites (CA) adalah bentuk asites yang jarang akibat dari kebocoran getah bening yang kaya akan lipid ke dalam rongga peritoneum. Hal ini biasanya terjadi karena trauma dan pecahnya limfatik atau peningkatan tekanan limfatik peritoneum sekunder akibat obstruksi. Etiologi yang mendasari CA telah diklasifikasikan antara lain trauma, kongenital, infeksi, neoplastik, pasca operasi, sirosis, atau kardiogenik. Keganasan dan sirosis menyumbang sekitar dua pertiga dari semua kasus CA di negara-negara Barat, CA diklasifikasikan berdasarkan etiologi portal dan non-portal. Diagnosis CA didasarkan pada karakteristik khas cairan asites seperti susu dan kadar trigliserida >200 mg/dL. Penatalaksanaan terdiri dari identifikasi dan mengobati proses penyakit yang mendasari, modifikasi diet, dan diuretik. Pengobatan dengan optimalisasi nutrisi dan pengelolaan etiologi yang mendasari adalah landasan terapi. Ketika tindakan konservatif gagal, intervensi lain seperti analog octreotide/somatostatin, ligasi pembedahan, embolisasi, dan pirau portosistemik intrahepatik transjugular pada pasien dengan sirosis dapat dipertimbangkan.

Kata kunci: *chylous ascites, hipertensi portal, cairan asites, sistem limfatik*

INTRODUCTION

Chylous ascites (CA) is the accumulation of lipid-rich lymph fluid in the peritoneal cavity due to disruption of the lymphatic system such as obstruction and traumatic injury. The incidence of CA in 1992 was reported to be approximately 1 in 20,000 hospitalized patients in major hospitals in the past 20 years. The incidence of CA tends to increase because of the increased survival of cancer patients who have undergone surgical intervention. CA formation is caused by a disruption of the lymphatic system.[1,2]

Malignancy is one of the most common causes of CA, especially malignant lymphoma with an incidence rate of about 50%, which can lead to fibrosis of the lymph nodes and lymphatic channels. In addition, other malignancies that can cause CA include colon, pancreatic, gastric, kidney, prostate, ovarian, testicular, and breast cancer, as well as intestinal carcinoids, Kaposi's sarcoma, and lymphangiomyomatosis. Nearly two thirds of CA cases in developed countries are associated with malignancy and cirrhosis. Another cause, such as infection, is tuberculosis peritoneum which often occurs in developing countries. Congenital disease is also a risk factor for CA which is often found in the pediatric population, namely primary lymphatic hypoplasia and yellow nail syndrome. The accumulation of chyle (lymph fluid) in the peritoneal cavity can cause serious consequences due to the loss of essential proteins, lipids, immunoglobulin, electrolytes and water which can lead to malnutrition, electrolyte imbalance, and immunosuppression.[10]

Radiation therapy to the abdominal region can lead to lymphatic fibrosis and considered to be the cause of CA. Post-operative CA events can occur after undergoing surgery for the thoracic and abdominal regions that have disorders of the lymphatic system or thoracic tract. The occurrence of CA in cirrhosis is not certainly known but it is presumed to be associated with excessive hepatic and gastrointestinal lymph flow (more than 20 L/day) caused by portal hypertension resulting in spontaneous rupture of the serosal lymph tract.[1]

PATHOPHISIOLOGY

The lymphatic system is a system consisting of vessels, cells, and organs that carry excess interstitial fluid into the bloodstream. This system functions to drain excess interstitial fluid, transport lipids, and produce immune cells. Lymph fluid (lymph) consists of cells, proteins, and chylomicrons that are formed from plasma exudation and are part of the capillaries to the interstitial compartment. The lymphatic system is a route that carries excess fluid from the interstitial space that is not reabsorbed by the post capillary venules and then flows into the vascular system. Lymph fluid will pass through the lymph capillary system to the lymph vessels which have one-way valves, and then to the lymph nodes and lymph trunk.

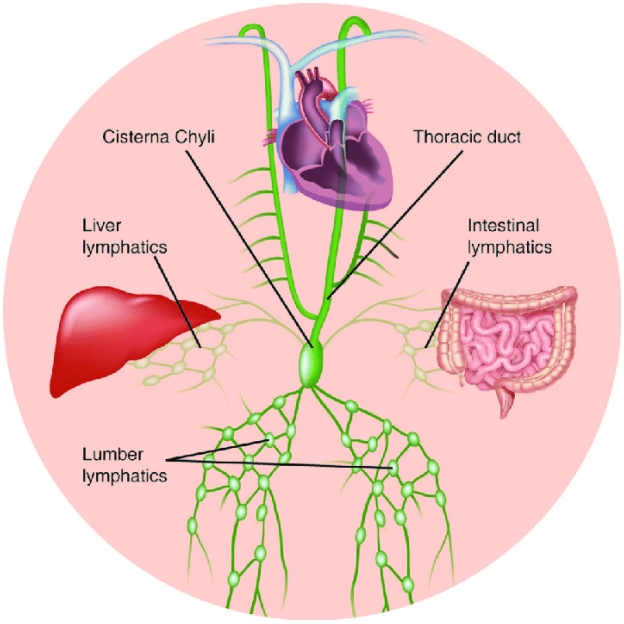

Figure 1. Illustration schemes of lymphatic drainage with main lymphatic tract on thoracoabdominal region[4]

Long chain triglycerides are reduced to monoglycerides and fatty acids in the intestine hence these breakdown products will be absorbed as chylomicrons into the lymphatic system. This explains the high amount of triglycerides and is turbid in color. The abdominal lymphatic system, namely the cisterna chyli or receptaculum chyli, is shaped like a sac that enlarges in the retroperitoneum at the level of the L1-2 lumbar vertebrae and continues as the thoracic duct. From the thoracic duct into the posterior mediastinum after passing through the aortic hiatus then from the aorta and azygous vein at the level of the thoracal vertebra T5 passes to the left into the superior mediastinum and empties into the venous system at the junction of the internal jugular veins and the subclavian veins.[5]

The mechanism for CA formation occurs due to lymphatic system disorders. There are 3 basic mechanisms of CA formation that are known based on lymphangiography and inspection during laparotomy, namely: (1) Exudation of lymph fluid through the retroperitoneal megalymphatic wall into the peritoneal

cavity that occurs with or without a fistula; (2) Lymph fluid leakage from the subserosal lymphatic extends from the intestinal wall to the peritoneal cavity due to the infiltration of carcinoma cells in the lymph nodes which inhibits the flow of lymph from the intestines to the cisterna chili; (3) Direct leakage of lymph fluid through fistulas in the lymph nodes associated with retroperitoneal megalymphs due to lymphatic disorders acquired due to trauma or surgery.[9,10]

In addition, the increase in hepatic and vena cava pressure is due to constrictive pericarditis, right heart failure, dilated cardiomyopathy that precipitates CA due to increased production of lymph fluid in the liver. Cirrhosis causes increased lymph formation in the liver. Portal vein decompression in patients with portal hypertension has been shown to reduce lymphatic hypertension.[10]

Browse et al proposed three pathophysiological mechanisms of CA (Table 1), including acquired lymphatic disorders, lymphatic system fibrosis, and congenital. Acquired lymphatic disorders result from surgery or trauma, causing retroperitoneal lymphatic dilation with lymphatic drainage into the abdomen via lymphoperitoneal fistulas. The dilation of lymph vessels also occurs as a result of increased lymph fluid production and increased hepatic venous pressure seen in patients with cirrhosis and constrictive pericarditis. Heart diseases such as right heart failure and dilated cardiomyopathy cause increased lymphatic pressure from impaired lymphatic drainage, leading to lymph stasis, lymphatic dilation, and chylous ascites. Hepatic venous and vena cava hypertension accelerate the increased production of lymph fluid in the liver. The second mechanism is lymph node fibrosis, often due to malignancy that causes obstruction of lymph flow from the intestine to the cysterna chyli, resulting in leakage of subserosal lymphatic dilatation into the peritoneum. The chronic effect of increasing lymphatic pressure leads to collagen deposition on the lymphatic basement membrane, impairing the absorption capacity of the intestinal mucosa. This process can lead to protein loss enteropathy with malabsorption, chronic steatorrhea, and malnutrition. Congenital causes such as congenital lymphangiectasia causing exudation and leakage of lymph fluid through the fistula into the peritoneal cavity due to the absence of lymphatic valves.[1]

EVALUATION AND DIAGNOSIS

CA diagnostic approach is by suspecting CA diagnosis followed by confirming the presence of lymph fluid in peritoneum cavity and determine the underlying etiology. Assessing the existence of history of disease, physical examination, and performing parasynthesis to make the diagnosis are important in evaluating CA.[10]

Clinical Findings

Progressive abdominal distension, unspecified pain, weight gain, and dyspnea due to distension are the symptoms of CA. Other symptoms include anorexia, malaise, malnutrition, fever, night sweats, and enlarged lymph nodes depending on the underlying etiology. On physical examination found ascites, pleural effusion, lower limb edema, lymphadenopathy, cachexia and other clinical findings consistent with the etiology.[10] The CA diagnostic approach is to suspect a diagnosis then confirm the location of CA in the peritoneal cavity and determine the underlying etiology. CA diagnosis criteria based on clinical symptoms and analysis of ascitic fluid which includes macroscopic appearance, cell count, chemical analysis, cytology, microbiological examination as described in Table 2.[4]

Laboratory

Ascites paracynthetic examination is an important diagnostic support in evaluating CA whatever the cause, either primary or secondary, which is characterized by the color of ascitic fluid to be milky, turbid, which is high in triglycerides with levels > 200 mg/dL.[1]

Table 1. Etiology, mechanism, and causes of chylous ascites[1]

Etiology	Mechanism	Causes
Received	Obstruction or disorder of thoracic duct	Trauma/surgery
		Infection (tuberculosis, filariasis), radiotherapy, autoimun (systemic lupus erythematosus sarchoidosis)
Fibrosis	Invasion and disorders of normal lymph flow	Lymphoma, neuroendocrine tumor, sarcoma, leukemia, malignancy of solid organs
Congenital	Disorders or dilatation of lymph vessel	Lymphangiectasia Waldmann's disease
		Yellow-nail syndrome
		Klippel-Trenaunay syndrome
		Lymphangioma

Figure 2. Chylous ascites fluid samples showing milky fluids[4]

The ascitic fluid was analyzed to assess cell count, gram stain, culture, glucose concentration, total protein, albumin, amylase and lactate dehydrogenase, triglyceride levels, and cytology. The serum ascites albumin gradient (SAAG) assessment was used to identify the cause due to portal hypertension. If the SAAG is less than 1.1 g/dL then portal hypertension as the underlying cause should be excluded and another etiology is sought. In patients with CA due to cirrhosis, SAAG is greater than 1.1 g/dL.[1]

Table 2. Characteristic of ascites fluid on chylous ascites[4]

Characteristic	Value
Color	Milk-like and turbid
Triglycerides level	> 200 mg/dL
Cell amount	> 500 (lymphocyte dominant)
Total amount of protein	2.5–7.0 g/dL
Albumin ascites serum gradient	Depends on the underlying cause
Cholesterol	Low (ascites ratio/serum < 1)
Lactate dehydrogenase	Between 110–200 IU/L
Culture	Likely positive
Amylase	Increased in cases of pancreatitis
Cytology	Positive on malignancy
Glucose	< 100 mg/dL
Adenosine deaminase	Increased in cases of tuberculosis

Total protein assessments range from 2.5 to 7.0 g/dL and will vary according to the underlying etiology. The total protein concentration in ascites in hepatic cirrhosis is less than 2.5 g/dL. Chylomicron identification using lipoprotein electrophoresis is the gold standard for diagnosing CA. However, this testing methodology is difficult and unavailable.

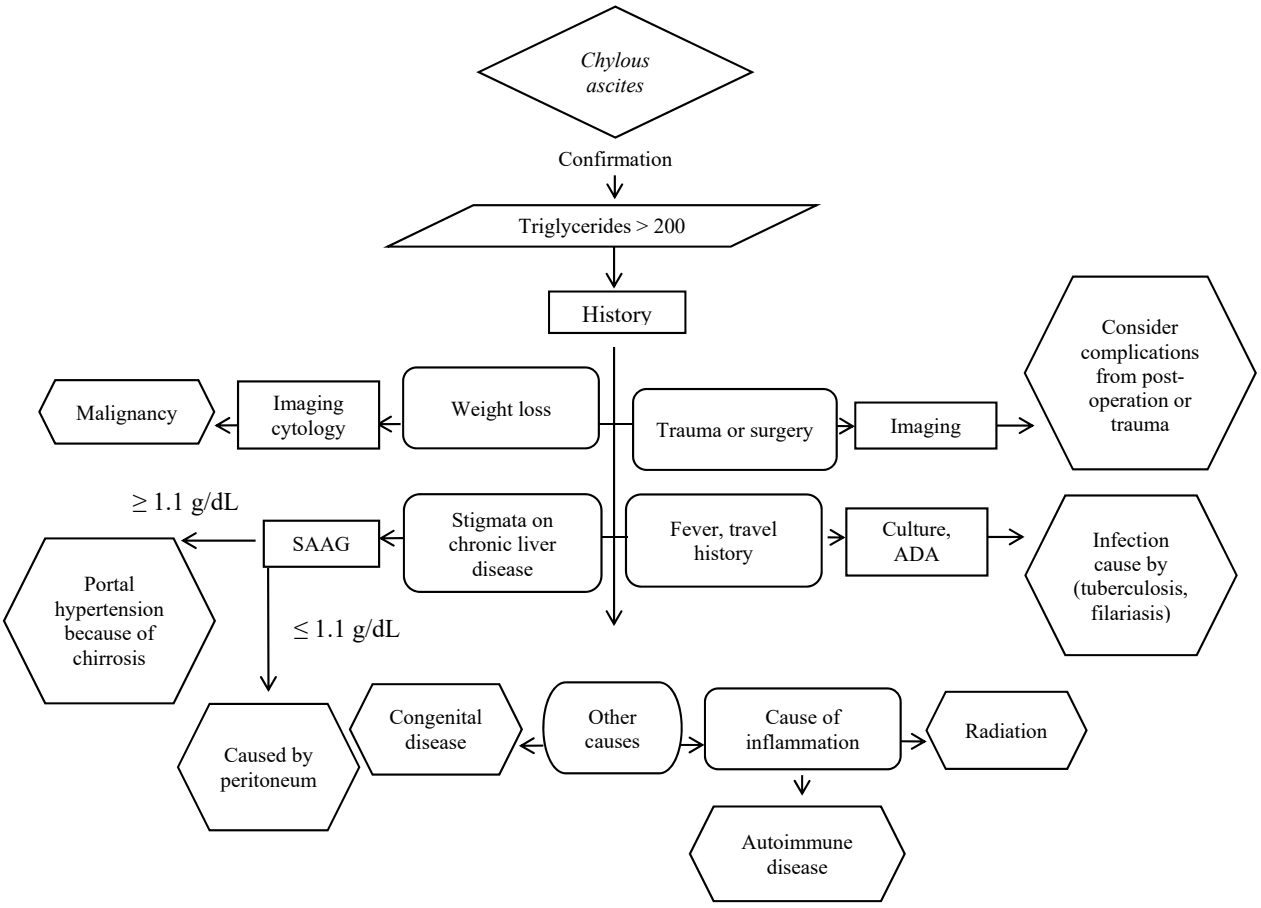

Figure 3. Differential diagnosis of chylous ascites[1]

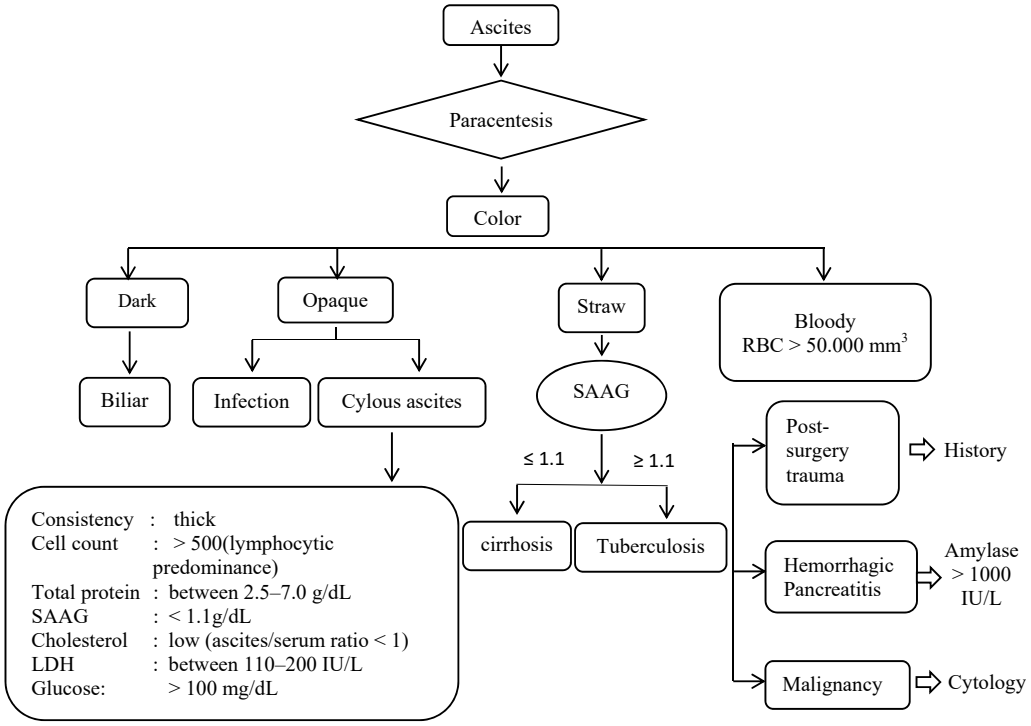

Figure 4. Ascites workup and characteristic of *chylous ascites* fluid[1]

SAAG: serum ascites albumin gradient; RBC: red blood cell count; LDH: lactate dehydrogenase

Acid-resistant bacilli, culture and PCR and adenosine deaminase (ADA) tests are performed if tuberculosis is suspected. The ADA enzyme converts adenosine to inosine and is released by macrophages and lymphocytes during a cellular immune response. The ADA value in peritoneal fluid can be used as an indirect marker for TB effusion. Study by Kawasaki et al has reported a sensitivity of up to 100%, and a specificity of 97% using a cutoff value of 36–40 IU/L. The diagnosis of TB peritonitis can be made by peritoneal biopsy.[10]

Cytologic examination and peritoneal biopsy are used to support the diagnosis of CA. The cytological sensitivity and specificity of ascitic fluid were 62.4% and 98% with a positive predictive value of 100.0% and a negative predictive value of 88.3%. Peritoneal fluid cytology can be supplemented by immunohistochemical studies to improve accuracy.[4] Ascitic fluid cytology of patients with chylous ascites: (1) Thinprep and cytospin preparation showing a medium cellular specimen with large cells and prominent nucleoli surrounded by small, benign lymphocytes, at 60x magnification; (2) Wright-Giemsa stain with 3 neoplastic cells and a very atypical nucleus, at 100x magnification; (3) Imunohistochemical identification of CD20 + B-cells, at 40x magnification; (4) Large neoplastic cells showed negative results for the T-cell marker of CD3, at 40x magnification (Figure 5).

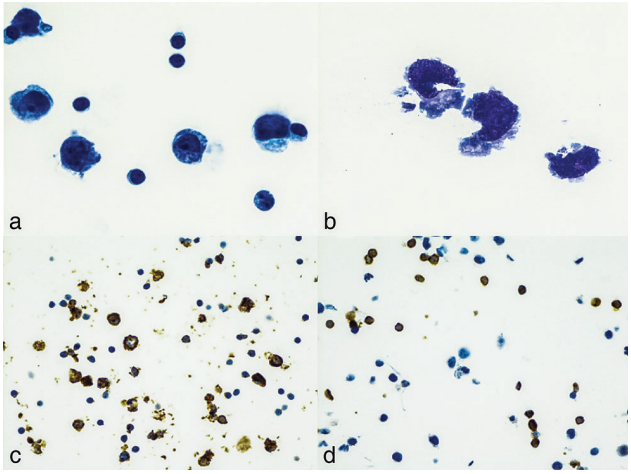

Figure 5. Ascites fluid cytology of patient with chylous ascites[1]

DIAGNOSTIC STUDY

Abdomen CT-Scan

Abdomen CT-scan is a useful modality to identify lymph glands and intraabdominal mass. This examination can also help in finding out the cause of post-surgery or traumatic CA and determining the possible width and location of thoracic duct injury.[10]

Lymphoscintigraphy

A modality that is able to assess the transporting function and detect abnormalities from lymphatic system drainage. This modality is useful in assessing

whether or not an operation is necessary for patients, to assess the success rate of the therapy, as well as usable when lymphangiography is contraindicated. The benefit of this method is the lack of side effects, and no contraindication. However, there are some drawbacks of this modality, namely technical constraints and it is rarely used, thus this modality has not been widely chosen as one for CA examination. Lymphoscintigraphy uses 99 Tc colloid antimony sulfide with human dextran or albumin. Accumulation of fluid can be identified by diffuse absorption. In addition, it can accurately identify the location of lymphatic leaks. The use of single photon emission computed tomography/ computerized tomography (SPECT/CT) technique can determine the anatomy location more specifically by tomographically separating the overlapping source, especially when planning surgery intervention.[14]

Lymphangiography

Lymphangiography is considered to be the golden standard diagnostic modality in cases of lymphatic obstruction. Lymphangiography is mainly used to detect the location of the leak points. In addition, it has the effect of reducing chyle leakage that other imaging modalities cannot. The lymphangiographic mechanism in reducing chyle leakage remains to be further investigated, although some researchers suggest that Lipiodol has an inflammatory and granulomatous reaction during its extravasation. The results of the study obtained 9 out of 14 patients with a high success rate of 100% in the small leakage group and not detected. This is an invasive procedure that has been associated with complications such as infection, pain, and extravasation of contrast during injection. Serious complications that occur include intra alveolar hemorrhage, contrast embolism in the lungs, extravasation of lipiodol into soft tissue and allergic reactions. Studies have reported that this method can detect the location of the leak in patients with chylothorax and CA in about 64–86%.[14] Lymphangiography can easily distinguish minor leaks and undetected leaks from large leaks. In contrast, the success rate was lower (28.6%) in the main leakage group. Prior transition to surgical management or other lymphatic intervention may be practiced when confirmed by lymphangiography. Thus, lymphangiography is a tool used for the postoperative treatment of chylothorax and chylous ascites, and the use of lymphangiography during medical treatment will help avoid surgery.[4] Bipedal lymphangiography is performed by a radiologist. A mixture of 0.5 mL 1% lidocaine and 0.5 ml methylene blue was injected cutaneously and subcutaneously between the first and second knuckles 30 minutes before the procedure. Then the patient is taken to the angiography room or treatment room. Local anesthetic given 1% lidocaine was injected cutaneously and subcutaneously over the lymphatic ducts that identified dye in the dorsalis pedis bilaterally after sterilization with povidone-iodine. The skin was incised longitudinally and cannulated with a 29 g needle. The needles are then tied with 3-0 thread and then glued with an adhesive strip. The same procedure is then performed on the opposite side. Iodinated poppy-seed oil (lipiodol, Japanese guerbet) at room temperature is injected with an automatic injector at an injection pressure of 1.5–2.0 kg/cm^2 per side. Contrast injection is monitored with fluoroscopy only when the procedure is performed in the angiography room. A total of 8 ml of lipidol was injected into both dorsalis pedis in each patient. Then the needle is removed and the wound is sutured together after the injection is complete.[11]

Laparoscopy

Laparoscopy can be used as a diagnostic tool, therapy and determining the stage of malignancy, this method can help to see the peritoneal cavity and take a biopsy sample. The sensitivity and specificity of the laparoscopic technique were 84% and 100% for TB peritonitis, while the peritoneal carcinomatosis reached 100% and 92%.[3]

MANAGEMENT

The management principles of CA include correcting the underlying cause and implementing conservative measures to improve patient comfort, reduce relapse and optimize outcomes.[1] Conservative steps mainly focused in maintaining the optimum balance of nutrients and giving therapy to reduce the lymph flow and production. Other than that, pharmacology therapy and surgery or percutaneous intervention on several cases can be considered as described in Figure 6.[1] Initial management is done by resting the intestine and modifying diet, enteral feeding, or total parenteral nutrition.

Diet Settings

The goals of nutritional therapy are to reduce chyle production, replace fluids and electrolytes, and maintain or improve nutritional status. The recommended diet is a high-protein and low-fat diet with medium-chain triglycerides (MCT), restriction of the long-chain

triglycerides (LCT) diet to prevent conversion to monoglycerides and free fatty acids (FFA) which will then be transported as chylomicrons to the intestinal lymphatic tract. Instead, MCTs are absorbed directly into intestinal cells and then transported as FFA and glycerol to the liver via the portal vein. Thus, a low-fat diet with MCT supplementation can reduce the production and flow of chyle. Coconut oil is a natural source of MCTs. Currently there is a lot of MCT oil but it tastes bad and can cause stomach distension, nausea and vomiting due to its high osmolarity, these factors which lead to poor diet adherence.[4,6]

In patients with cirrhosis, the benefits of administering MCT are still debated as several studies have shown that high serum MCT levels in these patients can cause neurotoxicity and worsen encephalopathy. Cirrhosis patients should be given a low-salt diet and diuretics such as furosemide spironolactone which will help reduce the volume of ascitic fluid. If it does not respond to dietary modifications, bowel rest and total parenteral nutritions are also the available options.[4,6]

Medication

Orlistat is a reversible inhibitor of gastric and pancreatic lipases that plays a role in preventing the conversion of triglycerides from food to free fatty acids in the intestinal lumen, thereby reducing absorption of fatty acids. Orlistat causes minimal systemic absorption, therefore a side effect that can occur is oily stools.[1,4,10]

Somatostatin can reduce portal pressure although the exact mechanism of somatostatin for drying lymphatic fistulas is not understood. Previously, somatostatin has been shown to reduce fat absorption in the intestines, lower triglyceride concentrations in the thoracic tract, and weaken lymph flow in the lymphatic tract. Somatostatin also works by reducing gastric, pancreatic and intestinal secretions, inhibiting intestinal motor activity, slowing down the intestinal absorption process, and reducing splanchnic blood flow which then contributes to decrease lymph production. Somatostatin can also reduce thoracic duct flow. Somatostatin has a very short half-life (1–3 minutes), is given intravenously and has side effects such as diarrhea, hepatotoxicity, dizziness and thrombocytopenia.[4]

Octreotide, is a somatostatin analogue that works by inhibiting the secretion of several pituitary hormones, reducing gastric, pancreatic and intestinal secretions and to reduce splanchnic blood flow so as to reduce lymph flow with a longer half-life (more than 2 hours) and can be administered subcutaneously. Apart from being able to lower portal pressure, octreotide also suppresses the exocrine function of the pancreas thereby reducing absorption of intestinal fat. Side effects of octreotide include reduced sperm motility, nausea, and flatulence. In the study, there was a side effect of octreotide observed in patients namely impaired blood glucose regulation, so close monitoring of blood glucose and gradual reduction of octreotide is recommended. The use of these drugs is recommended in conjunction with the administration of the described diet.[4,12]

Ethylephrine is a sympathomimetic drug used to treat postural hypotension. This drug can be used in the management of CA post esophagectomy with thoracic duct injury in patients with esophageal cancer. This drug works by contracting the smooth muscle of the thoracic duct, which decreases the flow of chyle. Side effects that can occur include headaches, tachycardia and anxiety.[4]

Percutaneous Embolization

CA can be treated by lymphangiography along with adjuvant embolization techniques including percutaneous injection directly into the site of the leak or into nearby lymph nodes. This procedure causes a significant reduction in the amount of drainage, and the consistency of the fluid can change from milky to clear. After this procedure, the drainage amount should be evaluated. At present, there is no general consensus on the cutoff value for post-procedure fluid drainage, but some investigators recommend a cutoff point of < 200–300 mL/d as a marker of successful embolization and an indication that the drainage tube can be removed. A complication that can occur is the migration of glue to the systemic venous system, therefore, the glue must be closely monitored under fluoroscopy when it is injected. The most commonly reported complaint post-procedure is pain in the groin or pelvis, but it is temporary and resolves with conservative management.[4,7]

Paracentesis for Ascites

This action is done to relieve symptoms. Multiple and repeated paracentesis may be a viable option for patients with cirrhosis. This procedure has both a diagnostic and palliative role. Paracentesis alone is not effective and is always combined with other conservative measures. Repeated drainage of ascitic fluid can lead to complications such as prolonged leakage, increased risk of bacterial peritonitis and increased nutritional requirements.[4,10]

Trans-jugular Intrahepatic Portosystemic Shunt (TIPS)

TIPS is an alternative to CA management that has been reported to be successful in patients with cirrhosis and CA resistant to conservative therapy. TIPS creates a path between the portal and the systemic circulation in the liver. By reducing portal pressure, can reduce lymphatic hypertension. When portal hypertension is the underlying pathophysiology, TIPS is a more appropriate procedure to use than others, such as peritoneovenous shunting, which treats the underlying cause of CA. TIPS has an occlusion rate of up to 80% of cases within 1 year of insertion with longer stents often requiring interventions such as stent angioplasty or stenting to restore stent patency.[3]

Peritoneovenous Shunting (LeVeen or Denver shunt)

Peritoneovenous shunting (LeVeen or Denver shunt) may be considered as an option for patients who are refractory to medical therapy and cannot be surgically removed. Shunts function by returning the chyle fluid back to the blood circulation, thereby improving hemodynamic, nutritional, and immunological status. However, this shunt was associated with a high rate of complications such as hypokalemia, sepsis, small bowel obstruction, disseminated intravascular coagulation, and air embolism. In addition, very thick chyle can result in a high rate of shunt occlusion.[4,10]

Surgery

If the conservative management is unsuccessful in treating CA, surgical intervention may be of benefit, especially in cases where CA is secondary to malignancy, postoperative, and congenital. Laparotomy can be performed for fistula closure, bowel resection, or peritoneovenous shunt placement. Preoperative lymphangiography or lymphoscintigraphy is useful in identifying the anatomic location of leaks or fistulas. Once the location of the leak is identified, lymphatic suture ligation can close the leak. If the location of the leak cannot be identified, the retro-aortic tissue can be sutured to stop the lymphatic leak. Tumor resection is a modality in patients with CA secondary to neoplasia.[1,4,10]

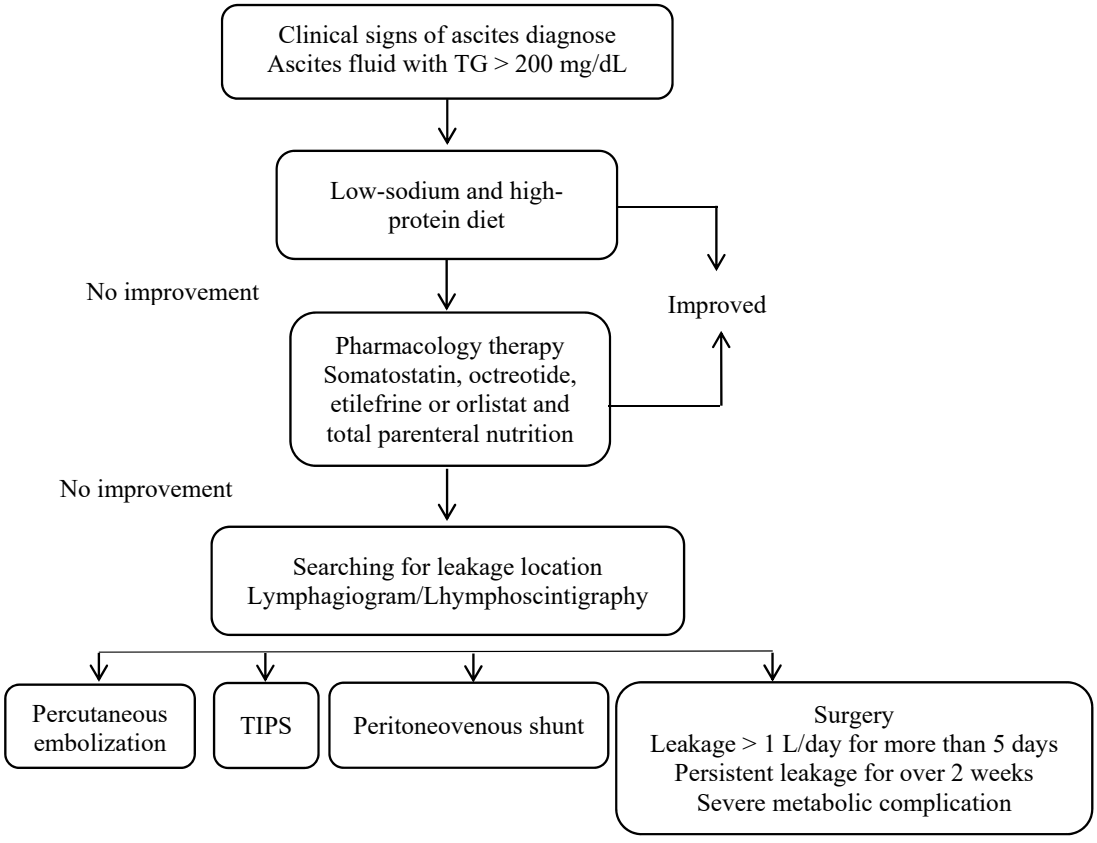

Figure 6. Algorithm of chylous ascites management[1]

CONCLUSION

CA is a pathology entity of rare occurrence. Cirrhosis and malignancy is the most common basic etiology associated with adults. CA characterized by thick milky fluid and increased level of triglycerides fluid. CA management is based on the underlying etiology accompanied by conservative measures, such as diet modification using medium triglycerides chain. Pharmacology therapy such as octreotide, somatostatin, orlistat, and etilephrine can be suitable in certain conditions. In refractory CA cases may require interventions such as embolization, TIPS, peritoneovenous shunting, or surgery. The outcome depends on the underlying etiology and treatment strategy.

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