Chylous, bloody, and pancreatic ascites

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CHYLOUS ASCITES

Chylous ascites is a milky-appearing peritoneal fluid that is rich in triglycerides. It is due to the presence of thoracic or intestinal lymph in the abdominal cavity [1,2].

Chylous ascites is an uncommon finding with a reported incidence of approximately 1 per 20,000 admissions at a large university-based hospital over a two-decade period [3]. Malignancy, cirrhosis, and lymphatic disruption after abdominal surgery are leading causes in adults. In children, congenital lymphatic abnormalities and trauma are the most common cause. Although there have been no recent large epidemiologic studies, it is generally believed that the incidence has increased due to the longer survival of patients with cancer and more aggressive abdominal and cardiothoracic interventions.

Pathophysiology — Chylous ascites develops when there is a disruption of the lymphatic system, which occurs due to traumatic injury or obstruction (from benign or malignant causes). Three underlying mechanisms have been proposed [4]:

- Obstruction of the lymph flow due to malignancy, causing leakage from dilated subserosal lymphatics into the peritoneal cavity. The effects of a continuous elevated pressure of the intestinal lymphatic system may lead to collagen deposition of the basement membrane of lymphatics, further impairing the absorptive capacity of the intestinal mucosa. This can ultimately lead to the development of a protein-losing enteropathy with chronic diarrhea (steatorrhea), malabsorption, and malnutrition. (See "Protein-losing gastroenteropathy".)

- Exudation of lymph through the walls of dilated retroperitoneal vessels lacking valves, which leak fluid through a fistula into the peritoneal cavity (ie, congenital lymphangiectasia).

- Acquired thoracic duct obstruction from trauma or surgery, causing direct leakage of chyle through a lymphoperitoneal fistula. (See "Etiology, clinical presentation, and diagnosis of chylothorax".)

Etiology — There are multiple causes of chylous ascites (table 1). The most common causes in Western countries are abdominal malignancy, lymphatic abnormalities, and cirrhosis, which account for over two-thirds of all cases. By contrast, infectious etiologies (ie, tuberculosis and filariasis) are responsible for the majority of cases in resource-limited countries. Other causes include congenital, inflammatory, postoperative, traumatic, and miscellaneous disorders.

In a systematic review of 131 studies (with a total of 190 patients) who had atraumatic chylous ascites, the most common causes in adults were lymphatic anomalies (32 percent), malignancy (17 percent), cirrhosis (11 percent), *Mycobacterium* infection (15 percent), and a variety of uncommon causes (21 percent) [5]. The high prevalence of lymphatic anomalies and mycobacterial infection reflects the inclusion of studies from developing and developed countries. In children, the most common causes were lymphatic anomalies (84 percent) followed by a variety of uncommon causes (15 percent).

The mechanisms leading to the formation of chylous ascites vary with each condition. As examples:

- Cardiovascular disease, mainly constrictive pericarditis, right-sided heart failure, and dilated cardiomyopathy, can lead to the development of chylous ascites by increasing lymphatic pressure [6,7].

- Increased caval and hepatic venous pressures (vascular obstruction) cause a large increase in the production of hepatic lymph.

- Cirrhosis causes an increased formation of hepatic lymph. Decompression of the portal vein in patients with portal hypertension may relieve lymphatic hypertension [8-11].

Malignancy — Malignancy is a common cause of chylous ascites in adults. Lymphoma accounted for at least one-half to one-third of the cases in one large series of patients identified over 20 years [3]. Obstruction and invasion into the lymphatic channels leads to the disruption of normal lymphatic flow [12]. Other neoplastic causes include breast, esophageal, pancreatic, colon, renal, testicular, endometrial, cervical, ovarian, and prostate cancer; Kaposi sarcoma;

carcinoid tumors; and lymphangiomyomatosis [3,13-15]. Carcinoid tumors should be excluded in patients with chylous ascites and secretory diarrhea. (See "Clinical characteristics of well-differentiated neuroendocrine (carcinoid) tumors arising in the gastrointestinal and genitourinary tracts".)

Cirrhosis — Chylous ascites is present in 0.5 to 1 percent of patients with cirrhosis who have ascites [8,9,16,17]. The underlying pathophysiology is due to the rupture of serosal lymphatic channels, which are dilated due to excessive lymph flow (up to 20 liters/day). Why only a subset of patients with cirrhosis develops it is unclear. It can be a presenting symptom or occur later in the disease as a consequence of hepatocellular carcinoma, following shunt surgery or a thoracic duct injury from sclerotherapy [17-19]. An aggressive approach (unless highly suspected) to exclude malignancy is usually not warranted in a patient with cirrhosis and chylous ascites. Other causes of portal hypertension, such as portal vein thrombosis, have also been implicated as a cause of chylous ascites [20,21].

Infectious — Peritoneal tuberculosis and filariasis are the common infectious causes of chylous ascites.

- Peritoneal tuberculosis occurs worldwide, particularly in areas of low socioeconomic status, malnutrition, and poor access to medical care [22]. (See "Abdominal tuberculosis".)
- Lymphatic filariasis, a disease caused by the parasite *Wuchereria bancrofti*, leads to chylous ascites. This parasite causes a severe inflammatory reaction in the lymphatic vessels, leading to lymphedema and chylous ascites [23]. (See "Lymphatic filariasis: Epidemiology, clinical manifestations, and diagnosis".)
- Infection with *Mycobacterium avium-intracellulare* has been described as a cause of chylous ascites in patients with AIDS [24-26].

Congenital — Congenital lymphatic abnormalities are more common in the pediatric population and should be sought in children with chylous ascites.

- Primary lymphatic hypoplasia is a condition characterized by lymphedema, chylothorax, and/or chylous ascites [27]. Chylous ascites may also be seen with primary lymphatic hyperplasia [28]. Two forms have been described: "bilateral hyperplasia," in which the lymphatics are not severely dilated and contain valves; and lymphangiectasia (megalymphatics), in which the lymphatics are very dilated and lack valves. (See "Clinical features and diagnosis of peripheral lymphedema".)

- The Klippel-Trenaunay syndrome is an autosomal-dominant disorder in which venous and lymphatic hypoplastic malformations cause hypertrophy of the tissues of an involved limb, and it is often associated with chylous ascites [29].

- The yellow nail syndrome is a disorder of unclear etiology seen in childhood. Patients have hypoplastic lymphatics leading to chylous effusions. The characteristic triad consists of lymphedema, pleural effusion and/or chylous ascites, and a yellow discoloration together with dystrophy of the nails [30]. (See "Protein-losing gastroenteropathy".)

Inflammatory — A variety of inflammatory conditions have been associated with chylous ascites, including:

- Radiation therapy to the abdomen, causing fibrosis and obstruction of the lymphatic vessels in the small bowel and mesentery [31].

- Acute or chronic pancreatitis, which can cause the compression of adjacent lymphatic channels, resulting in chylous ascites and pleural effusions [32].

- Constrictive pericarditis (as described above), which can cause chylous ascites by increasing hepatic venous pressure, thereby increasing lymph production [7]. This is a reversible and treatable cause of ascites.

- Other rare inflammatory disorders such as idiopathic retroperitoneal fibrosis or Ormond's disease, sarcoidosis, retractile mesenteritis, and Whipple's disease [33-37].

Postoperative and traumatic — Chylous ascites can occur early (around one week) after abdominal surgery due to the disruption of the lymphatic vessels or late (several weeks to months) due to adhesions or the extrinsic compression of lymphatic vessels [38,39]. The surgical procedures that have been associated with chylous ascites include abdominal aortic aneurysm repair, retroperitoneal lymph node dissection, pancreaticoduodenectomy, inferior vena cava resection, catheter implantation for peritoneal dialysis, laparoscopic Nissen fundoplication, distal splenorenal shunts, laparoscopic donor nephrectomy, small bowel and liver transplantation, and laparoscopic Roux-en-Y gastric bypass [40-54].

Blunt abdominal trauma resulting in intestinal and/or mesenteric injury can also cause chylous ascites [55]. In children, the Battered Child Syndrome, which accounts for approximately 10 percent of cases of chylous ascites in the pediatric population, should be excluded [56].

Other causes

- Right heart failure and dilated cardiomyopathy can cause impaired drainage of lymph, with an increase in lymphatic pressure producing stasis, dilation, and chylous ascites [6]. Chylous ascites can also arise as a result of heart failure secondary to thyrotoxic cardiomyopathy or cardiac amyloidosis [57,58]. Prompt treatment of the heart failure and hyperthyroid state can resolve the ascites [57].

- The nephrotic syndrome and focal segmental glomerulosclerosis have been reported to cause chylous ascites and chylothorax [59-61]. The underlying pathogenesis is not understood. An illustrative report included 140 patients with nephrotic syndrome, of whom 90 had ascites, and of those, 35 underwent paracentesis [62]. Sixteen patients (52 percent) had chylous ascites, defined by the presence of milky white ascitic fluid. A limitation of this study was that triglyceride levels were not measured; thus, some patients may have just had opalescent effusions.

- Lymphangioleiomyomatosis is a rare disorder of unclear etiology characterized by smooth muscle proliferation in the lung, lymphatics, and lymph nodes in the mediastinum, abdomen, and lower cervical region. It affects almost exclusively female patients of childbearing age. The disorder can be associated with a variety of clinical manifestations, including chylous pleural effusions and ascites. (See "Sporadic lymphangioleiomyomatosis: Epidemiology and pathogenesis".)

- Calcium channel blockers have also been implicated as a cause of chylous ascites in patients undergoing peritoneal dialysis [63-67].

Clinical manifestations — Chylous ascites frequently presents as progressive and painless abdominal distention, occurring over the course of weeks to months, depending upon the underlying cause. The most common presenting symptom was abdominal distension (81 percent) in a systematic review that included 131 studies (with a total of 190 patients) who had atraumatic chylous ascites [5]. Patients who have undergone abdominal or thoracic surgery may present with an acute onset.

Patients may complain of weight gain and/or dyspnea resulting from increased abdominal pressure. Other features include nonspecific abdominal pain, weight loss, diarrhea and steatorrhea, malnutrition, edema, nausea, enlarged lymph nodes, early satiety, fevers, and night sweats [3,4,13]. However, in the majority of cases, the diagnosis is not suspected before performing a diagnostic paracentesis.

Evaluation — A careful history and physical examination should be performed as in any patient presenting with ascites. The patient should be questioned regarding weight loss or gain, symptoms of malignancy, family history, recent abdominal surgery, travel, abdominal trauma,

and underlying liver or kidney disease. Constitutional symptoms such as anorexia, weakness, and malaise are very common, but nonspecific. (See "Evaluation of adults with ascites".)

Findings that may be present on physical examination include a fluid wave, shifting dullness, pleural effusions, lower extremity edema, lymphadenopathy, cachexia, temporal wasting, abdominal masses, and hernias. Stigmata of chronic liver disease such as jaundice, palmar erythema, spider angiomata of the chest, and encephalopathy may be present in patients with cirrhosis.

Paracentesis and laboratory data — Abdominal paracentesis is the most important diagnostic tool in evaluating and managing patients with ascites (table 2). Chyle typically has a cloudy and turbid appearance in contrast to the yellow and transparent appearance of ascites due to cirrhosis and portal hypertension. In some patients with cirrhosis, the appearance of ascites may be opalescent due to infection or malignancy without actually containing high levels of triglycerides [68]. (See "Evaluation of adults with ascites".)

The triglyceride levels in ascitic fluid are critical in defining chylous ascites. Triglyceride values are typically above 200 mg/dL, although some authors use a cutoff value of 110 mg/dL [1,3,69,70]. The total protein content varies depending upon the underlying cause, ranging between 2.5 to 7.0 g/dL (table 2) [69]. The serum to ascites albumin gradient should be calculated by subtracting the ascitic fluid value of albumin from the serum value to determine if the ascites is related to portal hypertension or other causes [16,71].

In addition to triglyceride levels, ascitic fluid should be sent for cell count, culture, Gram stain, total protein concentration, albumin, glucose, lactate dehydrogenase, amylase, and cytology [71,72]. A tuberculosis smear, culture, and adenosine deaminase (ADA) should be performed in selected cases when tuberculosis is suspected. ADA is an enzyme involved in the conversion of adenosine to inosine that is released by macrophages and lymphocytes during cellular immune response. ADA values in peritoneal fluid are used as an indirect guide for the diagnosis of tuberculous effusions. Studies outside the United States have reported high sensitivity and specificity in the diagnosis of tuberculous peritonitis in areas of high prevalence for tuberculosis. By contrast, the utility of ADA measurement in populations with a high prevalence of cirrhosis, such as in the United States, is limited [73]. The diagnosis of tuberculous peritonitis usually requires a peritoneal biopsy. (See "Abdominal tuberculosis".)

Standard blood tests including a complete blood count, electrolytes, liver tests, total protein, albumin, lactate dehydrogenase, triglycerides, cholesterol, amylase, and lipase should be performed [71,72]. Additional testing should be based upon the clinical setting.

Radiologic studies — Computed tomography (CT) of the abdomen is useful in identifying pathologic intra-abdominal lymph nodes and masses. In the setting of postoperative or traumatic causes of chylous ascites, it also helps in determining the extent and localization of fluid, particularly if there is a suspicion of thoracic duct injury. Other studies such as non-contrast magnetic resonance (MR) lymphography, lymphangiography and lymphoscintigraphy can assist in detecting abnormal retroperitoneal nodes, leakage from dilated lymphatics, fistulization, and patency of the thoracic duct [20,74]. Lymphangiography (LAG) is the gold standard in defining cases of obstruction, but it may be associated with several complications such as tissue necrosis, fat embolism, and hypersensitivity related to the volume and type of contrast used [74]. In an analysis of 16 patients with 17 chyle leaks who underwent LAG, the leak was identified in all cases. The initial LAG alone provided the diagnosis and localized the chyle leak in four patients, whereas a postprocedure CT scan alone provided the diagnosis and localized the leak in six patients. Both modalities localized the leak in the remaining six patients. No major side effects were reported [75]. In addition to imaging the lymphatics and identifying the site of leakage, lymphangiography may also have therapeutic potential. There are reports of successful embolization of sites of leakage using microcoils or glue in patients with chylothorax or chylous ascites [76,77].

Management — The underlying cause should be addressed whenever feasible. In most cases, correction of the underlying pathology will result in the resolution of symptoms and of the ascites. This is particularly true of patients who have an infectious, inflammatory, or hemodynamic cause.

Only a few studies have addressed specific treatments aimed at reducing the ascites formation [38,78,79]. Based upon the limited data, a reasonable initial approach for patients in whom the cause cannot be found or for those who do not respond to treatment of the underlying condition is to recommend a high-protein and low-fat diet with medium-chain triglycerides (MCT). Dietary restriction of long-chain triglycerides (LCT) avoids their conversion into monoglycerides and free fatty acids (FFA), which are transported as chylomicrons to the intestinal lymph ducts. By contrast, MCTs are absorbed directly into intestinal cells and transported as FFA and glycerol directly to the liver via the portal vein. Thus, a low-fat diet with MCT supplementation reduces the production and flow of chyle [80-82].

MCT is commercially available as MCT oil. The usual initial oral adult dose of MCT oil for use as a nutritional supplement is 1 tablespoon three to four times per day. Common adverse effects are nausea, occasional vomiting, abdominal pain, and diarrhea. MCT oils should be mixed with fruit juices; used on salads and vegetables; incorporated into sauces for use on fish, chicken, or lean meats; or used in cooking or baking. MCT oil should not be used in patients with advanced

cirrhosis because narcosis and coma may occur. Such patients should be managed with a low-sodium diet and diuretics such as spironolactone [72]. (See "Ascites in adults with cirrhosis: Initial therapy".)

Several other measures have been described in case reports or small observational studies.

- Orlistat, a reversible inhibitor of gastric and pancreatic lipases, was reported to minimize ascites and triglyceride levels in ascitic fluid in a patient with chylous ascites due to cirrhosis [83].

- Somatostatin and octreotide have been used successfully to treat chylous effusions in patients with the yellow nail syndrome, chylous pleural effusions, and lymphatic leakage due to abdominal and thoracic surgery [21,84-89]. Case reports have suggested that somatostatin and subcutaneous octreotide as well as midodrine are also effective in the management of chylous ascites [21,86,87,89-92]. The mechanism may involve inhibition of lymph fluid excretion through specific receptors found in the normal intestinal wall of lymphatic vessels [93,94].

Surgery may benefit patients with postoperative, neoplastic, and congenital causes [13]. However, a systematic review of 36 studies showed that for postoperative chylous ascites, conservative management was effective in almost all cases [78]. Prior to surgery, a lymphangiogram or lymphoscintigraphy is helpful in identifying the anatomical location of the leakage or the presence of a fistula.

In patients with a large amount of ascites, a total paracentesis to relieve abdominal discomfort and dyspnea should be performed and repeated as needed. The replacement of intravascular volume with albumin to prevent post-paracentesis circulatory dysfunction is not necessary unless the patient has cirrhosis. Repeated large-volume paracentesis is a reasonable option for patients who have end-stage disease not amenable to medical or surgical treatment.

For some patients, chylous ascites becomes loculated, and fluid removal cannot be achieved with paracentesis. Limited data suggest that intraperitoneal fibrinolytic therapy was associated with improved drainage for patients with loculated chylous ascites. In one report, tissue plasminogen activator was instilled via an intraperitoneal catheter for three days in combination with dornase alfa on days 1 and 3, and this approach was associated with increased output of chylous fluid [95]. Imaging performed five months after treatment showed sustained response.

In patients with cirrhosis and chylous ascites refractory to medical therapy and who have preserved liver function, the insertion of a transjugular intrahepatic portosystemic shunt (TIPS)

can be an effective measure that reduces ascites significantly by decreasing portal pressure [10,11]. A case series of four patients with cirrhosis revealed that TIPS is safe and effective for the treatment of cirrhosis-related chylous collections [96]. The placement of TIPS in a patient with cirrhosis must be carefully evaluated due to the significant problems that may arise after its placement. (See "Overview of transjugular intrahepatic portosystemic shunts (TIPS)".)

In the past, peritovenous shunting was considered an option for patients who were refractory to nonoperative treatment. However, shunts were associated with a high rate of complications, including sepsis, disseminated intravascular coagulation, electrolyte imbalance, small bowel obstruction, and risk for air embolism, and are thus seldom used. In addition, the high viscosity of the chyle results in a high rate of shunt occlusion [97,98]. (See "Hepatorenal syndrome", section on 'Peritoneovenous shunt'.)

BLOODY ASCITES

Bloody ascites is defined as ascitic fluid in the peritoneal cavity with a red blood cell (RBC) count greater than 50,000 mm^3. The RBC count of ascitic fluid is usually less than 1000 mm^3. Ascitic fluid will be pink colored at a level of approximately 10,000 RBCs.

Etiology — There are several causes of bloody ascites. Bloody ascites occurs in up to 19 percent of patients with cirrhosis [99]. In such patients, hemoperitoneum may develop spontaneously or follow a traumatic paracentesis. Most commonly, bloody ascites occurs due to the latter; in this setting blood usually clots, in contrast to non-traumatic bloody ascites in which the blood is lysed and will not clot [100]. The presence of non-traumatic bloody ascites in a patient with cirrhosis is associated with increased morbidity and mortality since such patients frequently have an underlying malignancy such as hepatocellular carcinoma [99,101,102]. Approximately 20 percent of ascitic fluid samples of patients with malignant ascites are bloody [69]. The majority of these cases (50 percent) are accounted for by hepatocellular carcinoma [69]. Of samples from patients with peritoneal carcinomatosis, only approximately 10 percent are bloody. (See "Malignancy-related ascites".)

Classification — Bloody ascites can be classified according to the underlying cause responsible for blood leakage. In most cases, the underlying pathophysiology is related to either mass effect eroding into small vessels or high shear stress over small vessels and lymphatics. These sites can be divided as follows:

- Hepatic parenchyma – Hepatocellular carcinoma [101], cirrhosis with ruptured varices [100,102], idiopathic noncirrhotic portal hypertension [100], lymphoma [103], ruptured

hepatic hemangioma [104], and metastatic liver tumors [100].

- Peritoneal involvement – Leiomyoblastoma [105], tuberculosis, and peritoneal dialysis with sclerosing encapsulating peritonitis [106].
- Adjacent organs – Ovarian cancer [100], endometriosis [107,108], lymphoma [103], prostate cancer [109], pancreatitis [100], splenic lymphoma [110], bladder rupture [111], and ruptured ovarian cyst [112].
- Systemic disease – Sarcoidosis [113], systemic lupus erythematosus, immunoglobulin A vasculitis (Henoch-Schönlein purpura) [114], and parasitic infections (*Fasciola hepatica*) [115].
- Cardiovascular disease – Heart failure [116].
- Trauma in patients with cirrhosis – Trauma to spleen or liver following procedures like liver biopsy, transjugular intrahepatic portosystemic shunt placement, laparoscopy, or paracentesis.

Management — In patients with grossly hemorrhagic ascites, paracentesis should be attempted at a different site to exclude local trauma of a vessel. Abdominal imaging should be performed with an ultrasound or computed tomography scan if bloody non-traumatic ascites is confirmed.

Therapy should be directed toward correction of the underlying cause. In cases of traumatic paracentesis, patients should be observed carefully with strict monitoring of vital signs, complete blood count, and coagulation studies.

Patients with spontaneously bloody ascites due to cirrhosis in the absence of hepatocellular carcinoma usually require no special treatment [102]. Patients with bleeding due to the tap itself usually also have renal failure (based on a study of almost 5000 procedures) and might benefit from desmopressin [117].

Patients whose bloody ascites is due to hepatocellular carcinoma may benefit from embolization of the bleeding tumor vessel. An angiogram and surgical consultation should be sought in patients who continue to show signs of ongoing blood loss (decreasing hemoglobin level, tachycardia, hypotension).

PANCREATIC ASCITES

Pancreatic ascites is defined as the massive accumulation of pancreatic fluid in the peritoneal cavity [118]. It mainly occurs due to an ongoing leakage of pancreatic secretions in the peritoneum due pancreatic duct injury. The level of amylase in the ascitic fluid is typically above 1000 international unit/L, and the ascitic fluid to serum amylase ratio is approximately 6.0 [119]. In one report of eight patients, the ascitic fluid amylase concentration ranged from 280 to 5730 international unit/L, a value more than three times that in the plasma [120].

The most common underlying cause is chronic pancreatitis secondary to alcohol use disorder [121]. It has been described in approximately 4 percent of patients with chronic pancreatitis and in 6 to 14 percent of those with pancreatic pseudocysts [122,123]. It can also occur after an episode of acute pancreatitis or following a traumatic injury to the pancreas [121,124]. It has also been reported to occur after therapeutic endoscopic ultrasonography with fine aspiration of the pancreas [125,126]. However, as many as two-thirds of patients do not give a history of a recent attack of pancreatitis [127].

Clinical manifestations — Patients may present with a history of chronic pancreatitis, a recent episode of acute pancreatitis soon after abdominal trauma, or with new onset ascites. Pain or symptoms of pancreatic disease may be absent, especially in patients with alcohol use disorder; as a result, the diagnosis may be confused with ascites due to cirrhosis and portal hypertension.

A pleural effusion may also be present. In one series, for example, pleural effusion was present (in addition to pancreatic ascites) in 15 of 28 patients (54 percent) [118,121].

Evaluation — As discussed above, diagnostic paracentesis should be performed in every patient with ascites. In some cases, the fluid may be serosanguineous or opalescent. If pancreatic ascites is suspected, routine tests of ascitic fluid such as cell count, culture, Gram stain, amylase, albumin, and total protein should be obtained. The combination of a serum-albumin ascites gradient below 1.1 g/dL, a total protein level >3 g/dL, and elevated ascitic amylase (>1000 units/L) is diagnostic of this condition [124]. In some cases, the white cell count may be elevated due to concomitant infection of the ascitic fluid. (See "Evaluation of adults with ascites".)

Once the diagnosis has been established, an abdominal computed tomography scan should be performed to evaluate for a pseudocyst. Many authorities agree that endoscopic retrograde pancreatography (ERCP) should be performed to localize the site of leakage and to perform endoscopic therapy (stenting of pancreatic duct) if possible [128-131]. Although there are no data on the role of magnetic resonance cholangiopancreatography (MRCP) for the diagnosis of this condition, this test along with secretin stimulation can help demonstrate the normal

pancreatic duct and detect any abnormalities arising from it [132]. Thus, MRCP should be considered in patients who are poor candidates for ERCP.

Management — Conservative medical treatment should be the first step in managing all patients. Withholding oral feedings and starting parenteral nutrition will minimize pancreatic exocrine secretion [133]. One-third of patients will improve on conservative management and will not require any further intervention [134]. Treatment with somatostatin or octreotide together with diuretics and repeated paracentesis may be beneficial for some patients with mild accumulation of pancreatic ascites [135,136].

Transpapillary pancreatic duct stenting can be attempted in patients with persistent pancreatic ascites and evidence of ductal disruption by ERCP [120,128-130]. The stent can facilitate healing of ductal disruptions by partially occluding the leaking duct or bypassing the pancreatic sphincter, thereby decreasing the intrapancreatic duct pressure. (See "Pancreatic stenting at endoscopic retrograde cholangiopancreatography (ERCP): Indications, techniques, and complications".)

Some patients fail medical therapy, ultimately requiring surgery [137]. Indications for surgery include a persistent or recurrent accumulation of ascites and/or a sudden deterioration of clinical status. The type of surgical intervention depends upon the ductal anatomy, the site of the leakage from the pancreatic duct or pseudocyst, and the operative findings. When the pancreatic duct is dilated, the ideal procedure is a wide anastomosis between the ruptured duct and a Roux-en-Y jejunal loop. Patients with a pseudocyst and a mature lining can undergo internal cyst drainage into a jejunal loop. Distal pancreatic resection followed by duct ligation is an acceptable alternative if the pancreatic duct is of normal caliber or the abnormality is localized in the tail of the pancreas [138].

An alternative for patients with pancreatic ascites and evidence of ductal disruption by ERCP is the placement of a transpapillary pancreatic duct stent [120,128-130]. The stent may facilitate healing of ductal disruptions by partially occluding the leaking duct or bypassing the pancreatic sphincter, thereby decreasing the intrapancreatic duct pressure. (See "Pancreatic stenting at endoscopic retrograde cholangiopancreatography (ERCP): Indications, techniques, and complications".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Portal hypertension and

ascites".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Fluid in the belly (ascites) (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Chylous ascites** – Chylous ascites is a milky-appearing peritoneal fluid that is rich in triglycerides. It develops when there is a disruption of the lymphatic system, which occurs due to traumatic injury or obstruction (from benign or malignant causes). (See 'Pathophysiology' above.)

  - There are multiple causes of chylous ascites (table 1). The most common causes in Western countries are abdominal malignancy and cirrhosis, which account for over two-thirds of all cases. The underlying cause should be addressed whenever feasible. (See 'Etiology' above.)

  - Abdominal paracentesis is the most important diagnostic tool in evaluating and managing patients with ascites (table 2). Radiologic evaluation may also be required. (See 'Evaluation' above.)

  - Only a few studies have addressed specific treatments aimed at reducing the ascites formation. In patients in whom the cause cannot be found or for those who do not respond to treatment of the underlying condition, we suggest a high-protein and low-

fat diet with medium-chain triglycerides (MCT) (**Grade 2C**). However, MCT oil should not be used in patients with advanced cirrhosis because narcosis and coma may occur. Patients who do not respond to the above measures often need to be treated with ongoing therapeutic paracentesis. In patients with compensated cirrhosis, a transjugular intrahepatic portosystemic shunt can be considered. (See 'Management' above.)

- **Bloody ascites** – Bloody ascites is defined as ascitic fluid in the peritoneal cavity with a red blood cell count greater than 50,000 mm^3. There are several causes. (See 'Etiology' above.)

  For patients with grossly hemorrhagic ascites, paracentesis should be attempted at a different site to exclude local trauma of a vessel. Abdominal imaging should be performed with an ultrasound or computed tomography scan if bloody nontraumatic ascites is confirmed. Therapy should be directed toward the correction of the underlying cause. (See 'Management' above.)

- **Pancreatic ascites** – Pancreatic ascites is defined as a massive accumulation of pancreatic fluid in the peritoneal cavity. The most common underlying cause is chronic pancreatitis secondary to alcohol use disorder. (See 'Pancreatic ascites' above.)

  - We suggest conservative medical treatment as the first step in managing all patients (**Grade 2C**). Patients should be kept fasted and started on parenteral nutrition. As many as one-third of patients will resolve with this measure alone.

  - For patients who do not respond to conservative management, we suggest treatment with octreotide (50 micrograms given subcutaneously three times daily for two consecutive weeks) and endoscopic retrograde pancreatography for pancreatic duct stenting (**Grade 2C**). For patients who do not respond, we suggest evaluation for surgery.

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Topic 1235 Version 29.0

GRAPHICS

Causes of chylous ascites

Neoplastic (common in adults)
Lymphoma
Other cancers (see text)
Lymphangiomyomatosis
Carcinoid tumors
Kaposi's sarcoma
Cirrhosis (common in adults)
Congenital (common in children)
Primary lymphatic hypoplasia
Yellow nail syndrome
Klippel-Trenaunay syndrome
Primary lymphatic hyperplasia
Bilateral hyperplasia
Intestinal lymphangiectasia
Postoperative
Abdominal aneurysm repair
Retroperitoneal node dissection
Catheter placement for peritoneal dialysis
Inferior vena cava resection
Laparoscopic nissen fundoplication
Laparoscopic nephrectomy
Other causes
Right heart failure
Dilated cardiomyopathy
Nephrotic syndrome
Calcium channel blockers

Infectious
Tuberculosis
Filariasis (Wuchereria bancrofti)
Mycobacterium avium intracellulare
Inflammatory
Radiation
Pancreatitis
Constrictive pericarditits
Retroperitoneal fibrosis
Sarcoidosis
Celiac sprue
Whipple's disease
Retractile mesenteritis
Trauma
Blunt abdominal trauma
Battered child syndrome

Graphic 61854 Version 2.0

Characteristics of ascitic fluid in chylous ascites

Color	Milky and cloudy
Triglyceride level	Above 200 mg/dL
Cell count	Above 500 (predominance of lymphocytes)
Total protein	Between 2.5 - 7.0 g/dL
SAAG	Below 1.1 g/dL*
Cholesterol	Low (ascites/serum ratio <1)
Lactate dehydrogenase	Between 110 - 200 IU/liter
Culture	Positive in selected cases of tuberculosis
Adenosine deaminase	Elevated in cases of tuberculosis
Cytology	Positive in malignancy
Glucose	Under 100 mg/dL
Amylase	Elevated (>40 IU/liter) in cases of acute or chronic pancreatitis

SAAG: serum-ascites albumin gradient; IU: international units.

* Level above 1.1 g/dL in chylous ascites secondary to cirrhosis.

Graphic 56791 Version 2.0

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