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Review

Non-Cirrhotic Ascites: Causes and Management

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Abstract: Ascites is a common syndrome characterized by an excess of fluid in the peritoneum. While cirrhosis is the most common cause, a wide range of other conditions—such as cancer, right heart failure, and tuberculosis—can also lead to ascites, and multiple etiologies may be present simultaneously. Effective diagnosis and management are essential, primarily relying on clinical examination and paracentesis, guided by specific tests.

Keywords: ascites; cirrhosis; paracentesis; serum-ascites albumin gradient

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1. Introduction

Ascites is defined as the accumulation of excess fluid, neither blood nor bile, in the peritoneal cavity. The term comes from "askos," meaning "bag" in ancient Greek. Normally, around 2 mL of fluid is present in the peritoneal cavity. However, physiological alterations can lead to changes, such as increased hydrostatic pressure or decreased oncotic pressure within the vessels, particularly in the portal circulation, often associated with low serum albumin levels. In case of cirrhosis, both mechanisms are implicated—portal hypertension and decreased serum albumin—according to Starling's law [1].

The fluid originates from sinusoidal capillaries in the liver and is typically protein-rich due to the fragility of these capillaries. Albumin is also present, with a passage from 3.8% to 4.7% in ascites quantification [2]. The fluid is usually absorbed by the peritoneal lymphatic system, specifically in the sub-diaphragmatic channels. Under normal circumstances, no more than 600 mL can be absorbed [3].

In portal hypertension, there is an excessive leakage from capillaries, but albumin filtration is lower than that of other proteins. This is particularly important in cirrhosis due to increased production in the fibrotic structures of the Disse space [4]. The lymphatic vascular system can also be impaired, promoting ascites formation [5].

Other mechanisms, not associated with portal hypertension, such as peritoneal invasion by cancer, or local invasion, more rarely, increased vascular permeability. Duct ruptures, though rare, involve the pancreas, bladder, or urinary tract.

If cirrhosis is the most common cause of ascites in Western countries, clinicians must be able to diagnose and differentiate the other causes of ascites. We wanted here to cover the different causes of ascites and help the clinician in their approach.

2. How to Evaluate Ascites

Before any paramedical exploration, a conscientious interrogatory, including past medical history, and a rigorous clinical examination are important. Blood tests are often performed in first intention.

2.1. Ascites' Analysis

Ascitic fluid analysis remains the gold standard for etiological assessment. Specific analyses are necessary and can be tailored based on the clinical presentation. Contraindications to paracentesis are rare but may include intestinal distension and severe coagulopathy.

The appearance of ascitic fluid can provide clues; it is generally clear, but a turbid presentation may suggest infection, a milky appearance is indicative of chylous ascites, and a bloody appearance is more common in cancer or infection.

The serum-ascites albumin gradient (SAAG) is crucial in management [6]. The cutoff is 11 g/L. In cases of portal hypertension, the value exceeds this threshold with over 95% accuracy [7]. While protein concentration in ascitic fluid is less useful for determining etiology today, it can still complement the SAAG for better diagnostic discrimination. The exudative/transudative concept has been replaced by the poor/rich protein concept, which better aligns with pathophysiology. Rivalta's test, which assesses protein concentration, is not commonly used in clinical practice [8].

Cholesterol concentration in ascitic fluid, and potentially the serum-ascites cholesterol gradient, may be more useful. A concentration equal to or greater than 4.5 mg/dL can help distinguish cancer from portal hypertension, including cirrhosis [9]. It may be more effective than protein concentration in differentiating ascites due to portal hypertension from other causes, particularly cancer. Routine cholesterol measurement cannot currently be recommended; even if it may be a helpful tool in malignant ascites, we need more studies to determine its place.

Other markers in ascitic fluid or serum are often less informative and are not usually screened in clinical practice. LDH may be elevated in cancer, with an elevated blood-to-ascites ratio, typically from 0.4 to 0.5, also occurring in infections. The glucose concentration may be reduced in infections due to bacterial or leukocyte consumption [10]. Creatinine testing is rare and mainly relevant for detecting urinary leakage.

Mycology and parasitology tests may be warranted in immunocompromised patients. Screening for tumor markers like AFP or CEA is uncommon and only considered when cancer is strongly suspected but undiagnosed. Elevated triglycerides indicate chylous ascites, with a cutoff at 2 g/L and a milky appearance. Bilirubin concentration is only relevant in cases of gallbladder or bowel perforation. Other analyses are generally unnecessary [11] and are summarized Table 1.

Table 1. Ascites markers. Which markers screen in paracentesis? Adapted from [12].

Important. Useful	**If Necessary, According to Orientation**	**Limited Interest**	**No Indication, No Interest**
Proteins. Albumin. Cells count. Gram coloration. Cytology in first time (repeat in cancer substation).	Amylase in pancreas origin. Cholesterol in cancer suspicion. Triglycerides if chylous. Adenosine desaminase in tuberculosis suspicion. Mycobacteria culture. Ziehl-Neessen. Parasitology. Creatinine in urine tract lack. Bilirubin if bile leak doubt.	Lipase. Lactodehydrogenase in cancer suspicion. ACE. Interferon Gamma Releasing Assay. PCR—RNA 16S.	Sodium. Glucose. Calcium. Other tumoral markers. Viral hepatitis. Thyroid hormones. Urea. Liver enzymes. Heart markers (pro-BNP). Lactates. Phosphatase alkaline. Ferritin. Autoantibodies. Hyaluronate acid.

Cytology is generally a poor tool for cancer detection. Repeating the examination three times increases the accuracy to over 50%. Advanced techniques like immunophenotyping and immunochemistry can improve the diagnostic yield when cancer is suspected [13]. Recent technologies, such as next-generation sequencing targeting circulating tumor DNA, show promise [14,15].

Infection should always be screened for in ascitic fluid, regardless of the underlying cause. In cirrhosis, the definition of infection is primarily based on the white blood cell count in the ascitic fluid. Cultures are always necessary in bacteriology. RNA 16s testing is a possible option, but data are limited [16–18]. Mycobacterial screening, particularly for tuberculosis, is possible, though cultures are often not helpful. Techniques such as PCR [19] and ascitic fluid QuantiFERON can assist in diagnosis. Adenosine deaminase concentration in ascitic fluid, both specific and sensitive, can aid in diagnosing tuberculosis.

Positive cultures with enteric bacteria can guide management and help diagnose infection in all causes of ascites. In other cases, clinical presentation may guide treatment.

2.2. Other Examinations

Additional evaluations depend on the general clinical examination and the differential diagnoses being considered. Cirrhosis should be routinely screened for. Ultrasound can be useful in diagnosing ascites, especially when the quantity of fluid is small or there is doubt, or sometimes before paracentesis. Doppler ultrasound can help assess venous flow. Key measures include mesenteric vein dilation over 11 mm, venous blood velocity, and spleen size, particularly in liver diseases. Elastography is technically challenging in the presence of ascites and is not commonly used in this context. CT scans are often available and allow for detailed exploration of abdominal vessels, the peritoneum, and solid organs. MRI is less helpful. Tomoscintigraphy can be useful, particularly when cancer is suspected, although sensitivity varies depending on the type of cancer. In some cases, surgical exploration may be necessary, with minimally invasive techniques preferred. Histological examination will complement surgery and may be particularly helpful in diagnosing cancer and tuberculosis.

Diagnostic approach is summarized in Supplementary Materials.

3. Epidemiology

In Western countries, liver cirrhosis is the leading cause of ascites, accounting for around 85% of cases [6]. Cancer is the second most common cause at about 7%, while heart failure and kidney diseases account for around 3% of cases. It is estimated that more than 5% of ascites cases are linked to two or more causes [7]. In developing countries, cancer and tuberculosis are the leading causes of non-cirrhotic ascites [6,20].

4. Causes (Table 2)

4.1. Portal Hypertension (Table 3)

4.1.1. Prehepatic

- Portal vein thrombosis.

Ascites is uncommon in portal vein thrombosis and is not typically a major symptom, unlike pain or intestinal distress in mesenteric venous ischemia [21]. When present, ascites is usually minimal. SAAG is generally high [22]. Ultrasound and Doppler are typically sufficient for diagnosis. Management is well established, with anticoagulation commonly used, and TIPS may be necessary for ascites control [23].

4.1.2. Hepatic

Presinusoidal

- Porto-sinusoidal vascular illness.

Porto-sinusoidal vascular disease is a specific entity, recently described, which includes histologic lesions such as peliosis, nodular regenerative hyperplasia, and sinusoidal dilation [24]. It is associated with portal hypertension and often presents with a clinical picture similar to cirrhosis [25]. However, it remains relatively rare. Liver stiffness is generally low, which helps differentiate it from cirrhosis, though this method is not applied in the presence of ascites [26]. CT scan characteristics, such as an enlarged segment IV and a smooth liver surface, along with histology, can often be useful for differentiation [27]. Screening is necessary to identify the underlying cause, which may be hematologic or

immune in nature. Schistosomiasis should also be investigated, particularly in African countries [28]. Treatment is based on managing the underlying cause. The management of ascites is generally the same as in cirrhosis.

Sinusoidal

- Cirrhosis.

Cirrhosis is the primary cause of ascites and typically results in high-SAAG ascites with low protein and cholesterol concentrations [20]. SAAG sensitivity is around 85–90% [29]. Co-occurring conditions like cancer or tuberculosis can also contribute to ascites. In about 20% of cirrhotic patients, ascites may be protein-rich, with levels exceeding 25 g/L [30,31]. The primary causes are listed in the table below and can be intrahepatic, prehepatic, or posthepatic. The underlying cause must always be identified before initiating treatment. In refractory cases, TIPS (transjugular intrahepatic portosystemic shunt) can be considered [32,33]. Cirrhosis may coexist with other causes of ascites.

Post-Sinusoidal

- Sinusoidal obstruction syndrome.

Sinusoidal obstruction syndrome is often associated with bone marrow transplantation in hematologic conditions. Specific criteria have been established, with the Baltimore and EBMT (European Society for Blood and Marrow Transplantation) criteria being the most used. The most commonly used treatment is defibrotide, which targets endothelial cell obstruction [34].

- Budd-Chiari syndrome.

Budd-Chiari syndrome is a rare condition involving thrombosis or compression of the hepatic veins or inferior vena cava and is less common than portal thrombosis. Ascites is present in 83% of patients [35], with a presentation similar to heart-related ascites: high SAAG and high protein concentration, typically above 30 g/L. Diagnosis is based on liver imaging with contrast or ultrasound. Recanalization via anticoagulation and invasive techniques is the cornerstone of treatment. Ascites usually decreases with treatment, but its presence can indicate a poorer prognosis due to a lower response to therapy [36].

4.1.3. Posthepatic

- Heart failure.

Heart failure is a common cause of ascites. Protein concentration is elevated, while the SAAG is typically between 12 and 15 g/dL. The diagnostic accuracy for heart failure-related ascites is 78.3%, with 53.3% sensitivity (95% CI, 28.1–78.6%) and 86.7% specificity (95% CI, 76.7–96.6%) [37,38]. Diagnosis is primarily clinical, but echocardiography can be helpful. Pro-BNP is a discriminant marker: a cutoff of 364 ng/mL aids in diagnosis, with 98% sensitivity, 99% specificity, and 99% diagnostic accuracy. A value below 182 ng/mL excludes the diagnosis [38]. Caution is needed in cases of cirrhosis, as pro-BNP can be elevated, complicating the distinction between the two conditions [39].

Constrictive pericarditis, generally idiopathic or post-surgical, or following acute pericarditis, can lead to elevated venous pressures, hepatic sinusoid congestion, and protein-rich ascites, though it is a rare complication, occurring in less than 10% of cases [40]. Pro-BNP is usually not elevated. Diagnosis is based on echocardiography when in doubt, and possibly other imaging modalities. Treatment primarily involves surgery.

Table 2. Causes of ascites, according to SAAG and proteins concentration.

Proteins	SAAG > 11 g/L	SAAG < 11 g/L
High	Cirrhosis. Heart failure. Pericarditis. Budd-Chiari syndrome.	Cancer. Tuberculosis. Pancreas. Urine leakage.
Low	Cirrhosis. Portal hypertension.	Malnutrition. Nephrotic syndrome.

Table 3. Portal hypertension. Causes [41,42].

Location	Causes
Prehepatic	Portal thrombosis. - Thrombophilia. - Local factors. Portal invasion. - Cancer - Chronic lymphoid leukemia. - Lymphoma. - Epithelioid hemangioendothelioma.
Pre-sinusoidal	Congenital. - Polycysts. - Rendu-Osler illness. - Arterial fistula. Biliary diseases. - Toxics. Vinyl chlorure. - Sclerosing cholangitis. - Auto-immune cholangitis. Granulomatosis. - Schistosoma. - Sarcoidosis. Others. Porto-sinusoidal illness: peliosis, hepatoportal sclerosis, idiopoathic portal hypertension.
Sinusoidal	Cirrhosis. Sinusoidal fibrosis. - Alcoholic hepatitis. - Medications: vinyl chloride. - Metabolic. - Infectious: CMV, HIV, fever Q, syphilis. Sinusoidal collapsus. - Acute inflammation. - Thrombophilia. Sinusoidal infiltration. - Mastocytosis infiltration. - Amylosis. - Gaucher illness. - Liver dysmyelopoiesis. Sinusoidal compression. - Leishmaniosis. - Amylosis. Porto-sinusoidal illness. Peliosis. Congenital disorder. Radiotherapy. Irradiation. Congenital.

Table 3. *Cont.*

Location	Causes
Post-sinusoidal	Veino-occlusive disease. - Liver irradiation. - Toxics. Bone transplantation. Alcaloid pyrrolidinic. - Medications. Hepatic veins sclerosis. - Irradiation. - Vitamin A. Malignant invasion. - Malignant thrombosis. - Angiosrcoma. - Epithelioid hemangioendothelioma. Granulomatosis. - Common immune deficit. - Mycobacteria. - Sarcoidosis. Budd-Chiari syndrome.
Post-hepatic	Cave thrombosis. Right heart failure. Constricitve pericarditis.

4.2. Without Portal Hypertension

4.2.1. Cancer, Malignant Ascites

In cases of ascites, the possibility of an underlying cancer should always be considered, even in patients with cirrhosis [43]. Cancer is the second most common cause of ascites, accounting for about 7% of cases, following liver cirrhosis but significantly more frequent than other causes.

Regarding mechanisms [12], ascites in cancer is generally due to peritoneal invasion by the tumor or increased vascular permeability. Lymphatic obstruction can also lead to chylous effusion. Additionally, some paraneoplastic syndromes can be associated with peritoneal effusion. Liver involvement by metastases can result in portal hypertension. Strictly speaking, malignant ascites differs from peritoneal carcinomatosis [44].

The primary cancers involved include ovarian, breast, colon, stomach, and pancreatic cancers. As gynecologic cancers are predominant, women are more commonly affected [45]. Hepatocellular carcinoma is rarely associated with carcinoma-related ascites formation. Generally, the primary mechanism is the appearance or worsening of portal hypertension. Cancer can also coexist with other causes, such as liver cirrhosis.

Hematologic causes are rare in this context, though lymphoma is possible but uncommon. TAFRO syndrome (thrombocytopenia, ascites, fever, reticulin fibrosis, and organomegaly), a condition related to idiopathic multicentric Castleman disease (iMCD), is exceptionally rare, although anasarca can occur in other forms [46]. Other hematologic disorders, such as extramedullary hematopoiesis, histiocytosis X, leukemia, macrocytosis, and amyloidosis, are rarely associated with ascites. Treatment in these cases is specific and guided by cytologic results.

Primary cancers associated with ascites are rare. Mesothelioma is mainly described but accounts for only 1% of malignant ascites. The incidence is about 1 per 500,000 to 1,000,000 per year. The serous layer is invaded, though ascites is not always present (sensitivity: 70%). Diagnosis is confirmed through cytology obtained during surgical exploration, with specific tumor markers such as cytokeratin 5/6, calretinin, WT-1, podoplanin, and thrombomodulin available [47,48]. Hyaluronate acid screening is possible, but its utility is limited with recent techniques [49].

Pseudomyxoma peritonei, sometimes called “jelly belly” ascites, is also rare, with an incidence of around 1 per million per year, generally affecting women over 40. In rare cases, ascitic tumor markers such as CEA may be elevated [50], though they are not particularly

useful, especially since ascites is often thick and difficult to aspirate. Diagnosis is based on histology obtained via surgery [51].

The SAAG in malignant ascites is low, while protein concentration is generally high, usually above 30 g/L. Around 10% of patients have protein concentrations under 25 g/L [52]. Cholesterol concentration is elevated, above 4.5 mmol/L (specificity: 100%, sensitivity: 50%), with various mechanisms possibly involved, such as cholesterol release, lymphatic obstruction, or peritoneal leak [53]. The association of ascitic ACE and cholesterol may be more useful [54]. LDH concentration might be of interest, but it is less useful than other markers; it is specific and fairly sensitive [55]. Diagnosis can be challenging in some cases due to the low yield of ascitic fluid in paracentesis. Furthermore, malignant-related ascites is not associated with carcinomatosis in one-third of cases, which partly explains the poor accuracy of ascites cytology [56].

Imaging, such as CT scans or 18FDG PET scans, may aid in diagnosis and etiology. Laparotomy may be necessary when the diagnosis remains unclear after these investigations.

Treatment depends on the cause. In some cases, where no specific origin is identified, chemotherapy with agents like Catumaxomab, a trifunctional antibody approved in Europe that targets epithelial cell adhesion molecule (EpCAM), may be considered. Its efficacy has been described mainly in patients with ascites linked to ovarian or gastric cancer, with treatment involving peritoneal infusion coupled with paracentesis [57–59]. This intraperitoneal immunotherapy approach, including various other techniques, such as vaccines, CAR-T cells, dendritic cells, and recombinant human endostatin, is promising [60–62]. Simple paracentesis can be performed regularly [62]. Due to the origin of malignant ascites, which is more often associated with effusion than serum leakage, as seen in cirrhosis, the benefit of albumin supplementation after paracentesis is sometimes debated but is likely low or absent in this context [63]. Older techniques, such as peritoneal shunting, are rare but still available [64]. In cases of recurrent malignant ascites, peritoneal ports or indwelling tunneled catheters can help patients [65,66]. They are contraindicated in cases of coagulopathy or infection, though the infection risk is low [67]. Diuretics are often used, even if the primary mechanism involved is portal hypertension [68].

Infection is rare in cancer-related ascites due to lower permeability to bacteria. The high protein concentration may limit the infection process. Additionally, the definition of infection in this context differs slightly from that in cirrhosis. Bacterial identification is crucial, rather than focusing on leukocyte concentration [69]. Procalcitonin levels in ascitic fluid could be of interest [70]. Albumin supplementation does not appear suitable here due to the different mechanisms involved. It is important to note that distinguishing between peritoneal ascites and infections like tuberculosis or cysticercosis can be challenging.

4.2.2. Infectious Causes

- Tuberculosis

Peritoneal tuberculosis affects 1 to 2% of patients with tuberculosis. It remains relatively rare in countries without widespread tuberculosis, but it is more common in Western and Eastern African countries. The clinical presentation can vary, including symptoms, such as abdominal pain, diarrhea or constipation, weight loss, and fever [71]. Pulmonary tuberculosis is associated with 38% of these patients [72]. The tuberculin skin test (TST) is positive in 50 to 80% of cases, but it is not particularly useful in clinical practice [73].

Imaging, particularly CT scans, can assist in the diagnostic process by revealing features such as smooth peritoneal thickening, lymph node necrosis, or calcifications, though the sensitivity is low [74].

A key diagnostic feature is a lymphocyte concentration above 1000/mm^3 in the ascitic fluid. Specific tests, such as mycobacterial cultures, are not routinely recommended in the initial analysis of ascitic fluid unless clinical suspicion is high. The ascitic fluid LDH/serum ratio is typically above 0.6, and the ascitic fluid protein/serum protein ratio is above 0.5.

Adenosine deaminase (ADA) levels in ascitic fluid can be informative, with good specificity and sensitivity—93% and 95%, respectively—when levels exceed 40 IU/mL [75–77].

In cases of coexisting cirrhosis, ADA levels may be lower [78]. These results, however, are based on older studies and may vary depending on the laboratory techniques used. False positives can occur in cases of lymphoma, hemorrhage, pus, or peritoneal carcinomatosis, and false negatives may occur in early cirrhosis [79]. It is recommended to perform cytological analysis of the ascitic fluid three times to exclude carcinoma cells [80].

Interferon Gamma Release Assays (IGRAs) have been studied in ascitic fluid with limited success and are not generally recommended [81–84]. However, interferon-gamma concentration is a strong marker, with 93% sensitivity and 99% specificity, but it is not routinely advised, especially without comparison to ADA [85]. Peripheral blood T-Spot, an IGRA-based test, shows 91% sensitivity and 78% specificity, with positive and negative likelihood ratios of 4.05 and 0.13, respectively [86]. In ascitic fluid, T-Spot sensitivity and specificity are 90% and 78%, with positive and negative likelihood ratios of 6.35 and 0.14, respectively [86].

Ziehl-Neelsen staining has low sensitivity, under 3%. Culture sensitivity is around 35%, but specificity is high when positive [71]. Newer markers, such as PCR-based diagnostics, are emerging. Xpert MTB/RIF is probably the most advanced; two systematic reviews reported sensitivities between 30% and 60% and nearly 100% specificity for tuberculosis-related ascites [87,88]. Multiplex PCR (using 16SrRNA, IS6110, and devR-based primers) is still under validation but shows promise [19].

In rare cases, diagnostic surgery, typically via laparoscopy, is required due to the low sensitivity of other tests. Laparoscopy reveals yellow-white peritoneal tubercles and peritoneal thickening [89]. Histology showing granulomas with caseous necrosis can further support the diagnosis.

Treatment typically involves a combination of pyrazinamide, ethambutol, isoniazid, and rifampicin [90]. While the traditional treatment duration is 9 months, recent data suggest a 6-month course may be sufficient [90–92]. Liver enzyme monitoring is recommended during treatment. Corticosteroids are not generally included in the standard treatment regimen [80].

- Filariosis

Filariasis is rare in Western countries and is typically found in sub-Saharan Africa or Asia. Ascitic fluid in these cases is milky or chylous, and parasites can be identified in the fluid [93]. Treatment involves antibiotics such as albendazole, ivermectin, or diethylcarbamazine.

- Local infections

Spontaneous infectious causes of ascites are very rare in the absence of liver cirrhosis, though immunosuppression is a risk factor. Antibiotics are usually sufficient to control the ascites in these cases.

Fitz-Hugh-Curtis syndrome can be associated with genital infections. Klebsiella is frequently reported in this context [94]. *Chlamydia trachomatis* is also implicated and should be screened for, especially in young women [95]. In these cases, SAAG is low, and lymphocyte levels are elevated. The source of infection must be identified and treated. Specific antibiotics may be required, and the prognosis is generally good. Surgery may be necessary in some cases.

Colitis can be implied. *Clostridium difficile*, particularly in cases of pseudomembranous colitis, is a concern, though diffuse and severe colon damage leading to ascites is rare [96].

4.2.3. Gynecologic Causes

- Ovarian conditions

Although ovarian cysts are common, they rarely cause ascites. Desmons-Meigs or Meigs syndrome, which is rare, involves pleural and peritoneal effusions along with elevated CA125 levels [97,98]. While the exact mechanisms are not fully understood, localized inflammation and fluid leakage are hypothesized as contributing factors. Management typically involves surgery.

More rarely, a pseudo-Desmons-Meigs syndrome can occur, characterized by the association between an ovarian malignant tumor and tissue effusions [99]. Primary or secondary ovarian cancers are frequently linked to malignant ascites, and the prognosis is generally poorer in cases where ascites is present [100].

Ovarian hyperstimulation syndrome, which can occur following fertility treatments, generally causes mild ascites [101]. Management generally involves surgery, and hormone therapy can be beneficial.

- Endometriosis

Endometriosis is a common benign disorder found in women of reproductive age, characterized by the presence of endometrial tissue outside the uterus [102]. Although the pathophysiology is not well understood, this condition is frequent. However, ascitic complications are rare and are typically hemorrhagic, often associated with pleural effusion [103]. Management generally involves surgery, and hormone therapy can be beneficial.

4.2.4. Serositis

- Lupus

Ascites is a rare manifestation in lupus. The underlying pathophysiological mechanisms of ascites formation can vary:

Serositis is the most common manifestation, involving mild criteria related to various tissue effusions. Serositis affects approximately 12.5% of lupus patients, while ascites (also known as lupus peritonitis) is even rarer and often associated with pleural effusion [104]. Two clinical presentations are typically recognized: an acute form with ascites and abdominal pain that responds to corticosteroids, and a chronic form that is more resistant to corticosteroids, requiring additional immunosuppressive treatments such as azathioprine, cyclophosphamide, or tocilizumab. In the chronic form, IL-6 levels are often elevated [105].

Tjalma Syndrome is a variant of pseudo-pseudo Meigs syndrome, involving ascites, pleural effusion, and elevated CA125 levels [106,107]. It is usually linked to local inflammation caused by peritoneal vasculitis, resulting in a high protein concentration in the ascitic fluid. Although rarely described in the literature, it may be more common than reported due to the frequent elevation of CA125 in ascites [108].

Amyloidosis is rarely involved, typically only in cases of cardiac or renal involvement.

Nephrotic syndrome is another possible cause [109].

Portal vein thrombosis has been described as a cause, though peritoneal perforation is rare.

Diagnosis and specific treatments depend on the underlying mechanisms.

Other serositis causes.

Other collagen vascular diseases are rare causes of ascites [110]. Sarcoidosis can be detected in younger patients [111].

4.2.5. Others

- Pancreas-Related Ascites

Pancreatic ascites is most commonly associated with pancreatic duct rupture, occurring in 17% to 38.5% of acute pancreatitis cases [112,113] and around 3% of chronic pancreatitis. Its presence is a poor prognostic indicator [114]. The origins of pancreatic ascites vary, with inflammation causing ductal obstruction or, in some cases, leakage from a pseudocyst [115]. Diagnosis is typically based on elevated amylase (or less commonly, lipase) levels in the ascitic fluid, with values often exceeding 1000 IU/L [31]. Amylase measurement is preferred due to its easier linearity at high concentrations, which may require dilution of the primary sample. Neutrophil levels in the ascitic fluid can also be elevated.

Management focuses on conservative treatment. Although surgery was traditionally favored [116], transpapillary duct stenting is now considered an excellent option [117]. The choice of treatment should be discussed between surgeons and endoscopists, with endoscopy currently regarded as the gold standard.

- Liver failure

Liver failure is a rare cause of ascites and an uncommon clinical presentation. In fact, around 10% of patients may be affected. This percentage can increase to 60-80% in cases of subacute liver failure [118]. Ascites in this condition is generally protein-poor. The mechanism is generally linked to a serum albumin decrease and portal hypertension appearance. Contrary to liver chronic disease, ascites quantity is lower. Diuretics are efficient.

- Urinary tract

In cases of a urinary tract rupture, there is a risk of developing ascites. Trauma is a potential cause. Bladder rupture, although less common, can also lead to ascites, potentially following surgery or occurring spontaneously in cases of diverticula, radiotherapy, infection, or, more rarely, neoplastic invasion [119]. This condition remains quite exceptional. Diagnosis is based on elevated creatinine levels in the ascitic fluid, typically 40- to 50-times higher than in blood [120]. Serum creatinine levels are also elevated due to reabsorption. Emergency management is often required due to this presentation [121], and surgical repair usually results in good outcomes.

- Malnutrition

Malnutrition as a cause of ascites is rare and is usually associated with chronic malnutrition. The ascites typically presents with low SAAG and low protein concentration due to low serum protein levels. Often, multiple contributing factors exist and may be involved in the ascites process, leading to nutritional deficiencies.

Decreased absorption can occur in cases of enteropathy, which is associated with a wide range of conditions. These include celiac disease, HIV, digestive lymphoma, congenital heart malformations, and Waldmann disease [122]. Extensive digestive investigations are often necessary, and management focuses on nutritional support.

- Radiotherapy

Radiotherapy is a potential cause of ascites [123]. The mechanism involves exudative effusion in the peritoneum and small bowel, with impaired albumin absorption. Chylous ascites can occur due to lymphatic leakage. Treatment depends on the clinical presentation and primarily involves nutritional support. Another possible mechanism is radiation-induced liver disease, though it remains rare [124].

- Thyroid

Ascites as a complication of hyperthyroidism or hypothyroidism is rare, and when it occurs, the ascites is usually mild with a high protein concentration [125]. SAAG levels vary, likely due to various mechanisms, including liver or heart toxicity [126]. TSH is absent in the ascitic fluid, so screening for TSH in ascites is not useful. Serum TSH should be assessed if thyroid dysfunction is suspected.

- Kidney

Nephrotic syndrome is often identified as a primary cause of ascites, frequently accompanied by other effusions, though it remains a rare presentation [127]. The diagnosis is based on significant proteinuria, with levels exceeding 3 g per day or 300 mg/g of creatinine.

Another specific cause is nephrogenic ascites, a distinct entity observed in cases of end-stage kidney disease. The diagnosis is characterized by ascitic fluid with a high protein concentration, low SAAG, and the exclusion of other common causes, such as portal hypertension, heart disease, cancer, or infection [128]. Mortality is high, and kidney transplantation is recommended [129]. In cases of kidney failure with significant water and sodium retention, minimal ascites may develop due to increased hydrostatic pressure, though this is not a primary sign.

Other related causes may arise, particularly in patients undergoing peritoneal dialysis, which carries specific infectious risks.

- Rare causes

Rare pediatric causes of ascites include conditions like yellow nail syndrome [130]. Uncommon infectious diseases, such as amebiasis, Whipple's disease, brucellosis, ascariasis, and salmonellosis, can also cause ascites. Advanced HIV can be associated with ascites [131]. Other rare conditions include Gaucher's disease, Osmond's disease, lymphangioleiomyomatosis, and ascites following ventriculoperitoneal shunting.

4.2.6. Specific Presentations

- Chylous Ascites

Chylous ascites is relatively rare [132], characterized by a milky appearance of the ascitic fluid. Diagnosis is based on triglyceride levels, representing chylomicrons, with a cutoff of 2 g/L (200 mg/dL) or 2.26 mmol/L [132]. However, this cutoff is not universally agreed upon in the literature [133,134]. Increased lymph production may be associated with a high protein concentration [135].

- Various causes and mechanisms must be investigated.

Lymphatic Obstruction: Neoplastic or infectious processes can obstruct lymphatic vessels. Waldenström's disease, with its high viscosity and lymph leakage, is one example [136]. Solid tumors are the most common cause, and lymphoma may also lead to lymphatic invasion, necessitating thorough screening. Congenital origins such as primary lymphatic hypoplasia or hyperplasia, or Klippel-Trenaunay syndrome, may also be observed [137,138]. Inflammation from conditions like radiotherapy or pancreatitis can also lead to chylous ascites [139,140].

Fistula: Trauma, including surgical procedures, especially abdominal or urinary surgery, is the most frequent cause of lymphatic duct rupture.

Increased Mesenteric Lymph Flow: Cirrhosis can lead to increased mesenteric lymph flow, though chylous ascites remains a rare manifestation, occurring in 0.5% to 1% of ascites cases [141].

Heart Failure: Conditions such as constrictive pericarditis can increase lymphatic production while decreasing thoracic duct flow.

The prognosis is often poor. Treatment depends on managing the underlying cause and optimizing nutrition. Surgical intervention may be necessary but is considered a last resort [142]. Low-pressure drainage may be proposed in certain cases [143]. Somatostatin or its derivatives may be used depending on the underlying cause [144].

Nutritional adaptation is often beneficial. Oral or enteral nutrition with a low content of long-chain triglycerides, enriched in medium-chain triglycerides, is a primary therapeutic option [145]. Unlike long-chain triglycerides, which are absorbed via the lymphatics and increase lymph flow, medium-chain triglycerides enter the bloodstream directly [146]. Parenteral nutrition may be necessary in some cases [147].

- Eosinophilic ascites

Eosinophilic ascites is a rare condition associated with an elevated concentration of eosinophils in the ascitic fluid. It is often linked to other systemic diseases. The most common causes include parasitic infections (such as Strongyloides), peritoneal tuberculosis, malignancies (genital, urinary tract, digestive, etc.), eosinophilic gastroenteritis due to serosal infiltration, and hypereosinophilic syndrome [148,149]. Less common causes include sarcoidosis, vasculitis, HIV, Crohn's disease, pheochromocytoma, hypothyroidism, chronic pancreatitis, and peritoneal disease. Due to serosal involvement, protein levels are elevated, and SAAG is low. Standard diagnostic workup is usually supplemented with infectious disease screening.

Treatment depends on the underlying cause. In cases of eosinophilic gastroenteritis and hypereosinophilic syndrome, corticosteroids are often rapidly effective [150]. However, parasitic infections must be ruled out before starting corticosteroids.

5. Conclusions

Ascites can present in various forms depending on the underlying etiology. While cirrhosis is the most common cause in Western countries, many other conditions can lead to ascites. A thorough diagnostic approach is essential to determine the cause, which in turn influences management. Sometimes, the diagnosis of the cause of ascites can be challenging, and exploratory laparotomy is the final step. Various causes must be considered, and treatment should be tailored accordingly.

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Abbreviations

18FDG PET scans	Fluorodeoxyglucose Positron Emission Tomography scans
ADA	Adenosine deaminase
AFP	Alpha-fetoprotein
CA125	Cancer Antigen 125
CAR-T cells	Chimeric Antigen Receptor T cells
CEA	Carcinoembryonic Antigen
CT scan	Computed Tomography scan
DNA	Deoxyribonucleic Acid
EBMT	European Society for Blood and Marrow Transplantation
EpCAM	Epithelial Cell Adhesion Molecule
HIV	Human Immunodeficiency Virus
IGRA	Interferon Gamma Release Assays
iMCD	Idiopathic Multicentric Castleman Disease
LDH	Lactate Dehydrogenase
MRI	Magnetic Resonance Imaging
PCR RNA	Polymerase Chain Reaction for Ribonucleic Acid
Pro-BNP	Pro-Brain Natriuretic Peptide
SAAG	Serum-Ascites Albumin Gradient
TAFRO syndrome	Thrombocytopenia, Ascites, Fever, Reticulin Fibrosis, and Organomegaly syndrome
TIPS	Transjugular Intrahepatic Portosystemic Shunt
T-spot	T-Spot Test
TST	Tuberculosis Skin Test
TSH	Thyroid-Stimulating Hormone
WT-1	Wilms' Tumor 1 protein
Xpert MTB/RIF	Xpert Mycobacterium Tuberculosis/Rifampicin

References

1. Starling, E.H. The Influence of Mechanical Factors on Lymph Production. *J. Physiol.* **1894**, *16*, 224–267. [CrossRef]
2. Schoenberger, J.A.; Kroll, G.; Sakamoto, A.; Kark, R.M. Investigation of the permeability factor in ascites and edema using albumin tagged with I 131. *Gastroenterology* **1952**, *22*, 607–622. [CrossRef]
3. Shear, L.; Ching, S.; Gabuzda, G.J. Compartmentalization of ascites and edema in patients with hepatic cirrhosis. *N. Engl. J. Med.* **1970**, *282*, 1391–1396. [CrossRef]
4. Møller, S.; Henriksen, J.H.; Bendtsen, F. Ascites: Pathogenesis and therapeutic principles. *Scand. J. Gastroenterol.* **2009**, *44*, 902–911. [CrossRef]
5. Jeong, J.; Tanaka, M.; Iwakiri, Y. Hepatic lymphatic vascular system in health and disease. *J. Hepatol.* **2022**, *77*, 206–218. [CrossRef]
6. Runyon, B.A. Care of patients with ascites. *N. Engl. J. Med.* **1994**, *330*, 337–342. [CrossRef]
7. Runyon, B.A.; Montano, A.A.; Akriviadis, E.A.; Antillon, M.R.; Irving, M.A.; McHutchison, J.G. The serum-ascites albumin gradient is superior to the exudate-transudate concept in the differential diagnosis of ascites. *Ann. Intern. Med.* **1992**, *117*, 215–220. [CrossRef]
8. Tincani, E.; Cristani, A.; Cioni, G.; Boldrini, E.; Roccato, S.; Sardini, C.; Ventura, E. Spontaneous bacterial peritonitis in liver cirrhosis. What tests to perform on the ascitic fluid? *Recent. Prog. Med.* **1996**, *87*, 150–153.

9. Du, L.; Zhu, S.; Lu, Z.; Xu, T.; Bai, T.; Xu, D.; Wei, X.; Li, J.; Xu, K.; Ye, J.; et al. Ascitic cholesterol is superior to serum-ascites albumin gradient in the detection of non-portal hypertensive ascites and the diagnosis of mixed ascites. *Aliment. Pharmacol. Ther.* **2019**, *49*, 91–98. [CrossRef]
10. Runyon, B.A.; Hoefs, J.C. Ascitic fluid chemical analysis before, during and after spontaneous bacterial peritonitis. *Hepatology* **1985**, *5*, 257–259. [CrossRef]
11. Tarn, A.C.; Lapworth, R. Biochemical analysis of ascitic (peritoneal) fluid: What should we measure? *Ann. Clin. Biochem.* **2010**, *47 Pt 5*, 397–407. [CrossRef]
12. Runyon, B.A.; Hoefs, J.C.; Morgan, T.R. Ascitic fluid analysis in malignancy-related ascites. *Hepatology* **1988**, *8*, 1104–1109. [CrossRef]
13. Pomjanski, N.; Grote, H.J.; Doganay, P.; Schmiemann, V.; Buckstegge, B.; Böcking, A. Immunocytochemical identification of carcinomas of unknown primary in serous effusions. *Diagn. Cytopathol.* **2005**, *33*, 309–315. [CrossRef]
14. Pomerantz, T.; Brooks, R. Circulating Tumor DNA (ctDNA) and Its Role in Gynecologic Malignancies. *Curr. Treat. Options Oncol.* **2024**, *25*, 510–522. [CrossRef]
15. Engels, M.; Michael, C.; Dobra, K.; Hjerpe, A.; Fassina, A.; Firat, P. Management of cytological material, pre-analytical procedures and bio-banking in effusion cytopathology. *Cytopathology* **2019**, *30*, 31–38. [CrossRef]
16. Rogers, G.B.; van der Gast, C.J.; Bruce, K.D.; Marsh, P.; Collins, J.E.; Sutton, J.; Wright, M. Ascitic microbiota composition is correlated with clinical severity in cirrhosis with portal hypertension. *PLoS ONE* **2013**, *8*, e74884. [CrossRef]
17. Goelz, H.; Wetzel, S.; Mehrbarzin, N.; Utzolino, S.; Häcker, G.; Badr, M.T. Next- and Third-Generation Sequencing Outperforms Culture-Based Methods in the Diagnosis of Ascitic Fluid Bacterial Infections of ICU Patients. *Cells* **2021**, *10*, 3226. [CrossRef]
18. Chen, Y.; Guo, J.; Shi, D.; Fang, D.; Chen, C.; Li, L. Ascitic Bacterial Composition Is Associated with Clinical Outcomes in Cirrhotic Patients with Culture-Negative and Non-neutrocytic Ascites. *Front. Cell. Infect. Microbiol.* **2018**, *8*, 420. [CrossRef]
19. Hallur, V.; Sharma, M.; Sethi, S.; Sharma, K.; Mewara, A.; Dhatwalia, S.; Yadav, R.; Bhasin, D.; Sinha, S.K.; Rana, S.; et al. Development and evaluation of multiplex PCR in rapid diagnosis of abdominal tuberculosis. *Diagn. Microbiol. Infect. Dis.* **2013**, *76*, 51–55. [CrossRef]
20. Runyon, B.A.; AASLD. Introduction to the revised American Association for the Study of Liver Diseases Practice Guideline management of adult patients with ascites due to cirrhosis 2012. *Hepatology* **2013**, *57*, 1651–1653. [CrossRef]
21. HPG-25-S2_Reco-AFEF-MVF-2018.pdf [Internet]. Available online: https://afef.asso.fr/wp-content/uploads/2018/10/HPG-25-S2_Reco-AFEF-MVF-2018.pdf (accessed on 23 January 2022).
22. Ramirez, C.B.; Preeshagul, I.; Sanchez, J.G.; Shrensel, J.A.; Kutner, M.; Favila, K. Portal Vein Thrombosis as a Cause of Massive Ascites in a Non-cirrhotic Patient: 898. *Am. J. Gastroenterol.* **2015**, *110*, S388. [CrossRef]
23. Chinchilla-López, P.; Hamdan-Pérez, N.; Guerrero-Ixtlahuac, J.; Barranco-Fragoso, B.; Méndez-Sánchez, N. The Role of TIPS in patients with Refractory Ascites and Portal Vein Thrombosis. *Ann. Hepatol.* **2017**, *16*, 619–620. [CrossRef]
24. Plessier, A.; Rautou, P.E.; Valla, D.C. Management of hepatic vascular diseases. *J. Hepatol.* **2012**, *56* (Suppl. S1), S25–S38. [CrossRef]
25. De Gottardi, A.; Rautou, P.E.; Schouten, J.; Rubbia-Brandt, L.; Leebeek, F.; Trebicka, J.; Garcia-Pagan, J.C. Porto-sinusoidal vascular disease: Proposal and description of a novel entity. *Lancet Gastroenterol. Hepatol.* **2019**, *4*, 399–411. [CrossRef]
26. Elkrief, L.; Lazareth, M.; Chevret, S.; Paradis, V.; Magaz, M.; Blaise, L.; Rubbia-Brandt, L.; Moga, L.; Durand, F.; Payancé, A.; et al. Liver Stiffness by Transient Elastography to Detect Porto-Sinusoidal Vascular Liver Disease with Portal Hypertension. *Hepatology* **2021**, *74*, 364–378. [CrossRef]
27. Valainathan, S.R.; Sartoris, R.; Elkrief, L.; Magaz, M.; Betancourt, F.; Pellegrino, S.; Nivolli, A.; Burgio, M.D.; Flattet, Y.; Terraz, S.; et al. Contrast-enhanced CT and liver surface nodularity for the diagnosis of porto-sinusoidal vascular disorder: A case-control study. *Hepatology* **2022**, *76*, 418–428. [CrossRef]
28. Sarin, S.K.; Khanna, R. Non-cirrhotic portal hypertension. *Clin. Liver. Dis.* **2014**, *18*, 451–476. [CrossRef]
29. Prabhu, M.; Gangula, R.S.; Stanley, W. Diagnostic Utility of Serum Ascites Lipid and Protein Gradients in Differentiation of Ascites. *Int. J. Hepatol.* **2019**, *2019*, 8546010. [CrossRef]
30. Gupta, R.; Misra, S.P.; Dwivedi, M.; Misra, V.; Kumar, S.; Gupta, S.C. Diagnosing ascites: Value of ascitic fluid total protein, albumin, cholesterol, their ratios, serum-ascites albumin and cholesterol gradient. *J. Gastroenterol. Hepatol.* **1995**, *10*, 295–299. [CrossRef]
31. Runyon, B.A. Amylase levels in ascitic fluid. *J. Clin. Gastroenterol.* **1987**, *9*, 172–174. [CrossRef]
32. Carrier, P.; Loustaud-Ratti, V.; Debette-Gratien, M.; Elkrief, L. Ascites in cirrhotic patients: A comprehensive review. *Explor. Dig. Dis.* **2024**, *3*, 362–381. [CrossRef]
33. Queck, A.; Schwierz, L.; Gu, W.; Ferstl, P.G.; Jansen, C.; Uschner, F.E.; Praktiknjo, M.; Chang, J.; Brol, M.J.; Schepis, F.; et al. Targeted decrease of portal hepatic pressure gradient improves ascites control after TIPS. *Hepatology* **2023**, *77*, 466–475. [CrossRef]
34. Lee, T.B.; Yang, K.; Ko, H.J.; Shim, J.R.; Choi, B.H.; Lee, J.H.; Ryu, J.H. Successful defibrotide treatment of a patient with veno-occlusive disease after living-donor liver transplantation: A case report. *Medicine* **2021**, *100*, e26463. [CrossRef]
35. Coilly, A.; Potier, P.; Broué, P.; Kounis, I.; Valla, D.; Hillaire, S.; Lambert, V.; Dutheil, D.; Hernández-Gea, V.; Plessier, A.; et al. Budd-Chiari syndrome. *Clin. Res. Hepatol. Gastroenterol.* **2020**, *44*, 420–425. [CrossRef]
36. Agrawal, D.; Gupta, D.; Nathani, R.; Dhore, P.; Meshram, M.; Bhatia, S.J.; Shukla, A. Presence of Ascites at Presentation is Associated with Absence of Long-Term Response Amongst Patients with Budd-Chiari Syndrome When Treated with Medical Therapy Alone: A Single Centre Real-Life Experience. *J. Clin. Exp. Hepatol.* **2021**, *12*, 861–870. [CrossRef]

37. Patel, Y.A.; Muir, A.J. Evaluation of New-Onset Ascites. *JAMA* **2016**, *316*, 340–341. [CrossRef]
38. Farias, A.Q.; Silvestre, O.M.; Garcia-Tsao, G.; Seguro, L.F.d.C.; Mazo, D.F.d.C.; Bacal, F.; Andrade, J.L.; Gonçalves, L.L.; Strunz, C.; Ramos, D.S.; et al. Serum B-type natriuretic peptide in the initial workup of patients with new onset ascites: A diagnostic accuracy study. *Hepatology* **2014**, *59*, 1043–1051. [CrossRef]
39. Ge, P.S.; Runyon, B.A. Role of plasma BNP in patients with ascites: Advantages and pitfalls. *Hepatology* **2014**, *59*, 751–753. [CrossRef]
40. Barbina, S.; Kavin, U.; Sutton, M.M.; Heincelman, M.; Thomas, M.K. Ascitic Fluid Analysis Leading to the Diagnosis of Constrictive Pericarditis in 2 Patients. *J. Investig. Med. High Impact Case Rep.* **2022**, *10*, 23247096221097530. [CrossRef]
41. FMC-HGE [Internet]. Ascite Non Cirrhotique. Available online: https://www.fmcgastro.org/textes-postus/no-postu_year/ascite-non-cirrhotique/ (accessed on 18 June 2023).
42. Kotani, K.; Kawada, N. Recent Advances in the Pathogenesis and Clinical Evaluation of Portal Hypertension in Chronic Liver Disease. *Gut Liver* **2024**, *18*, 27–39. [CrossRef]
43. Sangisetty, S.L.; Miner, T.J. Malignant ascites: A review of prognostic factors, pathophysiology and therapeutic measures. *World J. Gastrointest. Surg.* **2012**, *4*, 87–95. [CrossRef]
44. Smith, E.; Jayson, G. The current and future management of malignant ascites. *Clin. Oncol.* **2003**, *15*, 59–72. [CrossRef]
45. Ayantunde, A.; Parsons, S. Pattern and prognostic factors in patients with malignant ascites: A retrospective study. *Ann. Oncol.* **2007**, *18*, 945–949. [CrossRef]
46. Kanuganti, D.; Nagarjunakonda, V.S.; Bandarupalli, P.; Gorijala, V.K.; Konagalla, V.L.S.R.; Kowtha, P. POEMS Syndrome: A Case Report and Review of the Literature. *Cureus* **2022**, *14*, e27001. [CrossRef]
47. Kusamura, S.; Kepenekian, V.; Villeneuve, L.; Lurvink, R.J.; Govaerts, K.; De Hingh, I.H.J.T.; Moran, B.J.; Van der Speeten, K.; Deraco, M.; Glehen, O.; et al. Peritoneal mesothelioma: PSOGI/EURACAN clinical practice guidelines for diagnosis, treatment and follow-up. *Eur. J. Surg. Oncol.* **2021**, *47*, 36–59. [CrossRef]
48. Sugarbaker, P.H.; Acherman, Y.I.; Gonzalez-Moreno, S.; Ortega-Perez, G.; Stuart, O.; Marchettini, P.; Yoo, D. Diagnosis and treatment of peritoneal mesothelioma: The Washington Cancer Institute experience. *Semin. Oncol.* **2002**, *29*, 51–61. [CrossRef]
49. Dvoskin, S. Mesothelioma of the peritoneum; a case report in which the ascitic fluid contained hyaluronic acid. *Ann. Intern. Med.* **1954**, *40*, 809–811.
50. Wang, B.; Ma, R.; Rao, B.; Xu, H. Serum and ascites tumor markers in the diagnostic and prognostic prediction for appendiceal pseudomyxoma peritonei. *BMC Cancer* **2023**, *23*, 90. [CrossRef]
51. Govaerts, K.; Lurvink, R.; De Hingh, I.; Van der Speeten, K.; Villeneuve, L.; Kusamura, S.; Kepenekian, V.; Deraco, M.; Glehen, O.; Moran, B.; et al. Appendiceal tumours and pseudomyxoma peritonei: Literature review with PSOGI/EURACAN clinical practice guidelines for diagnosis and treatment. *Eur. J. Surg. Oncol. (EJSO)* **2021**, *47*, 11–35. [CrossRef]
52. Zhu, S.; Du, L.; Xu, D.; Lu, Z.; Xu, T.; Li, J.; Song, Y. Ascitic fluid total protein, a useful marker in non-portal hypertensive ascites. *J. Gastroenterol. Hepatol.* **2020**, *35*, 271–277. [CrossRef]
53. Castaldo, G.; Oriani, G.; Cimino, L.; Topa, M.; Mostarda, I.; Castellano, L.; Del Vecchio-Blanco, C.; Budillon, G.; Salvatore, F.; Sacchetti, L. Total discrimination of peritoneal malignant ascites from cirrhosis- and hepatocarcinoma-associated ascites by assays of ascitic cholesterol and lactate dehydrogenase. *Clin. Chem.* **1994**, *40*, 478–483. [CrossRef]
54. Gulyas, M.; Kaposi, A.D.; Elek, G.; Szollar, L.G.; Hjerpe, A. Value of carcinoembryonic antigen (CEA) and cholesterol assays of ascitic fluid in cases of inconclusive cytology. *J. Clin. Pathol.* **2001**, *54*, 831–835. [CrossRef]
55. Boyer, T.D.; Kahn, A.M.; Reynolds, T.B. Diagnostic value of ascitic fluid lactic dehydrogenase, protein, and WBC levels. *Arch. Intern. Med.* **1978**, *138*, 1103–1105. [CrossRef]
56. Runyon, B.A. Malignancy-related ascites and ascitic fluid « humoral tests of malignancy ». *J. Clin. Gastroenterol.* **1994**, *18*, 94–98. [CrossRef]
57. Heiss, M.M.; Murawa, P.; Koralewski, P.; Kutarska, E.; Kolesnik, O.O.; Ivanchenko, V.V.; Dudnichenko, A.S.; Aleknaviciene, B.; Razbadauskas, A.; Gore, M.; et al. The trifunctional antibody catumaxomab for the treatment of malignant ascites due to epithelial cancer: Results of a prospective randomized phase II/III trial. *Int. J. Cancer* **2010**, *127*, 2209–2221. [CrossRef]
58. Schmalfeldt, B.; Pfisterer, J.; Lichinitser, M.; Makhson, A.; Lindhofer, H.; Kimmig, R. Effective relief of malignant ascites in patients with advanced ovarian cancer by a trifunctional anti-EpCAM x anti-CD3 antibody: A phase I/II study. *Clin. Cancer Res.* **2007**, *13*, 3899–3905.
59. Wimberger, P.; Gilet, H.; Gonschior, A.-K.; Heiss, M.M.; Moehler, M.; Oskay-Oezcelik, G.; Al-Batran, S.-E.; Schmalfeldt, B.; Schmittel, A.; Schulze, E.; et al. Deterioration in quality of life (QoL) in patients with malignant ascites: Results from a phase II/III study comparing paracentesis plus catumaxomab with paracentesis alone. *Ann. Oncol.* **2012**, *23*, 1979–1985. [CrossRef]
60. Zhan, Z.; Wang, X.; Yu, J.; Zheng, J.; Zeng, Y.; Sun, M.; Peng, L.; Guo, Z.; Chen, B. Intraperitoneal infusion of recombinant human endostatin improves prognosis in gastric cancer ascites. *Futur. Oncol.* **2022**, *18*, 1259–1271. [CrossRef]
61. Thadi, A.; Khalili, M.; Morano, W.F.; Richard, S.D.; Katz, S.C.; Bowne, W.B. Early Investigations and Recent Advances in Intraperitoneal Immunotherapy for Peritoneal Metastasis. *Vaccines* **2019**, *6*, 54. [CrossRef]
62. Matsusaki, K.; Aridome, K.; Emoto, S.; Kajiyama, H.; Takagaki, N.; Takahashi, T.; Tsubamoto, H.; Nagao, S.; Watanabe, A.; Shimada, H.; et al. Clinical practice guideline for the treatment of malignant ascites: Section summary in Clinical Practice Guideline for peritoneal dissemination (2021). *Int. J. Clin. Oncol.* **2022**, *27*, 1–6. [CrossRef]

63. Stephenson, J.; Gilbert, J. The development of clinical guidelines on paracentesis for ascites related to malignancy. *Palliat. Med.* **2002**, *16*, 213–218. [CrossRef]
64. Sugawara, S.; Sone, M.; Arai, Y.; Sakamoto, N.; Aramaki, T.; Sato, Y.; Inaba, Y.; Takeuchi, Y.; Ueno, T.; Matsueda, K.; et al. Radiological insertion of Denver peritoneovenous shunts for malignant refractory ascites: A retrospective multicenter study (JIVROSG-0809). *Cardiovasc. Interv. Radiol.* **2011**, *34*, 980–988. [CrossRef]
65. Rosenberg, S.; Courtney, A.; Nemcek, A.A.; Omary, R.A. Comparison of percutaneous management techniques for recurrent malignant ascites. *J. Vasc. Interv. Radiol.* **2004**, *15*, 1129–1131. [CrossRef]
66. Lungren, M.P.; Kim, C.Y.; Stewart, J.K.; Smith, T.P.; Miller, M.J. Tunneled peritoneal drainage catheter placement for refractory ascites: Single-center experience in 188 patients. *J. Vasc. Interv. Radiol.* **2013**, *24*, 1303–1308. [CrossRef]
67. Coupe, N.A.; Cox, K.; Clark, K.; Boyer, M.; Stockler, M. Outcomes of permanent peritoneal ports for the management of recurrent malignant ascites. *J. Palliat. Med.* **2013**, *16*, 938–940. [CrossRef]
68. Becker, G.; Galandi, D.; Blum, H.E. Malignant ascites: Systematic review and guideline for treatment. *Eur. J. Cancer* **2006**, *42*, 589–597. [CrossRef]
69. Moon, H.; Kim, H.J.; Woo, S.M.; Park, I.H.; Sim, S.H. Discrimination of ascitic fluid infection in malignant ascites by polymorphonuclear neutrophil ratio and count: Investigating the validity of count $\geq 250/mm^3$ as the one-size-fits-all criterion. *Asia Pac. J. Clin. Oncol.* **2021**, *17*, e87–e93. [CrossRef]
70. Cekin, Y.; Cekin, A.H.; Duman, A.; Yilmaz, U.; Yesil, B.; Yolcular, B.O. The Role of Serum Procalcitonin Levels in Predicting Ascitic Fluid Infection in Hospitalized Cirrhotic and Non-cirrhotic Patients. *Int. J. Med. Sci.* **2013**, *10*, 1367–1374. [CrossRef]
71. Sanai, F.M.; Bzeizi, K.I. Systematic review: Tuberculous peritonitis--presenting features, diagnostic strategies and treatment. *Aliment. Pharmacol. Ther.* **2005**, *22*, 685–700. [CrossRef]
72. Kim, N.J.; Choo, E.J.; Kwak, Y.G.; Lee, S.O.; Choi, S.H.; Woo, J.H.; Kim, Y.S. Tuberculous peritonitis in cirrhotic patients: Comparison of spontaneous bacterial peritonitis caused by Escherichia coli with tuberculous peritonitis. *Scand. J. Infect. Dis.* **2009**, *41*, 852–856. [CrossRef]
73. Chow, K.M.; Chow, V.C.Y.; Hung, L.C.T.; Wong, S.M.; Szeto, C.C. Tuberculous peritonitis-associated mortality is high among patients waiting for the results of mycobacterial cultures of ascitic fluid samples. *Clin. Infect. Dis.* **2002**, *35*, 409–413. [CrossRef]
74. Chen, J.; Liu, S.; Tang, Y.; Zhang, X.; Cao, M.; Xiao, Z.; Ren, M.; Chen, T. Diagnostic performance of CT for differentiating peritoneal tuberculosis from peritoneal carcinomatosis: A systematic review and meta-analysis. *Clin. Radiol.* **2020**, *75*, 396.e7–396.e14. [CrossRef]
75. Sevinc, A.; Sari, R.; Fadillioglu, E. The utility of lactate dehydrogenase isoenzyme pattern in the diagnostic evaluation of malignant and nonmalignant ascites. *J. Natl. Med. Assoc.* **2005**, *97*, 79–84.
76. Liao, Y.-J.; Wu, C.-Y.; Lee, S.-W.; Lee, C.-L.; Yang, S.-S.; Chang, C.-S.; Lee, T.-Y. Adenosine deaminase activity in tuberculous peritonitis among patients with underlying liver cirrhosis. *World J. Gastroenterol.* **2012**, *18*, 5260–5265.
77. Suceveanu, A.I.; Todescu, D.; Mazilu, L.; Manousos, F.G.; Hulea, R.; Voinea, F.; Suceveanu, A.P. Modern Tools for Diagnosis in Tuberculous Ascites [Internet]. Ascites—Physiopathology, Treatment, Complications and Prognosis. IntechOpen. 2017. Available online: https://www.intechopen.com/chapters/56637 (accessed on 13 June 2022).
78. Hillebrand, D.J.; Runyon, B.A.; Yasmineh, W.G.; Rynders, G.P. Ascitic fluid adenosine deaminase insensitivity in detecting tuberculous peritonitis in the United States. *Hepatology* **1996**, *24*, 1408–1412. [CrossRef]
79. Dahale, A.S.; Puri, A.S.; Sachdeva, S.; Agarwal, A.K.; Kumar, A.; Dalal, A.; Saxena, P.D. Reappraisal of the Role of Ascitic Fluid Adenosine Deaminase for the Diagnosis of Peritoneal Tuberculosis in Cirrhosis. *Korean J. Gastroenterol.* **2021**, *78*, 168–176. [CrossRef]
80. Jha, D.K.; Pathiyil, M.M.; Sharma, V. Evidence-based approach to diagnosis and management of abdominal tuberculosis. *Indian J. Gastroenterol.* **2023**, *42*, 17–31. [CrossRef]
81. Greco, S.; Girardi, E.; Masciangelo, R.; Capoccetta, G.B.; Saltini, C. Adenosine deaminase and interferon gamma measurements for the diagnosis of tuberculous pleurisy: A meta-analysis. *Int. J. Tuberc. Lung. Dis.* **2003**, *7*, 777–786.
82. Le Vavasseur, B.; Gerin, M.; Similowsky, T.; Cadranel, J.; Tourret, J.; Ronco, P.; Carcelain, G. Intérêt des IGRAs (Quantiferon TBG-IT®, Tspot-TB®) dans les liquides biologiques pour le diagnostic de tuberculose extrapulmonaire. *Rev. Médecine Interne* **2015**, *36*, A77–A78. [CrossRef]
83. Lorenz, R.; Würl, P.; Haerter, G.; Cammerer, G.; Barth, T.; Hausladen, S.; Stenger, S.; Kern, P. Interferon-gamma release assay in the ascites: Early hint for diagnosis of abdominal tuberculosis. *Infection* **2009**, *38*, 69–72. [CrossRef]
84. Sharma, S.K.; Tahir, M.; Mohan, A.; Smith-Rohrberg, D.; Mishra, H.K.; Pandey, R.M. Diagnostic accuracy of ascitic fluid IFN-gamma and adenosine deaminase assays in the diagnosis of tuberculous ascites. *J. Interferon. Cytokine Res.* **2006**, *26*, 484–488. [CrossRef]
85. Su, S.-B.; Qin, S.-Y.; Guo, X.-Y.; Luo, W.; Jiang, H.-X. Assessment by meta-analysis of interferon-gamma for the diagnosis of tuberculous peritonitis. *World J. Gastroenterol.* **2013**, *19*, 1645–1651. [CrossRef]
86. Luo, Y.; Xue, Y.; Mao, L.; Lin, Q.; Tang, G.; Song, H.; Sun, Z. Diagnostic Value of T-SPOT.TB Assay for Tuberculous Peritonitis: A Meta-Analysis. *Front. Med.* **2020**, *7*, 585180. [CrossRef]
87. Sharma, V.; Soni, H.; Kumar, M.P.; Dawra, S.; Mishra, S.; Mandavdhare, H.S.; Singh, H.; Dutta, U. Diagnostic accuracy of the Xpert MTB/RIF assay for abdominal tuberculosis: A systematic review and meta-analysis. *Expert Rev. Anti-Infect. Ther.* **2021**, *19*, 253–265. [CrossRef]

88. Kohli, M.; Schiller, I.; Dendukuri, N.; Yao, M.; Dheda, K.; Denkinger, C.M.; Schumacher, S.G.; Steingart, K.R. Xpert MTB/RIF Ultra and Xpert MTB/RIF assays for extrapulmonary tuberculosis and rifampicin resistance in adults. *Cochrane Database Syst. Rev.* **2021**, *2021*, CD012768.
89. Bhargava, D.K.; Chopra, P.; Nijhawan, S.; Dasarathy, S.; Kushwaha, A.K. Peritoneal tuberculosis: Laparoscopic patterns and its diagnostic accuracy. *Am. J. Gastroenterol.* **1992**, *87*, 109–112.
90. Jullien, S.; Jain, S.; Ryan, H.; Ahuja, V. Six-month therapy for abdominal tuberculosis. *Cochrane Database Syst. Rev.* **2016**, *11*, CD012163.
91. Mandavdhare, H.S.; Singh, H.; Dutta, U.; Sharma, V. A real-world experience with 6 months of antitubercular therapy in abdominal tuberculosis. *JGH Open* **2019**, *3*, 201–205. [CrossRef]
92. Makharia, G.K.; Ghoshal, U.C.; Ramakrishna, B.S.; Agnihotri, A.; Ahuja, V.; Chowdhury, S.D.; Gupta, S.D.; Mechenro, J.; Mishra, A.; Pathak, M.K.; et al. Intermittent Directly Observed Therapy for Abdominal Tuberculosis: A Multicenter Randomized Controlled Trial Comparing 6 Months Versus 9 Months of Therapy. *Clin. Infect. Dis.* **2015**, *61*, 750–757. [CrossRef]
93. Patel, K.C. Filariasis, chyluria and chylous effusion. *J. Assoc. Physicians. India.* **1983**, *31*, 801–803.
94. Haight, J.B.; Ockner, S.A. Chlamydia trachomatis perihepatitis with ascites. *Am. J. Gastroenterol.* **1988**, *83*, 323–325.
95. Halford, B.; Piazza, M.B.; Liu, D.; Obineme, C. Chlamydia ascites: A call for sexually transmitted infection testing. *BMJ Case Rep.* **2018**, *11*, e226437. [CrossRef]
96. Salehi, H.; Salehi, A.M. A Rare Case of Pseudomembranous Colitis Presenting with Pleural Effusion and Ascites with Literature Review. *Case Rep. Gastrointest. Med.* **2021**, *2021*, 6019068. [CrossRef]
97. Meigs, J.V. Fibroma of the ovary with ascites and hydrothorax; Meigs' syndrome. *Am. J. Obstet. Gynecol.* **1954**, *67*, 962–987. [CrossRef]
98. Demons: Epanchements Pleurétiques Compliquant les...—Google Scholar [Internet]. Available online: https://scholar.google.com/scholar_lookup?title=Les+%C3%A9panchement+pleur%C3%A9tique+compliquant+les+kystes+de+l%E2%80%99ovaire&publication_year=1887&journal=Bull+M%C3%A9m+Soc+Chir+Paris&pages=771-776 (accessed on 27 March 2023).
99. Dalal, N.; Athwal, P.S.S.; Tharu, B.; Saravanan, L.; Mansour, H. A Rare Case of Pseudo-Meigs' Syndrome with Ovarian Metastasis Presenting as Meigs' Syndrome. *Cureus* **2020**, *12*, e11022. [CrossRef]
100. Rickard, B.P.; Conrad, C.; Sorrin, A.J.; Ruhi, M.K.; Reader, J.C.; Huang, S.A.; Franco, W.; Scarcelli, G.; Polacheck, W.J.; Roque, D.M.; et al. Malignant Ascites in Ovarian Cancer: Cellular, Acellular, and Biophysical Determinants of Molecular Characteristics and Therapy Response. *Cancers* **2021**, *13*, 4318. [CrossRef]
101. Timmons, D.; Montrief, T.; Koyfman, A.; Long, B. Ovarian hyperstimulation syndrome: A review for emergency clinicians. *Am. J. Emerg. Med.* **2019**, *37*, 1577–1584. [CrossRef]
102. Giudice, L.C.; Kao, L.C. Endometriosis. *Lancet* **2004**, *364*, 1789–1799. [CrossRef]
103. Pandraklakis, A.; Prodromidou, A.; Haidopoulos, D.; Paspala, A.; Oikonomou, M.D.; Machairiotis, N.; Rodolakis, A.; Thomakos, N. Clinicopathological Characteristics and Outcomes of Patients with Endometriosis-Related Hemorrhagic Ascites: An Updated Systematic Review of the Literature. *Cureus* **2022**, *14*, e26222. [CrossRef]
104. Man, B.L.; Mok, C.C. Serositis related to systemic lupus erythematosus: Prevalence and outcome. *Lupus* **2005**, *14*, 822–826. [CrossRef]
105. Watanabe, R.; Fujii, H.; Kamogawa, Y.; Nakamura, K.; Shirai, T.; Ishii, T.; Harigae, H. Chronic lupus peritonitis is characterized by the ascites with a large content of interleukin-6. *Tohoku J. Exp. Med.* **2015**, *235*, 289–294. [CrossRef]
106. Schmitt, R.; Weichert, W.; Schneider, W.; Luft, F.C.; Kettritz, R. Pseudo-pseudo Meigs' syndrome. *Lancet* **2005**, *366*, 1672. [CrossRef]
107. Tjalma, W.A. Ascites, pleural effusion, and CA 125 elevation in an SLE patient, either a Tjalma syndrome or, due to the migrated Filshie clips, a pseudo-Meigs syndrome. *Gynecol. Oncol.* **2005**, *97*, 288–291. [CrossRef]
108. Martins, A.; Pimenta, S.; Rato, M.; Oliveira, D.; Martins, F.R.; Pinheiro, F.O.; Fonseca, D.; Costa, L. Pseudo-pseudo Meigs syndrome: An uncommon onset of Systemic Lupus Erythematosus. *ARP Rheumatol.* **2022**.
109. Lee, S.; Jung, S.; Kim, M.-J.; Lee, J.S.; Jang, H.N.; Chang, S.-H.; Kim, H.-J. Clinical and histopathological analyses of kidney biopsies in a single center for 7 years. *Medicine* **2022**, *101*, e29695. [CrossRef]
110. Carrier, P.; Jacques, J.; Debette-Gratien, M.; Legros, R.; Sarabi, M.; Vidal, E.; Sautereau, D.; Bezanahary, H.; Ly, K.H.; Loustaud-Ratti, V. Non-cirrhotic ascites: Pathophysiology, diagnosis and etiology. *Rev. Med. Interne* **2014**, *35*, 365–371. [CrossRef]
111. Lee, S.W.; Lee, M.H.; Lee, J.E.; Choi, S.Y.; Yi, B.H.; Jung, J.M. Peritoneal sarcoidosis: A case report. *Medicine* **2019**, *98*, e16001. [CrossRef]
112. Maringhini, A.; Ciambra, M.; Patti, R.; Randazzo, M.A.; Dardanoni, G.; Mancuso, L.; Termini, A.; Pagliaro, L. Ascites, pleural, and pericardial effusions in acute pancreatitis. A prospective study of incidence, natural history, and prognostic role. *Dig. Dis. Sci.* **1996**, *41*, 848–852. [CrossRef]
113. Samanta, J.; Rana, A.; Dhaka, N.; Agarwala, R.; Gupta, P.; Sinha, S.K.; Gupta, V.; Yadav, T.D.; Kochhar, R. Ascites in acute pancreatitis: Not a silent bystander. *Pancreatology* **2019**, *19*, 646–652. [CrossRef]
114. Zeng, Q.-X.; Wu, Z.-H.; Huang, D.-L.; Huang, Y.-S.; Zhong, H.-J. Association Between Ascites and Clinical Findings in Patients with Acute Pancreatitis: A Retrospective Study. *Med. Sci. Monit.* **2021**, *27*, e933196-1–e933196-7. [CrossRef]
115. Sileo, A.V.; Chawla, S.K.; LoPresti, P.A. Pancreatic ascites: Diagnostic importance of ascitic lipase. *Dig. Dis. Sci.* **1975**, *20*, 1110–1114. [CrossRef]

116. Da Cunha, J.E.; Machado, M.; Bacchella, T.; Penteado, S.; Mott, C.B.; Jukemura, J.; Pinotti, H.W. Surgical treatment of pancreatic ascites and pancreatic pleural effusions. *Hepatogastroenterology* **1995**, *42*, 748–751.
117. Pai, C.G.; Suvarna, D.; Bhat, G. Endoscopic treatment as first-line therapy for pancreatic ascites and pleural effusion. *J. Gastroenterol. Hepatol.* **2009**, *24*, 1198–1202. [CrossRef]
118. Ellis, A.J.; Saleh, M.; Smith, H.; Portmann, B.; Gimson, A.; Williams, R. Late-onset hepatic failure: Clinical features, serology and outcome following transplantation. *J. Hepatol.* **1995**, *23*, 363–372. [CrossRef]
119. Newman, M.; Nityanandan, G.; Smyth, N. Pseudo-Renal Failure in the Context of Traumatic Bladder Rupture. *Cureus* **2022**, *14*, e32360. [CrossRef]
120. Wang, J.-H.; Kung, Y.-H.; King, T.-M.; Chang, M.-C.; Hsu, C.-W. Measurement of peritoneal fluid urea nitrogen and creatinine levels is useful to detect iatrogenic urinary tract leakage in colorectal surgery. *J. Chin. Med. Assoc.* **2015**, *78*, 283–286. [CrossRef]
121. Martens, P. Pseudorenal failure secondary to reversed intraperitoneal autodialysis. *Case Rep. Nephrol.* **2013**, *2013*, 982391. [CrossRef]
122. Vignes, S.; Bellanger, J. Primary intestinal lymphangiectasia (Waldmann's disease). *Orphanet J. Rare Dis.* **2008**, *3*, 5. [CrossRef]
123. Shelat, V.G.; Pandya, G.J.; Shabbir, A.; Diddapur, R.K. Post radiation chylous ascites: A case report. *Cases J.* **2009**, *2*, 9393. [CrossRef]
124. Munoz-Schuffenegger, P.; Ng, S.; Dawson, L.A. Radiation-Induced Liver Toxicity. *Semin. Radiat. Oncol.* **2017**, *27*, 350–357. [CrossRef]
125. Ebert, E.C. The thyroid and the gut. *J. Clin. Gastroenterol.* **2010**, *44*, 402–406. [CrossRef]
126. Ji, J.-S.; Chae, H.-S.; Cho, Y.-S.; Kim, H.-K.; Kim, S.-S.; Kim, C.-W.; Lee, C.-D.; Lee, B.-I.; Choi, H.; Lee, K.-M.; et al. Myxedema ascites: Case report and literature review. *J. Korean Med. Sci.* **2006**, *21*, 761–764. [CrossRef]
127. Ackerman, Z. Ascites in Nephrotic syndrome. Incidence, patients' characteristics, and complications. *J. Clin. Gastroenterol.* **1996**, *22*, 31–34. [CrossRef]
128. Han, S.H.; Reynolds, T.B.; Fong, T.L. Nephrogenic ascites. Analysis of 16 cases and review of the literature. *Medicine* **1998**, *77*, 233–245. [CrossRef]
129. Abdalla, M.S.; Saad, E.; Abdalla, M.; Musa, T.I.; Mohamed, K. Nephrogenic Ascites: An Unusual Culprit of Refractory Ascites in a Hemodialysis Patient—A Case Report and Review of Literature. *Cureus* **2022**, *14*, e30876. [CrossRef]
130. Kournoutas, I.; Tung, E.E. Yellow Nail Syndrome. *N. Engl. J. Med.* **2024**, *391*, 68. [CrossRef]
131. Khanna, R.; Sarin, S.K. Non-cirrhotic portal hypertension—Diagnosis and management. *J. Hepatol.* **2014**, *60*, 421–441. [CrossRef]
132. Lizaola, B.; Bonder, A.; Trivedi, H.D.; Tapper, E.B.; Cardenas, A. Review article: The diagnostic approach and current management of chylous ascites. *Aliment. Pharmacol. Ther.* **2017**, *46*, 816–824. [CrossRef]
133. Staats, B.; Ellefson, R.; Budahn, L.; Dines, D.; Prakash, U.; Offord, K. The lipoprotein profile of chylous and nonchylous pleural effusions. *Mayo Clin. Proc.* **1980**, *55*, 700–704.
134. Thaler, M.A.; Bietenbeck, A.; Schulz, C.; Luppa, P.B. Establishment of triglyceride cut-off values to detect chylous ascites and pleural effusions. *Clin. Biochem.* **2017**, *50*, 134–138. [CrossRef]
135. Cárdenas, A.; Chopra, S. Chylous ascites. *Am. J. Gastroenterol.* **2002**, *97*, 1896–1900. [CrossRef]
136. Stoffel, E.M.; Spigel, D.R.; Grace, N.D. Waldenstrom's macroglobulinemia presenting as new-onset ascites. *J. Hepatol.* **2004**, *41*, 689–690. [CrossRef]
137. Cohen, M.M. Klippel-Trenaunay syndrome. *Am. J. Med. Genet.* **2000**, *93*, 171–175. [CrossRef]
138. Unger, S.W.; Chandler, J.G. Chylous ascites in infants and children. *Surgery* **1983**, *93*, 455–461.
139. Hurst, P.A.; Edwards, J.M. Chylous ascites and obstructive lymphoedema of the small bowel following abdominal radiotherapy. *Br. J. Surg.* **1979**, *66*, 780–781. [CrossRef]
140. Goldfarb, J.P. Chylous effusions secondary to pancreatitis: Case report and review of the literature. *Am. J. Gastroenterol.* **1984**, *79*, 133–135.
141. Cheng, W.S.C.; Gough, I.R.; Ward, M.; Croese, J.; Powell, L.W. Chylous ascites in cirrhosis: A case report and review of the literature. *J. Gastroenterol. Hepatol.* **1989**, *4*, 95–99. [CrossRef]
142. Ha, G.W.; Lee, M.R. Surgical repair of intractable chylous ascites following laparoscopic anterior resection. *World J. Gastroenterol.* **2015**, *21*, 6077–6081. [CrossRef]
143. Shibuya, Y.; Asano, K.; Hayasaka, A.; Shima, T.; Akagi, K.; Ozawa, N.; Wada, Y. A novel therapeutic strategy for chylous ascites after gynecological cancer surgery: A continuous low-pressure drainage system. *Arch. Gynecol. Obstet.* **2013**, *287*, 1005–1008. [CrossRef]
144. Kong, P.-F.; Xu, Y.-H.; Lai, Z.-H.; Ma, M.-Z.; Duan, Y.-T.; Sun, B.; Xu, D.-Z. Novel management indications for conservative treatment of chylous ascites after gastric cancer surgery. *World J. Gastroenterol.* **2022**, *28*, 6056–6067. [CrossRef]
145. Sriram, K.; Meguid, R.A.; Meguid, M.M. Nutritional support in adults with chyle leaks. *Nutrition* **2016**, *32*, 281–286. [CrossRef]
146. Tran, T.T.T.; Buttet, M. Mécanisme d'absorption intestinale des acides gras à longue chaîne: Rôle émergent du CD36. *Cah. De Nutr. Et De Diététique* **2012**, *47*, 272–279. [CrossRef]
147. Baran, M.; Cakir, M.; Yüksekkaya, H.A.; Arikan, C.; Aydin, U.; Aydogdu, S.; Kilic, M. Chylous ascites after living related liver transplantation treated with somatostatin analog and parenteral nutrition. *Transplant. Proc.* **2008**, *40*, 320–321. [CrossRef]
148. Pinte, L.; Baicuş, C. Causes of eosinophilic ascites—A systematic review. *Romanian J. Intern. Med.* **2019**, *57*, 110–124. [CrossRef]

149. Khalil, H.; Joseph, M. Eosinophilic ascites: A diagnostic challenge. *BMJ Case Rep.* **2016**, *2016*, bcr2016216791. [CrossRef]
150. Galere, P.; Gilbert, A. Eosinophilic gastroenteritis with ascites: An enigmatic diagnosis. *Lancet* **2022**, *400*, 126. [CrossRef]

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