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Case Report

Cirrhosis-related ascites with right heart failure and pulmonary tuberculosis

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ABSTRACT

Ascites is defined as an excess of fluid within the peritoneal cavity. Causes of ascites may be classified into two pathophysiologic categories: associated with the normal peritoneum and occur due to a diseased peritoneum. Ascites is clinically apparent when the patient presents either symptomatically with abdominal distension, thus leading to further testing, or asymptomatically when radiological imaging shows unexpected peritoneal fluid accumulation. The serum ascites-albumin gradient (SAAG) and total protein levels from ascitic fluid are useful to distinguish the etiology of ascites. We report a case of a 30-years-old man with shortness of breath, the stomach has been getting bigger, nausea, and cough which was subsequently diagnosed with cirrhosis-related ascites accompanied with right heart failure and pulmonary tuberculosis.

Keywords: Ascites, Cirrhosis, Serum ascites-albumin gradient

INTRODUCTION

Ascites is a pathophysiological condition with increased fluid in the intraperitoneal space.[1,2] Normally, a few millilitres of serous fluid (approximately 50-75 ml) is present between the visceral and parietal membrane of the peritoneal space. Under pathological conditions this amount of fluid can increase to several litres, often with different composition compared to interstitial space fluid. Ascites is clinically apparent when the patient presents either symptomatically with abdominal distension, thus leading to further testing, or asymptomatically when radiological imaging shows unexpected peritoneal fluid accumulation.[3,4]

Ascites can develop for many reasons including heart or kidney disease but is seen most commonly in patients with liver cirrhosis. In the United States, the 3 main causes of ascites are cirrhosis (85%), peritoneal malignancy (7%), and heart failure (3%), with causes such as nephrotic syndrome and tuberculosis.[5] Causes of ascites may be classified into two pathophysiologic categories: associated with the normal peritoneum and occur due to a diseased peritoneum.[1,2] The introduction of the concept of the serum-ascites albumin gradient (SAAG) has also resulted in better characterization of ascites than the traditional exudate or transudate concept. SAAG is useful for distinguishing ascites caused by portal hypertension from non-portal hypertensive ascites. The SAAG is calculated by subtracting the ascitic albumin concentration from the serum albumin level and does not change with diuresis. SAAG >1.1 g/dl reflects the presence of portal hypertension and indicates the ascites is due to increased pressure in the hepatic sinusoids, then SAAG <1.1 g/dl indicates that the ascites is not related to portal hypertension, as in tuberculous peritonitis, peritoneal carcinomatosis, or pancreatic ascites (97.0% sensitivity and 90.2% specificity).[2,3,5] Here we presented a case report of a 30-years-old man diagnosed with ascites due to cirrhosis accompanied by right heart failure and pulmonary tuberculosis.

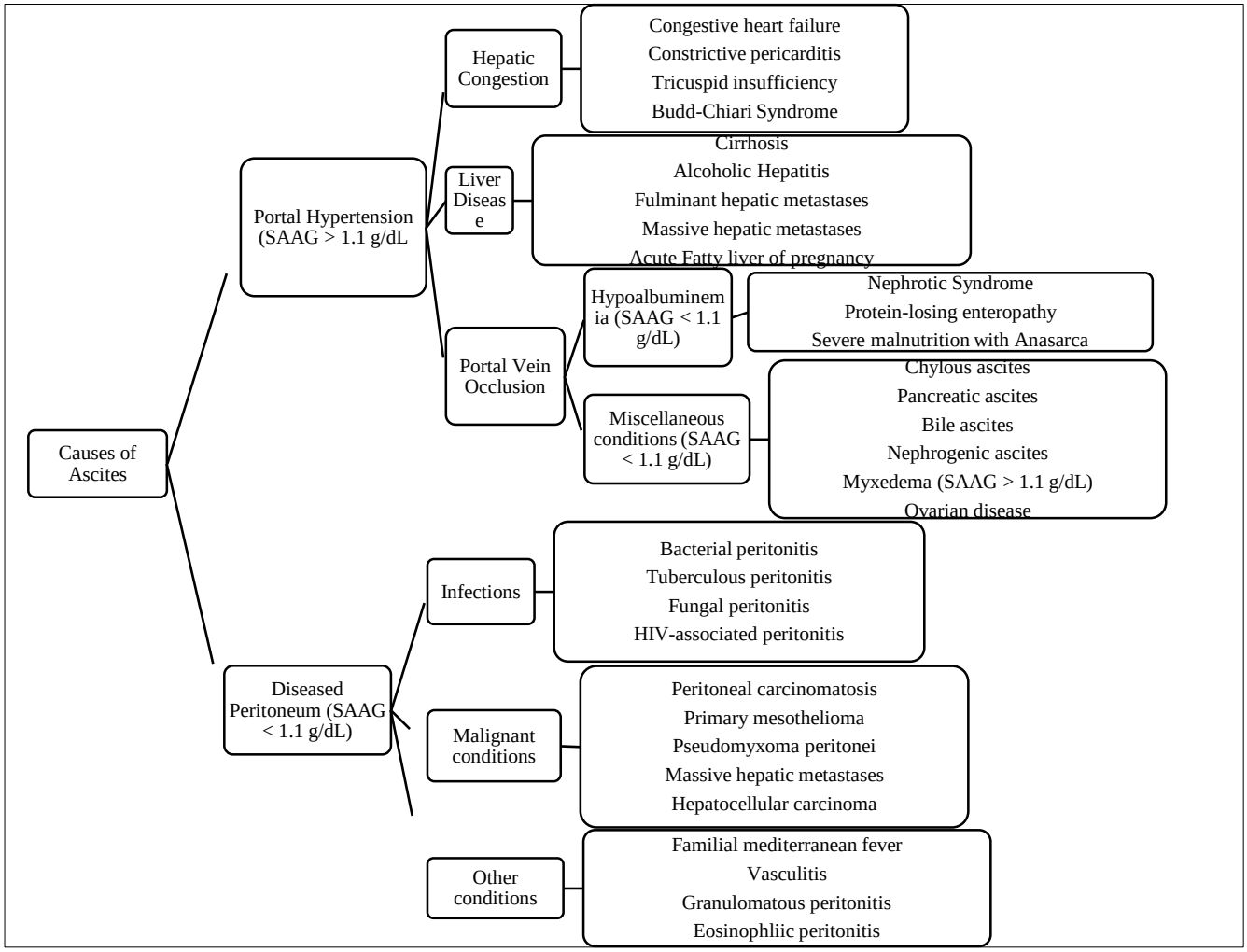

Figure 1: Causes of ascites.[2]

CASE REPORT

A 30-years-old man complained about shortness of breath which started to develop 2 weeks before admission. Shortness of breath is felt come and go, then getting worse when doing activities or lying down. While sleeping, the patient should use 3-4 pillows. Patient also complained about coughing which started to develop 3 months before admission. The cough feels more severe at night. Patient also complained of fever at night, weight loss has been felt since the last 2 months, nausea, and the stomach has been getting bigger since last week. This patient had a history of pulmonary tuberculosis, but dropped out of treatment.

At admission, the patient's consciousness is compos mentis with blood pressure was 111/72 mmHg, heart rate was 84x/minute, respiratory rate was 24x/minute, temperature was 36.2^{O}C, and oxygen saturation was 99% with 10 liters oxygen via non rebreathing mask. On physical examination, icterus was found on both eyes and skin. Cardiac examination reveals increased jugular venous pressure with cardiac murmur on auscultation. On lung examination, ronchi was found on both lungs. Abdominal distended and shifting dullness was found on abdominal examination. Scattered spider angiomata was found on his chest and upper abdomen. Laboratory test showed leukocytosis [11.00×10^3 µl (4.0-10.0)], transaminitis [serum glutamic pyruvic transaminase (SGPT) 925 U/l (0-42), serum glutamic-oxaloacetic transaminase (SGOT) 321 U/l (0-37)], hyponatremia [114 mmol/l (130-145)], hyperkalemia [5.8 mmol/l (3.5-5.5)], and hypoalbuminemia [serum albumin 2.8 g/dl (3.8-5.1)]. Chest X-ray revealed infiltrate and fibrotic on left lung which gives the impression of old pulmonary tuberculosis and abdominal ultrasound showed echo-free fluid appears in the abdominal cavity and right pleural cavity which shows the impression ascites, and right pleural effusion. Echocardiography examination showed right heart failure with ejection fraction 61%, severe tricuspid regurgitation and moderate pulmonal regurgitation. Ascites fluid aspiration was performed on this patient with result clear yellow on color, Rivalta test was negative, glucose 203 mg/dL (70-110), total protein 0.8 g/dl, albumin in ascites fluid 0.60 g/dl (3.80-5.10), and lactate dehydrogenase (LDH) 127 U/l. The SAAG was 2.2 g/dl. Based on examination, the patient was diagnosed with ascites due to cirrhosis, accompanied by right heart failure, pulmonary tuberculosis, right pleural effusion, and transaminitis.

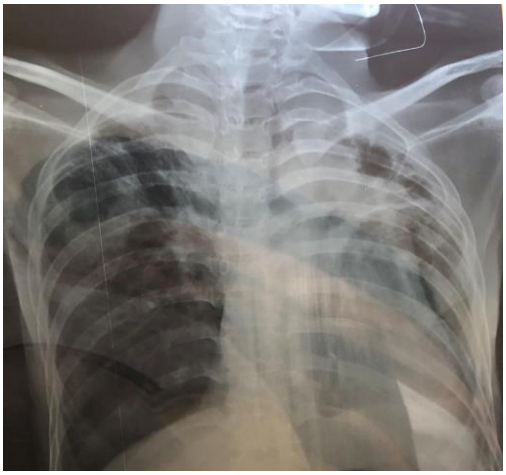

Figure 2: Chest X-ray showed infiltrate and fibrotic on left lung.

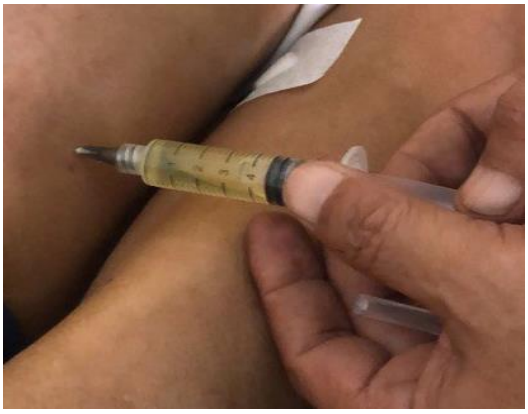

Figure 3: Ascites fluid aspiration showed clear yellow on color.

The patient was treated by spironolactone to reduce ascitic fluid, then furosemide and hydrochlorothiazide. Then, cardiac problems were treated by digoxin, bisoprolol, and trimetazidine. Oral albumin was given to treat hypoalbuminemia. For pulmonary tuberculosis, the patient was treated by modification of anti-tuberculosis drugs (streptomycin, levofloxacin, and ethambutol), ipratropium bromide and salbutamol nebulizer, and methylprednisolone. Curcuma was given to treat the transaminitis. All medications given were tolerated during admission. Patient was discharged after 17 days of treatment.

DISCUSSION

In this study, we reported a case of a patient with ascites due to cirrhosis accompanied by right heart failure and pulmonary tuberculosis. Ascites is defined as an excess of fluid within the peritoneal cavity. Patients typically show progressive abdominal distension that may be painless or associated with abdominal discomfort, weight gain, early satiety, shortness of breath, and dyspnea resulting from fluid accumulation and increased abdominal pressure. Ascites is clinically apparent when the patient presents either symptomatically with abdominal distension and bulging flanks. Typically flank dullness to percussion is noted, often appreciated higher up the abdominal wall than normally expected and accompanied by shifting when the patient is repositioned. Evidence of pleural effusions can be found. Ascites can be asymptomatically when radiological imaging shows unexpected peritoneal fluid accumulation.[1,3,6]

Ascites is known to be multifactorial and seems to result from the combination of portal hypertension and liver insufficiency. The reorganization of hepatic structure in cirrhosis is responsible for an increase in hydrostatic pressure in sinusoid capillaries, which leads to an increase in local synthesis of vasodilator substances, such as nitric oxide. Thus, there is a decrease in splanchnic arterial resistance.[7] Compensatory mechanisms then occur, especially an increase in cardiac output and activation of metabolic pathways to increase effective volaemia (sympathetic nervous system and renin-angiotensin-aldosterone pathway) with the development of hyperaldosteronism and sodium retention. Sodium retention causes fluid accumulation and expansion of extracellular fluid volume which results in the formation of peripheral edema and ascites. Moreover, hypoalbuminemia owing to hepatic insufficiency is responsible for a decrease in oncotic pressure, which also facilitates the fluid leakage from the intravascular to interstitial space. Because of the reorganization of the hepatic structure in cirrhosis, the capillaries are no longer fenestrated and protein concentration is then poor in this fluid.[2,7]

Ascites can occur in advanced right heart failure from various causes such as constrictive pericarditis, mitral stenosis, tricuspid regurgitation, cor-pulmonale and cardiomyopathy. Patients with ascites due to heart failure may report dyspnea, orthopnea, and peripheral edema. Clinical signs seen in right heart failure include right ventricular heave, S3, tricuspid valve murmur, jugular venous distention, a Kussmaul sign and oedema to the point of anasarca.[3,8] Ascites can also occur from peritoneal infection, such as tuberculosis. Peritoneal tuberculosis with the formation of ascites results from hematogenous spread of mycobacteria from a primary pulmonary source. As up to 20% of patients with pulmonary disease may have extrapulmonary manifestations, with general features of a persistent cough, hemoptysis, weight loss, fever, night sweats, abdominal distention, abdominal pain, and diarrhea. Ascites may be localized or generalized in the peritoneal cavity. Ultrasonography findings that are most specific include localized ascites, septations, fine strands, and lymphadenopathy with hypoechoic centers indicating caseating necrosis.[3,9]

SAAG and total protein levels from ascitic fluid are useful to distinguish the etiology of ascites. In patients with ascites of unclear etiology, and an elevated SAAG ≥1.1 g/dl, a low total protein level is more suggestive of

underlying cirrhosis whereas an elevated total protein level >2.5 g/dl is more suggestive of ascites from underlying heart failure. The presence of unexplained lymphocytic ascites with a SAAG of <1.1 g/dl and an ascites fluid protein content >3 mg/dl refers to the possibility of tuberculosis.[1,3]

This patient has multiple potential causes of ascites. From this patient, we found increased jugular venous pressure, abdominal distended and shifting dullness on abdominal examination, then scattered spider angioma was found on his chest and upper abdomen, suggesting this patient has cirrhosis. This patient also has transaminitis and hypoalbuminemia. The patient has an elevated SAAG (>1.1 g/dl), which is found in both cirrhosis-related ascites and heart failure. But he has an ascites total protein level of 0.8 g/dl, which is less than 2.5 g/dl threshold and suggest his ascites is related to cirrhosis. In cirrhosis, hepatic sinusoids are less permeable due to fibrous tissue deposition, resulting in ascites with low protein content.[5] The treatment of ascites is based on symptomatic therapies, including sodium restriction and diuretics.[5,7] This patient was treated with a combination of spironolactone, furosemide and hydrochlorothiazide to reduce the ascitic fluid. Sodium intake is also restricted during treatment. Our case showed a progressive improvement in response to the treatment.

CONCLUSION

Ascites has a major impact on quality of life and is associated with a poor outcome. The formation of ascites can be from many causes and provides unique challenges in distinguishing and treatment. SAAG and total protein levels from ascitic fluid are useful to distinguish the etiology of ascites. The management of ascites is focused on regimen of diuresis and sodium restriction. This symptomatic treatment should always be associated with treatment of related causes.

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