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Case Report

Ascites in peripartum cardiomyopathy: case report

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ABSTRACT

Ascites is the abnormal accumulation of excess fluid in peritoneal cavity.1 Normally, peritoneal cavity contains 25–50 mL of ascitic fluid, which allows for the movement of bowel loops past one other and helps hydrate serousal surfaces. Cirrhosis is the most common cause of ascites in the Western world (75%), followed by peritoneal malignancy (12%), heart failure (5%) (include peripartum cardiomyopathy), and peritoneal tuberculosis (2%). A 19-years-old woman diagnosed with peripartum cardiomyopathy. She came with chest pain and increased of her abdominal perimeter since 6 month ago, a month after she got cesarean delivery. She was given high protein and low sodium diet, water restriction, treated with furosemide 40 mg, spironolactone 25 mg, and abdominal paracentesis with total 1500 cc of yellowish ascites fluid was evacuated. Early detection is required in this case to ensure effective management without any complications. Treatment depends upon the cause of the ascites. Dietary sodium restriction and diuretics remains the first line therapy for its management. The use of diuretics needs close monitoring and follow up, including weight loss, electrolytes, and patient's condition daily.

Keywords: Ascites, Peripartum cardiomyopathy, Furosemide, Spironolactone, Abdominal paracentesis

INTRODUCTION

Ascites is the abnormal accumulation of excess fluid in peritoneal cavity.1 Normally, peritoneal cavity contains 25-50 mL of ascitic fluid, which allows for the movement of bowel loops past one other and helps hydrate serousal surfaces.[1] Pathogenetically based approach to the choice of pharmacotherapy and optimization of minimal invasive treatment may improve the quality of life and increase the survival rate.[2] The key to successful management of patients with ascites may be the stratification of the risk of an adverse outcome and personalized therapy.3 Cirrhosis is the most common cause of ascites in the Western world (75%), followed by peritoneal malignancy (12%), heart failure (5%), and peritoneal tuberculosis (2%).[3]

Cardiomyopathies are not very common diseases, but may cause severe complications, making a substantial contribution to maternal morbidity and mortality during pregnancy, in the immediate peripartum period, and up to months later.[4] Peripartum cardiomyopathy (PPCM) has to be differentiated from other causes of heart failure and it is unclear in what percentage PPCM persists to chronic, stable heart failure as patients with non-specific symptoms around pregnancy may remain undiagnosed and are only identified months or years later.[5] Presenting symptoms in PPCM patients are highly variable but may include fatigue, dyspnea, orthopnea, palpitations, chest pain, and decreased exercise tolerance.[6] Fluid overload or sign of congestion can be found in patients with chronic heart failure, such as rales or ronchy in auscultation of the lungs, signs of pleural effusion, distension/ increased jugular venous pressure, hepatomegaly, peripheral edema, S3 gallop in auscultation due to increased left ventricular diastolic end pressure, and ascites.[7] Here we presented a

case of 19-years-old woman diagnosed with ascites caused by peripartum cardiomyopathy.

CASE REPORT

We report a case of 19-years-old woman diagnosed with peripartum cardiomyopathy. She came to the emergency room with chest pain and increased of her abdominal perimeter since 6 month ago, a month after she got cesarean delivery, which resulted in dyspnea. She also complained fatigue, abdominal pain, yellowish eyes, and her arms and feet getting bigger since a week. Those were the reason she sought medical assistance. Hematochezia, melena, hematemesis, haemoptysis, menstrual disorder, and cough were not present. She never consumed alcohol and drugs, neither having tattoo. Her pregnancy history was unknowned. The examination showed 120/80 mmHg blood pressure, 112 bpm regular heart rate, 23 breaths per minute, 37oC of temperature, SpO2 99% with oxygen 2 liter per minute by nasal cannula, 47.9 kg of body weight. Pale and icteric conjunctiva, caput medusae, prominent abdoment with fluid wave, balloted spleen with splenomegaly (schuffner 3), and bilateral upper and lower limb edema were found. Thorax examination was found normal.

Table 1: Follow up of body fluid balance and weight loss.

Hospitalized	Body Fluid Balance (mL)	Weight loss / weight	Serum Potassium (mmol/L)
Day 1	-1600	0.6 kg / 47.3 kg	4.0
Day 2	-900	0.3 kg / 47.0 kg	-
Day 3	-1200	0.4 kg / 46.6 kg	-
Day 4	-600	0.2 kg / 46.4 kg	3.0
Day 5	-600	0.2 kg / 46.2 kg	-
Day 6	-500	0.1 kg / 46.1 kg	-
Day 7	-1000	0.3 kg / 45.8 kg	2.4
Day 8	-800	0.2 kg / 45.6 kg	4.0
Day 9	-1700 (1500 mL by paracentesis)	0.5 kg / 45.1 kg	-
Day 10	-400	0.1 kg / 45.0 kg	-

Laboratory work-up upon admission showed: haemoglobin 8.6 g/dL, red blood cell count 4.49x106/µL, white blood cell count 6.31×103/µL with neutrophil 64.1% and lymphocyte 28.7%, platelets count 255×103/µL, haematocrit 29.7%, iron serum 26 µg/dL, TIBC 460 µg/dL, UIBC 434 µg/dL, ferritin 10.2 ng/mL, total protein 6.2, albumin 2.7, globulin 3.5, total bilirubin 3.41 mg/dL, direct bilirubin 2.3 mg/dL, ALT 130 U/L, AST 59 U/L, blood sugar 91 mg/dL, BUN 55 mg/dL, serum creatinine 0.8 mg/dL, sodium 137 mmol/L, potassium 4.0 mmol/L, chloride 94 mmol/L, also anti HCV and HbsAg non reactive. Abdominal ultrasound revealed hepatomegaly due to congestive liver and ascites, adnexal mass was not pressent. Electrocardiography (ECG) 12-lead revealed sinus tachycardia. Posteroanterior chest x-ray revealed cardiomegaly and pleural effusion hemithorax dextra.

The patient was hospitalized for 10 days. She was given high protein and low sodium diet, water restriction, and treated with furosemide 40 mg once a day, spironolactone 25 mg once a day, bisoprolol 1.25 mg once a day, sildenafil 25 mg twice a day, and lactulose. Body weight loss and body fluid balance was measured everyday as seen in Table 1. Echocardiography examination was shown ejection fraction 17%, dilated right atrial, decreased systolic and diastolic function of left ventricle, and global hypokinetic. In the ninth day, we performed abdominal paracentesis and ascites fluid analysis was checked for cytology, total 1500 cc of yellowish ascites fluid was evacuated. The ascites fluid analysis showed negative rivalta, glucose 107 mg/dL, protein 3.4 g/dL, albumin 1.45 g/dL, LDH 359 U/L. Cytology examination showed white blood cell count 0.085 10.3/µL with mononuclear cell 95.3 and polimorphonuclear cell 4.7%, and red blood cell count 0.002 10.3/µL. Thus, serum-ascites albumin gradient (SAAG) was 1.86 g/dL.

In the last day, the patient no longer feel dyspnea and there was no sign of peripheral edema. The patient got discharge at the tenth day, and advice to regularly visit outpatient ward for cardiomyopathy and ascites follow up, diuretics and supportive treatment was given.

DISCUSSION

In this case, the ascites and chest pain emerged since 6 months ago (after caesarean). In echocardiography examination, ejection fraction was 17% and its showed dilated right atrial, decreased systolic and diastolic function of left ventricle, and global hypokinetic. Davis MB. et al defined peripartum cardiomyopathy as symptomatic heart failure presenting in the last month of pregnancy and up to 5 months postpartum, an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricle systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found, left ventricle ejection fraction is less than 45% and there may or may not be ventricular dilatation.[8] Liver disease as a consequence of heart failure has been known as congestive hepatopathy which produces liver damage through several pathogenic mechanisms: increased sinusoidal pressure leads to hepatic

stellate cell activation and decreases nitric oxide production by endothelial cells through shear stress, all of which induce sinusoidal ischemia and promote fibrogenesis, sinusoidal stasis and congestion promote sinusoidal thrombosis, which in turn contributes to liver fibrosis by causing parenchymal extinction and by activating hepatic stellate cells via protease-activated receptors, accumulation of exudate into the space of Disse due to the existing congestion impairs diffusion of oxygen and nutrients to hepatocytes and accelerates fibrosis pathways, decreased hepatic blood flow further aggravates liver ischemia, and portal venous inflow is reduced as a result of the transmission of the elevated central venous pressure to the sinusoidal network, while arterial flow can also be compromised in patients who also harbor a left-sided heart failure, then causes increased in both intravascular pressure in the portal venous system and capillary hydrostatic pressure causing fluid to leak into the abdominal cavity and become ascites.[9,10]

Ascites is free fluid within the peritoneal cavity and formed because of conditions directly involving the peritoneum (infection, malignancy), or diseases remote from the peritoneum (liver disease, heart failure, hypoproteinaemia).[3] Ascites is graded as: grade 1, mild ascites detectable only by ultrasound examination; grade 2, moderate ascites manifested by moderate symmetrical distension of the abdomen; and grade 3, large or gross ascites with marked abdominal distension.[3] The most frequent symptoms associated with clinically evident ascites are increased abdominal girth that cause recent weight gain, shortness of breath, satiety, and generalized abdominal pain.[3] Patients must have at least 1500 mL of fluid to be detected on physical examination.[3] Shifting dullness is the most sensitive finding in ascites examination.[3] On the other hand, fluid wave sign has the poorest sensitivity in the diagnosis of peritoneal fluid, but its specificity is high.[3] Pleural effusion develops in patients with ascites and it is right-sided in 85%, left-sided in 13%, and bilateral in 2% of the cases.[3] Peripheral oedema usually follows ascites and is related to hypoproteinaemia.[3] Ultrasound and CT-scan can be used as modality to diagnose ascites that show a space around the liver and these can be used to demonstrate quite small subclinical amounts of fluid.[3]

The formation of ascites is a resulted from several factors, such as:[10]

Portal hypertension: cirrhosis leads to portal hypertension, through deformation and occlusion of intra-hepatic vessels; blockage of portal venous blood flow. An increased in both intravascular pressure in the portal venous system and capillary hydrostatic pressure causing fluid to leak into the abdominal cavity and become ascites. Enhancement of RAAS activity: Portal hypertension enhances RAAS activity by triggering splenic and systematic circulation changes, which resulted in sodium and water retention. Increased secretion or enhanced activity of other vasoactive substances: during cirrhosis, the secretion and activity of atrial natriuretic peptide, prostaglandins, and other vasoactive peptides increased and stimulates extensive dilation of the splenic arterioles and, hence, increases venous inflow. Hypoalbuminemia: during cirrhosis, albumin synthesis is markedly reduced and caused a drop in plasma colloid osmotic pressure, which promotes the leakage of fluid from the plasma into the abdominal cavity, forming ascites. Obstruction of lymphatic drainage: intra-hepatic vasculature can be obstructed during ascites, while the production of hepatic lymph increases. Ascites is formed when the lymph reflux exceeds the drainage capacity of the thoracic ducts. Chylous ascites is formed if there is obstruction and rupture of the cisterna chyli.

Ascites fluid appearance is clear, green, straw coloured, or bile stained.[3] Ascites total protein and serum-ascites albumin gradient (SAAG) are two inexpensive tests but the most useful in determining the source of ascites (Table 2).[3] Ascites polymorphonuclear cells increased with peritoneal infection or with other intraabdominal inflammatory conditions such as diverticulitis or cholecystitis.[3] According to Table 2, SAAG (1.86g/dL) and ascites protein (3.4 g/dL) of this patient is high, we can conclude that the cause of ascites is cardiac problem.

Table 2: Differential diagnosis among the three most common causes of ascites.[3]

Causes of Ascites	Serum-Ascites Albumin Gradient (cut-off 1.1 g/dL)	Ascites Protein (cut-off 2.5 g/dL)
Cirrhosis	High	Low
Cardiac ascites	High	High
Peritoneal malignancy/ peritoneal TB	Low	High

Therapy of ascites, whether by diuretics or paracentesis, reduces clinical symptoms and improves quality of life, but may be associated with side effects and because they act downstream of the pathophysiological cascade, they are mainly symptomatic and are not associated with an improvement in survival.[10] Treatment must therefore be appropriate to the clinical state and must be properly monitored and tailored to the patient. The spectrum of therapeutic intervention ranges from sodium restriction alone (rarely used), or combination with diuretic use, therapeutic paracentesis, and, for the most severe groups, transjugular intrahepatic portosystemic shunt (TIPS) and eventually liver transplantation.[9] The patient with cirrhosis who is accumulating ascites on an unrestricted sodium intake excretes less than 10 mmol (approximately 0.2 g) sodium daily in the urine. Extrarenal loss is about 0.5 g. Sodium taken in excess of 0.75 g will result in ascites, with every gram retaining 200 mL of fluid.[10]

Diuretics can be divided into two main groups according to their site of action.[10] The first group inhibits Na+–K+–

Cl− (NKCC2) co-transporter in the ascending limb of the loop of Henle, including furosemide and bumetanide. It is not appropriate to use diuretics alone since the sodium remaining in the tubule is reabsorbed in the distal tubule and collecting duct because of hyperaldosteronism.[10] A randomized controlled trial has shown furosemide alone to be less effective than spironolactone. Thiazides inhibit reabsorption in the distal convoluted tubule, have a longer half-life, may cause hypotension, and should not be used in the treatment of ascites.[10] The second group, spironolactone, amiloride, and triamterene, block sodium reabsorption in the distal tubule and collecting duct.[10] Because spironolactone is an aldosterone antagonist and aldosterone is the main driver of sodium retention in cirrhosis, it is the drug of first choice in the treatment of ascites due to cirrhosis.[10] They are weakly natriuretic but conserve potassium. Potassium supplements are not usually necessary indeed this type of diuretic sometimes needs to be temporarily stopped because of hyperkalaemia.[10]

In a controlled trial, daily large volume (4–5 L) paracenteses (LVP) associated with 40 g of intravenous albumin reduced hospital stay compared with standard diuretic treatment.[10] However, readmissions to hospital, survival, and causes of death did not differ significantly between the LVP and diuretic groups.[10] Later on, the safety of a single total paracentesis combined with intravenous albumin was shown to be equally effective and safe.[10] LVP is not entirely benign as a percentage of patients, particularly those in whom >5 L of ascites is removed, will develop 'paracentesis circulatory dysfunction' (PCD), an entity that results in worsening vasodilation, with consequent increased incidence of hyponatremia, renal failure, and death.[10] The concomitant use of intravenous albumin (6–8 g/L of ascites removed) has been able to minimize the rate of PCD.[10]

Therapy of ascites, whether by diuretics or paracentesis, reduces clinical symptoms and improves quality of life, but may be associated with side-effects and, because they act downstream of the pathophysiological cascade, they are mainly symptomatic and are not associated with an improvement in survival.[3] General management of ascites is listed below:[3]

Diagnostic paracentesis with first presentation or with any symptom/sign suggest SBP. 70–90 mmol sodium diet; weigh daily; check serum creatinine and electrolytes. Spironolactone 50-100 mg daily. Consider paracentesis if tense ascites. Consider adding furosemide 40 mg daily after 4 days; check serum creatinine and electrolytes before. Give combination therapy of spironolactone/frusemide in tense ascites cases and performed closed monitoring. Daily weight loss maximum 0.5 kg/day (1.0 kg/day in those with peripheral oedema). Stop diuretics if precoma ('flap'), hypokalaemia, azotaemia, or alkalosis. Continue to monitor weight; increase diuretics as necessary. Avoid non-steroidal anti-inflammatory drugs.

In this case, the patient got sodium retention dietary, as suggested in literature, because sodium taken in excess of 0.75 g will result in ascites, with every gram retaining 200 mL of fluid.[3] Historically, such patients were recommended a diet containing 22-40 mmol/day of sodium (approximately 0.5-1.0 g/day).[3] There are two therapeutic approaches that can be used initially: spironolactone alone, or a combination of spironolactone with frusemide.[3] Both have their advocates and may be chosen depending on the degree of ascites and the clinical setting, with combined therapy being more appropriate for tense ascites where close follow-up is feasible.[3] Spironolactone alone, the starting dose is 50-100 mg/ day according to the degree of ascites and the clinical setting.3 If there has been insufficient clinical response after 3-4 days (weight loss less than 300 g/day), then the dose is increased to 100 mg/day (if initiated at 50 mg/day) or by 100 mg/day every 4 days to a maximum of 400 mg/day, unless hyperkalaemia develops.[3] The disadvantage of starting with spironolactone alone is the delay before its clinical effect and associated hyperkalaemia, if there is insufficient clinical response or no response on spironolactone alone (when taking 200 mg/day) or associated hyperkalaemia, a loop diuretic such as furosemide is added at a dose of 20-40 mg/day.[3] In combination therapy, The treatment is started with the combination of spironolactone (100 mg) and furosemide (40 mg) daily.[3] The disadvantage of starting with combination therapy may be the need for closer laboratory monitoring and monitoring of daily weight is also necessary.[3] As mentioned earlier, the rate of ascitic fluid reabsorption is limited to 700–900 mL/day.[3] If a diuresis of 2–3 L is induced, much of the fluid must come from non-ascitic, extracellular fluids including oedema fluid and the intravenous compartment.[3] This is safe so long as oedema persists.[3] Indeed, diuresis may be rapid (greater than 2 kg daily) until oedema disappears.[3] To avoid the risk of renal dysfunction there should be a maximum daily weight loss of 0.5 kg/day, with a maximum of 1.0 kg/day in those with peripheral oedema.[3] In this case, we use combination of furosemide 40 mg daily, and spironolactone 25 mg daily, and we also monitoring daily weight loss, serum creatinine and electrolytes. We could keep her weight loss not over 0.5 kg/day, except in the first day because of peripheral oedema. In the 7th day, she got hypokalemia (2.4 mmol/L) thus the furosemide was stopped and KCl 25 meq was given until serum potassium ≥ 4.0, the furosemide can be given again. In the ninth day, we performed abdominal paracentesis. Total 1500 mL of ascites fluid was evacuated. McGibbon, et al. Paracentesis in determining the etiology of the ascites, the presence of infection, and will often give temporary relief of dyspnea, chest and abdominal discomfort, and anorexia in patients.[11]

CONCLUSION

This report illustrate a patient with ascites caused by peripartum cardiomyopathy. Early detection is required in this case to ensure effective management without any

complications. Ascites may occurred due to hepatic or extra-hepatic causes. Treatment depends upon the cause of the ascites. Dietary sodium restriction and diuretics remains the first line therapy for its management, which is also effective in this patient. The use of diuretics needs close monitoring and follow up, including weight loss, electrolytes, and patient's condition daily. Adequate management of ascites is important since this improves quality of life, thus several up-to-date clinical guidelines are available and can be used.

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