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Chylous Ascites as a Rare Manifestation of Congestive Cardiac Failure in a District Hospital

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Abstract Chylous ascites is an uncommon type of ascites which may be associated with liver diseases, tuberculosis or malignancy. However, in a minority of cases, it is due to heart failure. This case report describes a 59 year old Malay gentleman who presented with multiple episodes of abdominal distension for the past six months associated with exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea and bilateral leg swelling. He denied having any fever, night sweats, anorexia or loss of weight. He did not exhibit any altered bowel habit or vomiting. Physical examination revealed a non tender, distended abdomen with an everted umbilicus. His legs were swollen bilaterally till the knees. Jugular venous pulsations were raised. Lungs revealed bibasal crepitations. Abdominal paracentesis showed milky turbid appearing peritoneal fluid. Laboratory studies revealed an exudative chylous ascites. Tuberculous workout, serum tumor markers and viral hepatitis screening were negative. His transthoracic echocardiogram revealed global hypokinesia with a left ventricular ejection fraction of 30%. Contrast Enhanced Computed Tomography scan of the thorax, abdomen and pelvis showed features consistent of liver cirrhosis with portal hypertension most probably attributable to the severe heart failure. There was no evidence to suggest malignancy. He was started on anti-heart failure drugs in which his ascites showed significant improvement within three months. Congestive cardiac failure should be considered as one of the causes for chylous ascites.

Keywords: *ascites, swelling, abdomen, chylous, turbid*

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1. Introduction

Chylous ascites is an uncommon form of ascites, defined as leakage of the lipid-rich lymph into the peritoneal cavity. [1] It is described as milky, cloudy and turbid peritoneal fluid often seen during the process of abdominal paracentesis. Chylous ascites is a rare condition (<1%) defined by the presence of high concentration of triglycerides in the ascitic fluid (>200 mg/dl). [2] Among the most common etiologies are intra abdominal malignancy, peritoneal tuberculosis, liver cirrhosis and trauma. Peritoneal tuberculosis may be a more common cause for chylous ascites in the developing world and therefore should be considered by attending physicians. Constrictive pericarditis, dilated cardiomyopathy, ischemic heart disease and rheumatic mitral stenosis have sometimes been associated with chylous ascites. [3] Although ascites is a common complication of liver disorders, only 3% of cases are associated with cardiac pathologies such as left heart failure. [4] Other seldom seen inflammatory causes include sarcoid, retroperitoneal fibrosis, systemic lupus erythematous (SLE), chronic ambulatory peritoneal dialysis (CAPD) and thyrotoxicosis. We report on a patient who developed chylous ascites secondary to congestive heart failure.

2. Case Presentation

A 59 year old Malay gentleman presented with multiple episodes of abdominal distension for the past six months.

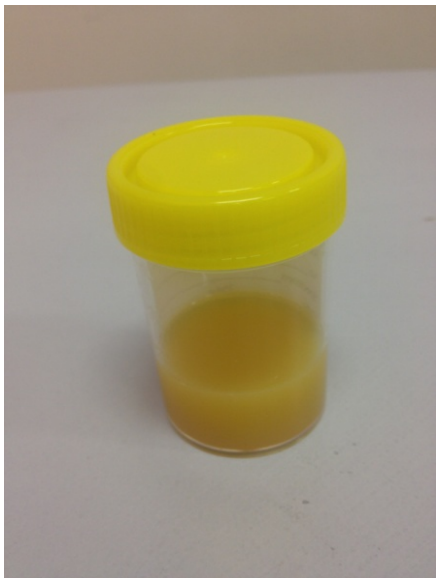

Figure 1. (Before treatment)

He complained of mild abdominal discomfort without any significant abdominal pain. The abdominal discomfort was associated with exertional dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea. He also frequently complained of bilateral leg swelling till the knees which he noted was pitting in nature. There was no nausea, vomiting or altered bowel habit. He denied having any prolonged fever or bleeding tendency. He did not complain of any epigastric pain or swellings in the axilla, groin or neck. There was no history of anorexia or loss of weight. He denied any usage of traditional or complementary medications. He was mildly jaundiced. His past medical history included a 10 year history of Type 2 Diabetes Mellitus on which he was on dual oral hypoglycemic agents. He also had 2 vessel coronary artery disease which was diagnosed on a coronary angiogram and he subsequently underwent angioplasty with stenting for two vessels five years ago. Since then, he has been relatively chest pain free. His other significant past medical history included a prosthetic mechanical mitral valve replacement done in year 1999 for severe mitral regurgitation. He is currently on lifelong warfarin. He is an active chronic smoker with 20 pack years of smoking. Besides that, he is a teetotaler. He works as a teacher in a secondary school in the district of Segamat. There was no family history of malignancy. Physical examination revealed a thin well built gentleman. He was hypervolaemic with pink conjunctiva. He had a pulse rate of 92 beats per minute, regularly regular with good pulse volume. He was mildly tachypnoeic with a respiratory rate of 26 breaths per minute. There was no stigmata of chronic liver disease. No palpable lymph nodes were felt at the neck, axilla and groins. The jugular venous pulsation was raised. His apex beat was displaced to the sixth intercostal space at the mid axillary line. There was a pansystolic murmur heard loudest at the apex suggestive of mitral regurgitation. Lungs revealed bibasal crepitations. His abdomen was not tender but distended with tense flanks. There was obvious bilateral leg swelling till the knees. Abdominal paracentesis revealed cloudy and milky ascites (Figure 1). It was exudative in nature with the serum ascitic albumin gradient of less than 11.1 g/L. The ascitic fluid showed a triglyceride level of 250 mg/dl (11.1 mmol). The peritoneal fluid for adenosine deaminase activity was not elevated. The acid fast bacilli (AFB) Polymerase Chain Reaction in peritoneal fluid was also not detected. The fluid cytology showed no evidence of malignancy except for some mesothelial cells with reactive changes. The complete blood count, renal profile, liver function test and fasting lipid profile as shown in Table 1. His platelet count was 187000/uL. He had a baseline platelet count of 350000/uL which was taken a year previously. The peripheral blood film was not significant. His thyroid function tests tests were within normal range. Antinuclear antibody and rheumatoid factor titers were not elevated. All serum tumor markers were not elevated. The tuberculous and viral hepatitis screening were negative. Chest radiograph showed gross cardiomegaly. His transthoracic echocardiogram revealed global hypokinesia with a left ventricular ejection fraction of 30% and moderate mitral regurgitation. There was no thickening or calcification of the pericardium. Contast Enhanced Computed Tomography scan of the thorax, abdomen and pelvis showed liver cirrhosis with spenomegaly measuring a span of 15.2 cms. There was circumferential wall thickening in the sigmoid and mid transverse colon. There was no evidence of other solid organ malignancy. Subsequent colonoscopy revealed normal colonic mucosa. No peritoneal biopsy was carried out. He was started on multiple anti heart failure drugs comprising of a beta blocker, angiotensin converting enzyme inhibitor, diuretics, antiplatelet, statin, aldosterone antagonist, digoxin and other essential drugs. He was placed on a low fat diet. He refused for any invasive cardiology intervention. He was followed up regularly and his failure symptoms responded within three months of intensive medical treatment. His chylous ascites also showed dramatic improvement (Figure 2) with frequency of abdominal paracentesis significantly reduced.

Figure 2. (After treatment)

Table 1. Laboratory Values

Variables (Serum)	Value
Hemoglobin (g/l)	8.7
Total White Cell Count(per mm^3)	4720
Platelet (per mm^3)	187000
Urea (mmol/l)	7
Sodium(mmol/l)	129
Potassium(mmol/l)	3.9
Creatinine (umol/l)	120
Total protein (g/l)	71
Albumin (g/l)	22
Aspartate Transaminase (U/L)	45
Alanine Transaminase (U/L)	43
Alkaline Phosphatase (U/L)	278
Total Bilirubin (umol/l)	60
Direct Bilirubin (umol/l)	29
Lactate Dehydrogenase (U/L)	212
Total Cholesterol (mmol/l)	5.7
Triglyceride (mmol/l)	2.2

3. Discussion

Chylous ascites is an uncommon condition featured by accumulation of ascitic fluid with high triglyceride content in the peritoneal cavity. [5] Detection rates would have

been more significant if abdominal paracentesis were performed in all patients with ascites. Chylous ascites may be the result of many pathological conditions, including congenital defects of the lymphatic system, nonspecific bacterial, parasitic and tuberculous peritoneal infection, liver cirrhosis, malignant neoplasm, blunt abdominal trauma and surgical injury. [6] In a developing country like Malaysia, mycobacterium infection must be considered in all cases of chylous ascites. Other risk factors such as low social background, retroviral disease and malnutrition could contribute to peritoneal tuberculosis being an important consideration for chylous ascites. In a recent systematic review including 131 studies from developing and developed countries (with a total of 190 patients) who had atraumatic chylous ascites, the most common causes in adults were malignancy (25%), cirrhosis (16%), mycobacterium infection (15%), and a variety of uncommon causes (23%). [7] Liver involvement in cardiac failure is common and it depends on the type and severity of heart disease. In patients with moderate to severe heart failure, 95% show hepatomegaly, 75% peripheral edema, 20–25% pleural effusion and up to 25% show ascites. [2] A complete focused history taking and physical examination are essential in guiding us towards the etiology of chylous ascites. Surgical history or previous trauma in the abdomen may differentiate between atraumatic and traumatic chylous ascites. In this case, the patient presented with severe heart failure which contributed to his chylous ascites. The liver cirrhosis with splenomegaly seen on the computed tomography imaging was most probably due to the congestive cardiac failure. This gives rise to the term 'cardiac cirrhosis'. Decompensated right ventricular or biventricular heart failure causes transmission of elevated right atrial pressure to the liver via the inferior vena cava and hepatic veins.[9] At a cellular level, venous congestion impedes efficient drainage of sinusoidal blood flow into terminal hepatic venules. [9] Sinusoidal stasis results in accumulation of deoxygenated blood, parenchymal atrophy, necrosis, collagen deposition, and, ultimately, fibrosis. [9] It was obvious that the chylous ascites in this patient was certainly due to the cardiac cirrhosis caused by congestive cardiac failure as all other causes for his liver cirrhosis were ruled out. Congestive cardiac failure causing chylous ascites may result from two distinct mechanisms. They are: an increase in the abdominal lymph production and an ineffective development of collateral flow. [2] High venous pressure increases the abdominal lymph production due to an augmented capillary filtration and, even though lymphatic flow increases in response, the augmented central venous pressure reduces lymphatic drainage. [8] The lymph flow of the thoracic duct can increase by up to 12 fold the normal rate, but the stiffness of the veno-lymphatic junction in the neck limits lymphatic flow. [8] Unlike mechanical obstruction of the thoracic duct, where the development of lymphaticovenous collaterals channels provides lymphatic drainage, generalized central venous hypertension caused by cardiac disease prevents the development of an effective collateral flow. [2] However, the fact that heart failure is a common disorder but chylous ascites is clinically rare suggests that some undiscovered factors are responsible for its pathogenesis.[5]

4. Conclusion

Congestive cardiac failure should be considered as one of the causes for chylous ascites. It may present as a diagnostic challenge in a district hospital. In chylous ascites secondary to congestive cardiac failure (CCF), management of underlying congestive cardiac failure is the most crucial**.** Pharmacological treatment with diuretics, anti-heart failure drugs and optimization of modifiable risk factors are the gold standard.

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Statement of Competing Interests

The authors declare that there is no conflict of interest regarding the publication of this paper.

List of Abbreviations

None.

Informed Consent

Informed consent was obtained from the patient for the publication of the study.

References

[1] A. Cárdenas and S. Chopra. "Chylous ascites." *American Journal of Gastroenterology* 2002; vol. 97, no. 8, pp. 1896-1900.

[2] Ezequiel Ridruejo et. al. Chylous ascites as main manifestation of left ventricular dysfunction. A case report. *BMC Gastroenterology*; 2005; 5:25.

[3] Hurley MK, Emiliani VJ, Comer GM et. al. Dilated cardiomyopathy associated with chylous ascites. *Am J Gastroenterol*; 1989; 84: 1567-1569.

[4] Hüseyin Altuğ Çakmak et. al. Development of chylothorax and chylous ascites in a patient with congestive heart failure. *Arch Turk Soc Cardiol*. 2011; 39(6):495-49.

[5] Min-Huang Hsieh, M.D. Chylous Ascites as a Manifestation of Thyrotoxic cardiomyopathy in a Patient with Untreated Graves' disease. *Mary Ann Liebert, Inc*. 2010; Vol 20, Number 6.

[6] Thaworn Subtaweesin, M.D. Chylous Ascites caused by Lutembacherûs Syndrome: A Case Report and Review of the Literature. *Siriraj Med* J. 2008; 60:78-80.

[7] D. C. Steinemann, D. Dindo, P.A. Clavien et. al. "Atraumatic chylous ascites: systematic review on symptoms and causes." *Journal of the American College of Surgeons* 2011; vol. 212, no. 5, pp. 899-905.

[8] Dumont AE, Clauss RH, Reed GA et. al. Lymph drainage in patients with congestive heart failure: comparison with findings in hepatic cirrhosis. *N Engl J M.1963*; 269: 949-952.

[9] Moller S, Bernardi M. Interactions of the heart and the liver. Eur Heart J. 2013 Sep. 34(36):2804-11.