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CASE STUDY

Chylous Ascites in Disseminated Tuberculosis; A Rare Case Report

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ABSTRACT
Chylous ascites (CA) is an uncommon clinical entity and often associated with a poor outcome. It occurs as a result of disruption of abdominal lymphatics either due to trauma or secondary to obstruction. It is easily diagnosed by the milky appearance of ascitic fluid and a definitive diagnosis is only made by elevated triglyceride levels in the fluid. Hereby we report a 38-year-old labourer, a case of disseminated tuberculosis who presented with spontaneous chylous ascites. He was treated with Antitubercular drugs, diuretics with dietary support including law salt, high-protein and low-fat and therapeutic paracentesis.
Keywords*; Chylous Ascites, Disseminated Tuberculosis, Antitubercular Drugs, Therapeutic Paracentesis*

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INTRODUCTION

Chylous ascites is a rare challenging clinical condition which is developed due to the accumulation of milky chyle in the peritoneal cavity. It is a rare form of ascites, occurs as a result of an interruption in the lymphatic system. It is diagnosis when the concentration of triglycerides in the ascitic fluid is >200 mg/dl.[1]

Worldwide, the commonest causes in adults are abdominal malignancy, cirrhosis, and tuberculosis, the latter being most prevalent in developing countries. While in paediatric age group, congenital abnormalities of the lymphatic system and trauma are commonest causes.[2] We are highlighting this rare clinical finding associate with cirrhosis because an appropriate treatment depends on accurate diagnosis.

CASE PRESENTATION

A 38-year-old man labourer by occupation was admitted to our medicine department with complaints of law grade fever for 6 months, cough with expectoration on and off for 5 months followed by progressive distension of abdomen. He had multiple prescriptions containing many antimicrobial agents but he was partially relieved on medication. Currently he was treating as a case of chronic liver disease. Though, he had negative history for alcoholism, viral markers or significant family history. He also denied for history suggestive of malena or hemetemesis.

Patient had distended abdomen with everted umbilicus.[Figure 1] On examination, he had pallor but no icterus, B.P-110/80 mmHg, pulse rate-78/ min, respiratory rate was-21/min and temp-99.4°F. Abdominal examination revealed free fluid in peritoneum and splenomegaly. Chest showed bilateral minimal crepitations. Cardiac and central nervous system examinations were normal. He had no history of diabetes mellitus, hypertension, malignancy, trauma, drug intake, alcoholism, malena or any similar episodes in past.

His blood routine revealed haemoglobin 11.0 g/dl, total leukocyte count 1100/mm3 and platelet counts 35,0000/mm3. Serum electrolytes, renal function tests and fasting lipid profile were within normal range.

His random blood sugar was 100 mg/dL. His liver function test revealed serum bilirubin 0.5 mg/dL, serum aspartate aminotransferase (AST) 47 U/L, alanine aminotransferase (ALT) 18 U/L, serum alkaline phosphatase 295 U/L, serum protein 4.8 g/dL and serum albumin 2.6 g/dL. Prothrombin time and INR were within normal limits. Routine and microscopy of urine was normal. ELISA for surface antigen (HBsAg) and human immunodeficiency virus (HIV) were nonreactive. Serology for Hepatitis A Virus Hepatitis B and Hepatitis C Virus were negative. Chest X Ray showed clustered opacities in bilateral lung fields with bilateral minimal effusion (R>L). ECG was normal. Sputum for AFB was negative.

CECT abdomen showed gross ascites along with thickening of peritoneum, multiple lymph nodes noted at pariportal, pre/para-aortic, mesentery and aortocaval location (largest measuring 12 mm), enlarged spleen, mild hepatomegaly with rounded outline. It was surprisingly that peritoneal fluid tapping revealed milky fluid.[Figure 2] Though, there was no evidence of infection or trauma. Routine and microscopic examinations of ascitic fluid revealed total protein– 5.4 g/dL, glucose level of 122 mg/dL, triglycerides– 400 IU/L, cholesterol– 112 mg/dL, ADA level of 27 IU/L and total cell count-400 cells/mm3 (95% lymphocytes and 5% Polymorphs). Culture of fluid was sterile and cytological examination was negative for malignant cells. Upper GI Endoscopy showed D1ulcer with clear base, healing edges and normal surrounding mucosa without any evidence of portal hypertension.[Figure 3]

Based on history, CXR and ascetic fluid findings, we started daily regimen of antitubercular drugs. Patient was further managed with Diuretics and large-volume Paracentesis under blood pressure monitoring. He was promoted to take law salt diet with high-protein and low-fat contents. He was found to respond well on antitubercular treatment and discharged. He was apparently asymptomatic on 5 months of follow up. Unfortunately he could not follow to OPD further. Even though we tried to contact him but could not trace.

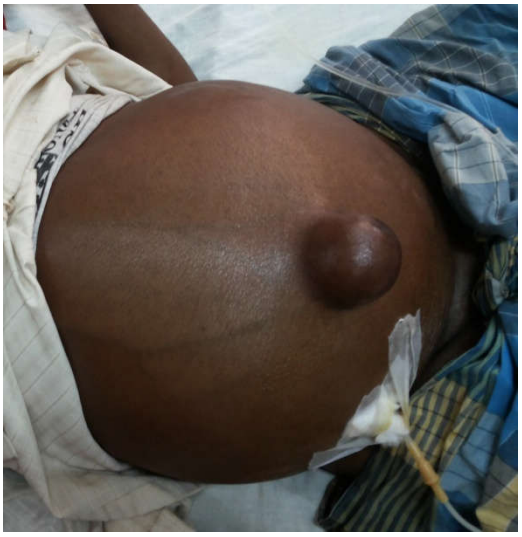

Figure 1; Distension of abdomen showing gross Ascites Figure 2; Milky colour of Ascitic fluid (Chylous fluid)

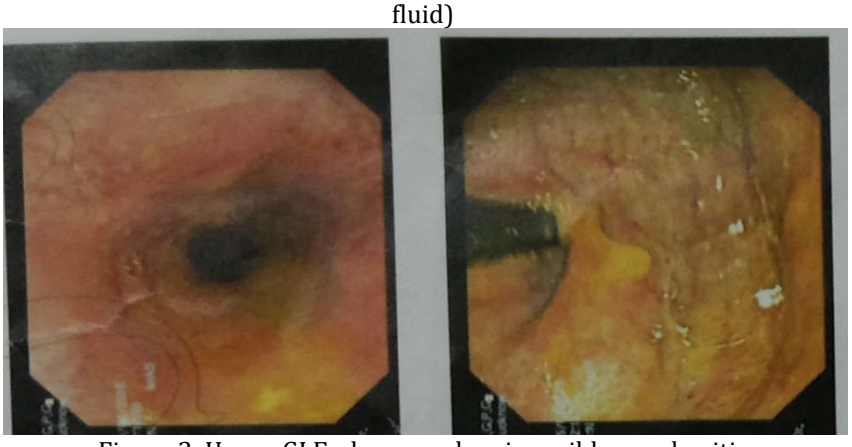

Figure 3; Upper GI Endoscopy showing mild oesophagitis

DISCUSSION

According to a study at a large university based hospital over a 20-years period, the incidence rate of chylous ascites was reported 1 per 20,000 admissions.[3] Morton, in 1694, reported the first case of CA in a 2-year-old boy who died with tuberculosis.[2]

On the basis of lymphangiography and inspection at laparotomy, three basic mechanisms have been proposed for the development of chylous ascites. One is due to lymph flow obstruction caused by external pressure (mass) which causes leakage from dilated subserosal lymphatics into the peritoneal cavity. Second is due to exudation of lymph through the walls of dilated retroperitoneal vessels, which may occur with or without a visible fistula into the peritoneal cavity as in congenital lymphangiectasia. Third is due to direct leakage of lymph through a lymphoperitoneal fistula as a result of traumatic thoracic duct obstruction.[4]

It is also proposed that the increased caval and hepatic venous pressures may precipitate Chylous Ascites through large increased production of hepatic lymph in cases of right-sided heart failure, pericarditis and dilated cardiomyopathy.[2] It is very essential to know the aetiology of Chylous Ascites because underlying cause determines the ongoing management.

CA may be due to traumatic and non traumatic causes.[2] Non traumatic cases are as (I) Congenital causes-Primary lymphatic hypoplasia, Klippel-Trenaunay syndrome, Yellow nail syndrome, Primary lymphatic hyperplasia, Lymphangioma and Familial visceral myopathy. (II) Neoplastic- Solid organ cancers, Lymphoma, Sarcoma, Carcinoid tumours, Lymphangioleiomyomatosis, Chronic lymphatic leukemia. (III) Infectious causes- Tuberculosis, Filariasis, Mycobacterium avium in AIDS and Ascariasis. (IV) Inflammatory causes- Fibrosing mesenteritis, sarcoidosis, idiopathic retroperitoneal fibrosis, systemic lupus erythematous, peritoneal dialysis and hyperthyroidism. (V) Miscellaneous causes- Cirrhosis, Congestive heart failure, Constrictive pericarditis, nephritic syndrome, Celiac disease and Whipple's disease. (VI) Drug causes- Calcium channel blockers, sirolimus.

Traumatic causes may be as (I) Surgical causes-Abdominal aneurysm repair, Placement of peritoneal dialysis catheter, Radical and laparoscopic nephrectomy, Retroperitoneal lymphadenectomy, Inferior vena cava resection, Pancreaticoduodenectomy, Vagotomy, Laparoscopic Adrenalectomy and Gynecological operative procedures. (II) Nonsurgical causes-Penetrating abdominal trauma, Blunt abdominal trauma, Battered child syndrome and Shear forces to the root of the mesentery.

The underlying pathophysiology of chylous ascites in cirrhosis of liver is due to rupture of serosal lymphatic channels which are dilated because of excessive lymph flow.[5] Spontaneous chylous ascites is reported in 0.5% patients of cirrhosis with ascites.[6] Chylous ascites is generally presented as progressive and painless abdominal distention, occurring over the course of weeks to months. Duration depends upon the underlying pathology. Patients with trauma or surgery of abdoman or thorax may present with an acute onset.[4]

Routine and microscopic examination of ascetic fluid is an important diagnostic tool which shows cloudy and turbid appearance with triglyceride values above 200mg/dl.[7] Computed tomography of the abdomen is useful in identifying pathological intra-abdominal lymph nodes and masses. Lymphangiography and Lymphoscintigraphy can assist in detecting abnormal retroperitoneal nodes, leakage from dilated lymphatics, fistulization, and patency of the thoracic duct. Lymphangiography is considered gold standard in cases of obstruction but limited used due to more complications, such as tissue necrosis, fat embolism, and contrast induced hypersensitivity.[4]

Management of chylous ascites is largely based on Dietary, medical and surgical therapies especially depending on cause.

Dietary management includes high protein and low fat diet with medium chain triglycerides. Patients with advanced cirrhosis should be managed with a low-sodium diet and diuretics such as spironolactone. Total parenteral nutrition TPN should be tried in patients who do not respond to the above measures. Somatostatin and octreotide are also used successfully for chylous effusions due to lymphatic leakage. Patients with end-stage disease not amenable to medical or surgical treatment, repeated large-volume paracentesis is beneficial. Patients with cirrhosis and CA resistant to conservative therapy treated successfully with the use of TIPS are reported. Surgical intervention may be beneficial especially in patients with chylous ascites of postoperative, neoplastic or congenital origin. Peritoneovenous shunts were reasonable options for patients, who are refractory to medical therapy and high risk for surgery.[2]

CONCLUSION

Chylous ascites is a relatively uncommon and challenging clinical disorder. Majority of cases are due to intra-abdominal malignancy and liver cirrhosis. Treatment of the underlying cause is paramount step of

management, especially in the context of treatable cause. Therefore, treating physicians should be aware of this relatively uncommon entity associated with tuberculosis.

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