

Case report

Tuberculous Chylous Ascites in a HIV Infected Adolescent; Challenges of Management in a Low Resource Country.

Abstract.

Tuberculous chylous ascites is an uncommon condition even in sub-Saharan Africa where the burden of tuberculosis (TB) and Human immunodeficiency virus (HIV) is very high. In low resource countries, TB is a major cause of chylous ascites while elsewhere; the common causes are lymphomas, cirrhosis and congenital abnormalities involving the lymphatic channels. When tuberculous chylous ascites occurs in a child with HIV/TB co-infection, it poses great challenges in management. We present a case of a HIV/TB co-infected Nigerian adolescent with tuberculous chylous ascites.

KEYWORDS; Chylous, Ascites, Tuberculosis, HIV

Introduction.

Chylous ascites is the leakage of the lipid-rich lymph into the peritoneal cavity giving rise to the milky appearing peritoneal fluid rich in triglyceride.¹ This leakage usually follows an obstruction, fibrosis or malignant infiltration of the lymphatic channels. In non-resource challenged countries, abdominal malignancies and cirrhosis are the common cause of chylous ascites, unlike in low resource countries of sub-Saharan Africa where tuberculosis and filariasis remain the leading causes.^{1,2}

The global incidence of chylous ascites is 1 in 20,000 hospitalized patients and there is no sex predilection³ Diagnosis is made in the presence of peritoneal fluid with turbid hue and high triglyceride level exceeding 200mg/dl² Prognosis is largely dependent on the underlying cause. Outcome is good in tuberculous ascites following the institution of anti-tubercular therapy and diuretics or paracentesis when ascites is massive. Outcome is further improved with specialized dietary management using high protein, low fat diet with medium chain triglyceride (MCT), total parenteral nutrition (TPN) and drugs such as somatostatins/octreotide.⁴⁻⁶

Case Presentation.

We present the case of a 19 year old HIV/TB co-infected Nigerian male who presented with a three month history of progressive abdominal distension and weight loss. Other complaints were easy satiety, cough, weakness with occasional abdominal pain and shortness of breath. On physical examination he weighed 40kg, was cachectic with prominent ribs, zygomatic, mandibular bones and generalized lymphadenopathy with suppurating cervical lymph nodes. The abdomen was uniformly distended with a girth of 76cm and ascites demonstrable by fluid thrill. The abdominal distension made palpation of the intra-abdominal organs difficult. Remarkable investigation results included peripheral blood film which showed dimorphic cells with predominance of hypochromic cells suggestive of anemia of chronic disease, haemoglobin level of 7g/dl, erythrocyte sedimentation rate of 150mm/1st hour and HIV viral load of 1,604copies/ml. Histology sample obtained using fine needle aspiration of cervical lymph node demonstrated presence of caseating granulomatous inflammatory cells which was suggestive of Tb-lymphadenitis. Immunohistochemistry of the lymph nodes revealed paracortical hyperplasia with dermatophobic lymphadenopathy which is consistent with reactive lymphadenopathy. Abdominal ultrasound scan revealed multiple paraortic hypoechoic lymph nodes and multiple

hypoechoic masses in the spleen. Similarly, abdominal computed tomographic scan demonstrated marked ascites with hepatosplenomegaly, irregular hypodense splenic nodules with soft tissue mass superficial to the superior mesenteric artery and enlarged lymph node. Ascitic fluid examination showed a milky white fluid with a triglyceride of 846mg/dl, negative cytology and negative genexpert for tuberculosis. Prior to onset of abdominal distension, patient had protracted cough and chest x ray findings of miliary shadows suggestive of Koch's disease and had been started on anti-tubercular therapy but he defaulted. He was recommenced on anti-tubercular therapy with fixed dose combination of rifampicin, isoniazid, ethambutol and pyrazinamide and later continued on antiretroviral therapy also fixed dose combination of tenofovir, lamivudine, dolutegravir. He also had sessions of abdominal paracentesis. Patient completed anti-tubercular therapy and currently on antiretroviral therapy. Total parenteral nutrition and MCT diet were not available in the region. However within 12 months of commencement of management for chylous ascites, during which there were several follow up visits, patient weight progressively increased to 54kg, HIV viral load reduced to 224copies/ml with good physical activities and improved quality of life.

Discussion.

Tuberculosis has remained a major opportunistic infection co-existing with HIV in children and adults, particularly in sub-Saharan African where the burden of HIV and TB is still high.⁷ However, association of HIV/TB co-infection with chylous ascites is largely uncommon. In low resource countries, common causes of chylous ascites are mycobacterium tuberculosis, filariasis and ascariasis.^{6,8} Tuberculous lymphadenitis could cause lymph node enlargement or lymph node fibrosis leading to lymphatic channel obstruction and leakage of chyle into the peritoneal space.⁶

Rarely, chylous ascites may occur from immune reconstitution inflammatory reaction following antitubercular therapy in patients with HIV infection and on antiretroviral therapy.⁹ This is a paradoxical reaction that could result in lymph node enlargement and fibrosis leading to chylous ascites. This patient is an adolescent who is HIV/TB co-infected on antiretroviral therapy who defaulted on antitubercular therapy prior to onset of abdominal distension. Other causes of chylous ascites include malignancies (commonly lymphoma), cirrhosis, congenital malformations of the lymphatics, radiotherapy, blunt abdominal trauma, and surgical injury to the lymphatics.^{1,2} Diagnosis of chylous ascites is confirmed with the presence of milky white lipid rich peritoneal fluid with triglyceride level >200mg/dl.² The Mainstay of management of tuberculous chylous ascites is the use of anti-tubercular therapy. Further management of chylous ascites involves the use of somatostatins analogue such as octerotide which decreases gastric and intestinal secretion thereby reducing splanchnic blood flow leading to reduced lymph flow. Supportive management includes dietary recommendation of MCT based formular, TPN, somatostatins and repeated paracentesis. Surgery is recommended in failed medical and supportive treatment.^{6,9} All of these management options are not readily available in a low resource setting like ours.

Conclusion.

Tuberculous chylous ascites though uncommon can occur as a complication of HIV/TB co-infection particularly in sub-Saharan Africa. Management with antituberculous therapy, TPN, MCT based formular and abdominal paracentesis produces good outcome. However, despite the unavailability of TPN, MCT-Based formular and somatostatins, we had a good outcome.

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