Somato Publications ISSN: 2688-1071

Archives of Clinical Case Reports

Case Report

The Cirrhotic-Milky Man

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Received: 09 April 2020; Accepted: 27 May 2020; Published: 28 May 2020

Citation of this article: : Maadarani, O., Abdelhafiz, H., Bitar, Z., Shoeb, S., Alshehab, D. (2020) The Cirrhotic-Milky Man. Arch Clin Case Rep, 3(2): 10-15.

Abstract

Chyloperitoneum as a manifestation of liver cirrhosis and portal hypertension is rare. Chylothorax can be an association in such cases due to trans-diaphragmatic passage of chylous through microspores. A 58 -year-old man with a known history of Nonalcoholic steatohepatitis (NASH) related liver cirrhosis was admitted to hospital with a 2 weeks history of progressive shortness of breath and abdominal distention. He was found to have significant left sided pleural effusion and ascitic fluid. Thoracentesis showed transudative fluid of milky and cloudy appearance proved to be chylous by analysis and a similar fluid was also obtained on a diagnostic paracentesis. No history of trauma, tumor or recent medical procedures. Conservative management including dietary modification, subcutaneous somatostatin analogues (octreotide) and diuretics led to gradual reduction of pleural drainage and the milky appearance of the pleural effusion vanished.

Keywords: Liver cirrhosis, Chylothorax, Chyloperitoneum

Learning Points

- Chyloperitoneum and chylothorax can be a manifestation of decompensated liver cirrhosis.
- In cirrhotic liver trans-diaphragmatic passage of chylous may explain chylothorax.
- Triglyceride (TG) level >200 mg/dL and >110 mg/dL is diagnostic for chyloperitoneum and chylothorax respectively.

Introduction

The milky and cloudy appearance of ascitic or pleural fluid raises suspicion of chylous effusion. The level of triglyceride and cholesterol in the fluid and serum and their ratio can help in confirming chylous effusion. We present a case of decompensated liver cirrhosis with portal hypertension and fluid accumulation in abdominal and pleural cavity proved to be chylous effusion and responded well to conservative treatment.

Case Description

A 58-year-old male patient with a 3-year history of non alcoholic steatohepatitis (NASH) related liver cirrhosis, type 2 diabetes mellitus and a history of endoscopic band ligation of esophageal varies one year back, presented to the Emergency Department with progressive shortness of breath and abdominal distension of 2 weeks

duration. No history of tumor, trauma or recent medical procedures. He denied fever or change of bowel habits. His regular medications included Spironolactone 100 mg once daily, Furosemide tablet 40 mg once daily, Propranolol 20 mg three times daily, Insulin glargine and Lactulose syrup. The patient denied alcohol or tobacco consumption.

He had history of therapeutic paracentesis 6 months back which was related to increase salt intake and non-compliance with diuretics. 5L fluid removed with serum-ascites albumin gradient (SAAG) > 1.1 g/dL.

Physical examination was remarkable for underweight (body mass index 17.5 kg/m^2), tachypnea (respiratory rate of 25/minute),-jaundiced skin and mucosa with palmar erythema and few spider nevi. There was no fever, his Blood pressure was 110/60 mm of hg,heart rate of 100 beats/min and oxygen saturation of 94% on room air. Lung auscultation revealed significantly decreased air entry in left hemithorax. Abdominal examination showed distention with positive shifting dullness. Lower limb edema was also noticed.

On presentation, complete blood count was remarkable for platelet count of 120,000 (normal range 150,000–450,000/l). The International Normalized Ratio (INR) was 1.6 (normal INR 1.1 or less). His metabolic panel showed Serum sodium of 128mEq/l, blood glucose level of 152 mg/dl (normal 70–140 mg/dl), total bilirubin of 40 mcmol/L (normal 1.7-20.5 mcmol/L), mildly elevated liver enzymes and an albumin level of 2.5 g/dl (normal range 3.5–5.1g/dl). Alpha fetoprotein and thyroid function were within normal limits. Chest X ray showed significant left sided pleural effusion (Figure 1). Echocardiography showed normal systolic function, with no evidence of valvular pathology. Abdomino-pelvic ultrasonography revealed signs of portal hypertension, splenomegaly, ascites and features of cirrhotic liver but no focal lesions.Thoracentesis performed and the pleural fluid turned to be turbid and milky (Figure 2) with transudative character and high level of triglyceride keeping with chylothorax (Table 1). A Pigtail catheter was inserted to drain the fluid, and almost 2 L of chylous fluid was drained in the first day (Figure 3). Similar cloudy and milky fluid was also obtained from a diagnostic abdominal paracentesis where serum-ascites albumin gradient (SAAG) was 1.4 g/dL indicates ascites due to portal hypertension. Triglyceride level in ascitic fluid was high and keeping with chyloperitoneum

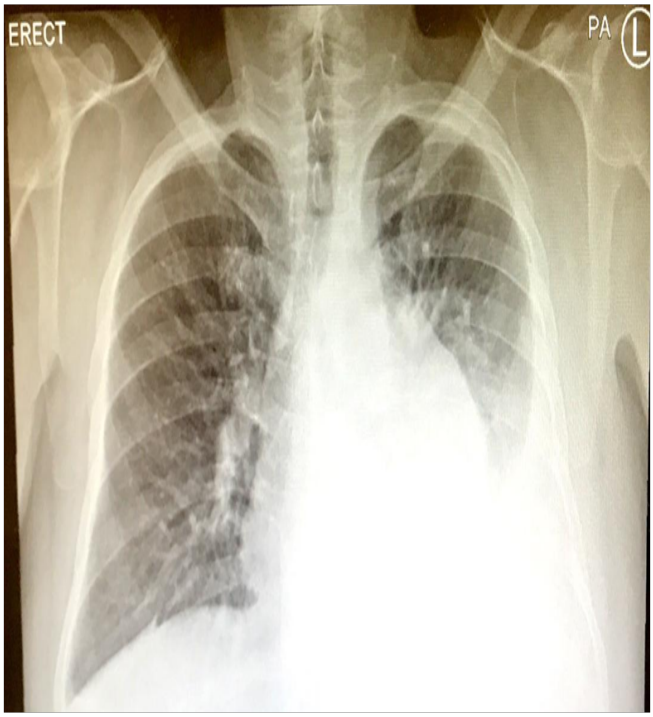

Figure 1: CXR on presentation.

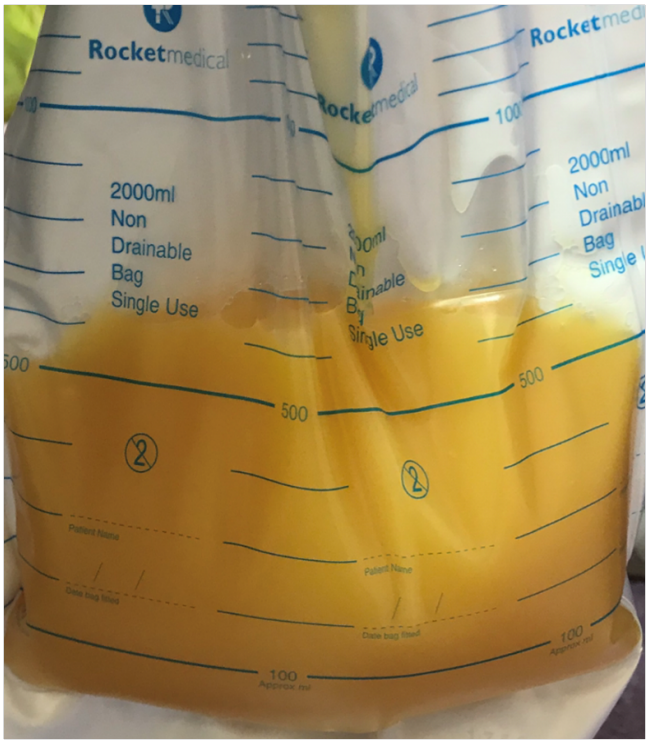

Figure 2: Chylothorax.

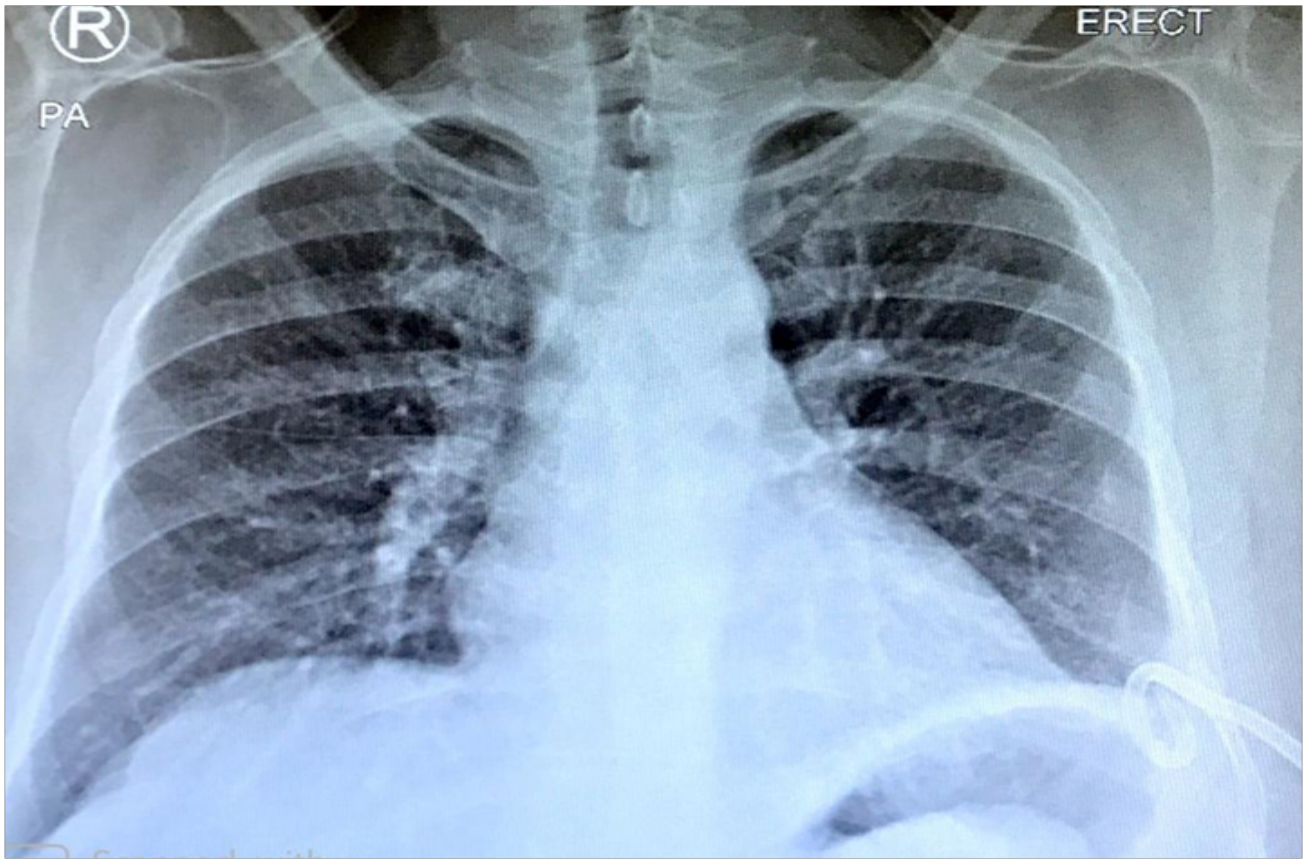

Figure 3: CXR after pigtail insertion.

(Table 2). Computed tomography of abdomen and chest showed no masses or lymph nodes. In addition to pleural fluid drainage and diuretics, patient was started on low fat diet with medium chain triglycerides and Octreotide subcutaneously 100 mcg every 8 hours. Gradually, the pleural drain became serous and the amount decreased to less than 100 ml on day 8. Pigtail catheter removed (Figure 4) and Octreotide discontinued.

The presence of classical characteristics of chylous fluid in the abdominal and pleural cavity in the absence of recent trauma, surgical procedures or tumors (history and imaging), raises the possibility of chyloperitoneum associated with chylothorax related to decompensated liver cirrhosis due to portal hypertension which considered a rare entity.

Discussion

A combination of lymph and emulsified lipids in a fluid gives a milky appearance and is called chyle. Small intestine is the place where chyle is formed during digestion of fatty food. Through the lymphatic vessels, chyle travels in one direction passing through lymph nodes directed to lymph trunks. Chyloperitoneum is the leakage of chyle in the peritoneal cavity where Chylothorax is the leakage of chyle in the pleural cavity.

The classical etiology of chylous leakage is trauma, malignancy, idiopathic and miscellaneous that cause disruption to lymphatic vessels and ducts [1]. In liver cirrhosis, ascites and hepatic hydrothorax are frequent findings, however the combination of chylous ascites and chylothorax is a rare manifestation.

Cirrhosis is an important cause of portal hypertension which can complicate with ascites. The mechanisms of portal hypertension are often classified as prehepatic, intrahepatic and posthepatic. Intrahepatic causes constitute the majority of cases of portal hypertension, and can be further classified into presinusoidal (e.g. schistosomiasis), sinusoidal (cirrhosis) and postsinusoidal (he-

Table 1: Pleural fluid analysis and interpretation.

Parameter	Pleural fluid (PF)	Interpretation
Color	Turbid, milky	
pH	7.76	No empyema
PF protein (g/dL)	1.7	
Serum protein (g/dL)	7.1	
PF/Serum protein ratio	0.23	Transudate (2)
PF Lactate Dehydrogenase (LDH) (IU/L)	67	Transudate (2)
Serum LDH (IU/L)	151	
PF/Serum LDH ratio	0.4	Transudate (2)
PF TG (mg/dL)	520	> 110, diagnostic for chylothorax (6)
Serum TG (mg/dL)	45.1	
PF to serum TG ratio	11.5	> 1 (diagnostic for chylothorax)(6)
PF Cholesterol (mg/dL)	48	< 200 (diagnostic for chylothorax)(6)
Serum cholesterol (mg/dL)	199	
PF to serum cholesterol ratio	0.24	< 1 (diagnostic for chylothorax)(6)
Cell count (/cmm)	2800	predominantly lymphocytes
Gram stain and Culture	Negative	
TB culture	No growth	
Cytology	No malignant cells	

Table 2: Ascitic fluid analysis and interpretation.

Parameter	Ascitic fluid	Interpretation
Color	Turbid, milky	
Total protein(g/dL)	2.3	
Serum- ascites albumin gradient (SAAG) (g/dL)	1.4	>1.1 diagnostic for portal hypertension (8)
Ascitic fluid Triglyceride (mg/dL)	400	> 200, diagnostic for chylous ascites (7)
Ascitic fluid Cholesterol (mg/dL)	50	
Serum cholesterol (mg/dL)	199	
Ascitic fluid to serum cholesterol ratio	0.25	Ratio <1,characteristic for chylous ascites(7)
Lactate dehydrogenase (IU/L)	140	Usually 110–200 in chylous ascites (7)
Amylase and lipase	Not elevated	
Cell count (/cmm)	900	lymphocytic predominance
Gram stain and Culture	Negative	
Glucose (g/dL)	70	
TB culture	No growth	
Cytology	No malignant cells	

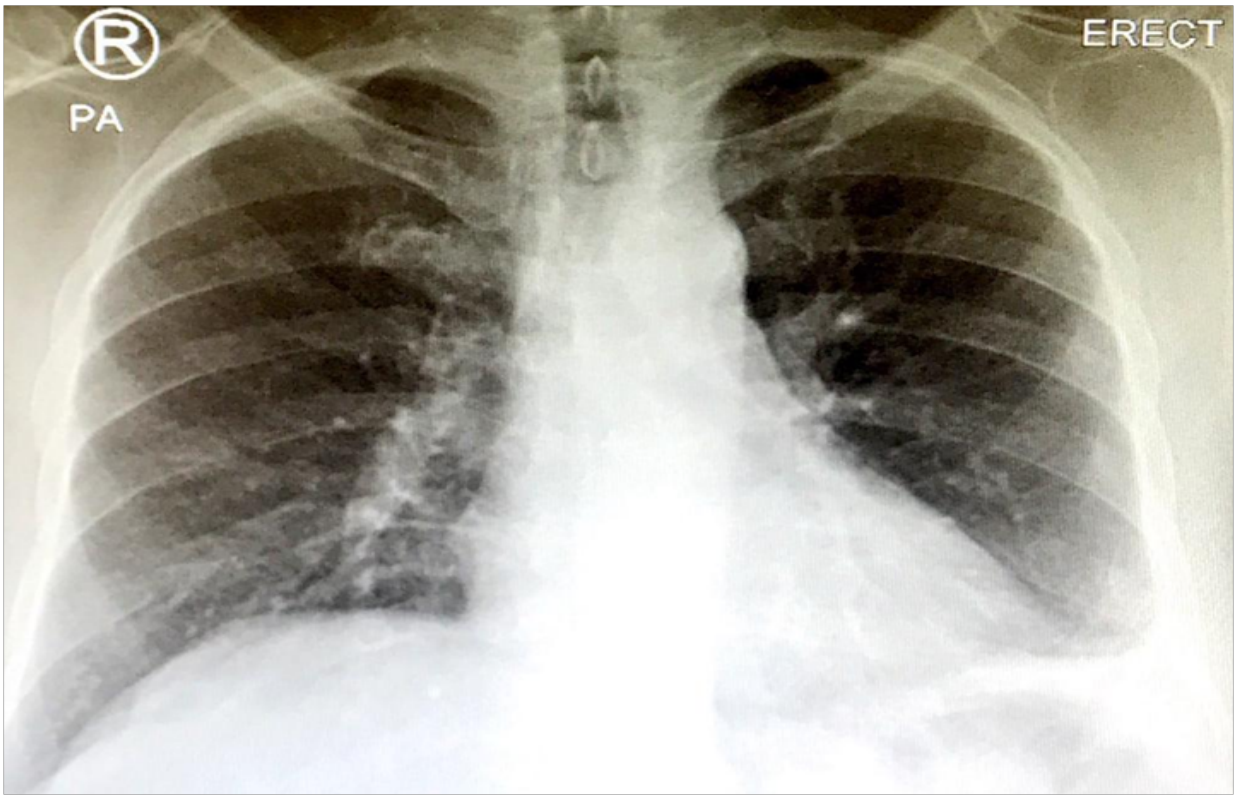

Figure 4: CXR on day 8 after removal of pigtail.

patic veno-occlusive disease) causes. The mechanism through which the chylous ascites appears due to portal hypertension is not well recognized and it has been proposed that increased caval and hepatic venous pressures cause a large increase in the production of hepatic lymph. Elevated lymphatic pressure secondary to portal hypertension can cause endothelial compromise or rupture of serosal dilated lymphatic channels and leads to chylous ascites formation [2]. Similar to hepatic hydrothorax, chylous ascites leaks through micro defects in the diaphragm from peritoneum into pleural cavity with the help of decreased intrapleural pressure forming chylothorax [3]. Chylothorax in liver cirrhosis is transudative, as opposed to the exudative nature of most of the chylous effusions from other etiologies [4]. In a retrospective study by Maldonado et al. [5], the characteristics of chylothorax were exudative in 86% of cases and transudative in 14% where patients with cirrhosis were mostly transudative.

Turbid and milky appearance of the fluid raises the possibility of chylous fluid. In our case, light's criteria of the pleural fluid showed transudative nature (Table 1). The measurement of TG and cholesterol level in fluid and serum and their ratio can generally differentiate the nature of fluid. A TG levels>110 mg/dL with a cholesterol level <200 mg/dl in pleural fluid is considered diagnostic for chylothorax. Pleural fluid to serum TG ratio >1 and Pleural fluid to serum cholesterol ratio <1 is also considered another diagnostic criteria for chylothorax [6]. Pseudochylothorax develops when an exudative effusion remains in the pleural space for a long period of time (often years) gradually becoming enriched with cholesterol. A TG level < 50 mg/dl with a cholesterol > 200 mg/dl is found in pseudochylothorax [9]. Leukocyte count in fluid >1000/uL with a lymphocyte predominance is charac-

teristic for chylothorax. For chylous ascites, a cutoff value of TG level>200 mg/dL and ascitic fluid to serum cholesterol ratio <1 is currently used for the diagnosis [7]. Serum to ascites albumin gradient(SAAG) in cirrhosis patient who presents with chylous ascites is usually above 1.1 g/dL which keeps with portal hypertension [8].

Low fat diet with medium-chain triglycerides can help in reducing chylous effusion. Somatostatin or its synthetic analogue (octreotide) reduces portal pressure by inhibiting glucagon and vasodilatation mediated by other splanchnic intestinal peptides. Somatostatin also decreases thoracic duct flow and its triglyceride content [2].

In our case, the symptoms of the patient were caused by fluid accumulation which proved to be a chylous ascites and chylothorax. In the absence of recent trauma or medical procedures by history and exclusion of obstructive lesions (malignancy) by imaging, with no evidence of infection (no empyema or TB), such combination would most probably considered as a manifestation of decompensated liver cirrhosis. Conservative treatment was successful with our patient.

Conclusion

The combination of chylous ascites and chylothorax can be a manifestation of decompensated liver cirrhosis.

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