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Case Report

A case of idiopathic chylous ascites

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Abstract

Chylous ascites is a rare condition found in 1 in 20 000 patients admitted to hospital with abdominal distention. It is caused by a limited number of pathologies but can, in rare situations, be idiopathic. Its management is difficult and usually involves correcting the primary pathology, making idiopathic chylous ascites particularly difficult to manage. We present a case of idiopathic chylous ascites extensively investigated over a period of several years. An incidental finding of B cell lymphoma was initially suspected to have been the primary cause of the ascites; however, after successful treatment of this condition, the patient's ascites did not resolve. Diagnostic difficulties and management are discussed and an overview of the diagnostic process is outlined through this case.

INTRODUCTION

Chylous ascites is an uncommon finding in patients with abdominal distension. Incidence is 1 in 20 000 hospital admissions [1]. It is associated with malignancy, infections, cirrhosis, trauma or previous abdominal surgery. We present a case of idiopathic chylous ascites in a patient where all known causes of underlying pathology were excluded.

SUMMARY

A 72- year-old man was referred to the gastroenterology clinic due to abdominal distension, weight loss and change in bowel habit. He had no significant past medical history, previous trauma, risk factors for liver disease or abdominal surgery.

A computed tomogram (CT) chest, abdomen and pelvis was performed demonstrating a right-sided pleural effusion and large volume ascites (Fig. 1). The liver, other abdominal viscera and lymph nodes appeared normal except for small simple liver and splenic cysts.

Both the pleural effusion and ascites were sampled—the aspirate appeared light pink and milky in color consistent with chyle. Analysis of the fluid revealed the presence of chylomicrons and raised levels of triglycerides (13.6 mmol/L) in keeping with chylous ascites. Fluid and serum LDH were normal and the serum-ascitic albumin gradient (SAAG) was 9 g/L, suggestive of an exudative process or malignancy rather than portal hypertension or heart failure. Fluid cytology revealed a small clonal B cell population (13% of total white cells; CD5-/CD10-/CD20+/CD23+) together with reactive T-cells, suggestive of possible low grade B-cell lymphoma. Myd88 mutation analysis was indeterminate. As part of work-up for lymphoma, bone marrow trephine biopsy and aspiration was performed and did not reveal evidence of malignancy. However, repeat sampling of the ascitic fluid returned clonal B-cells with identical phenotype. The working diagnosis was low-grade B-cell lymphoma, and the patient was given first-line (Rituximab single agent) and then second-line (Bendamustine and Rituximab) treatment. Neither treatment resulted in the improvement of ascites, despite disappearance of the clonal B cells from the ascitic fluid. PET scans showed no evidence of FDG-avid disease on three separate occasions, with no radiological thickening of the pleura or peritoneum suggestive of non-FDG avid lymphomatous deposits.

Echocardiogram did not show evidence of heart failure, valvular pathology or constrictive pericarditis and blood tests were negative for autoimmune liver diseases, hepatitis B and C, HIV and TB. Transjugular liver biopsy failed due to inadvertent brachiocephalic artery puncture, so transgastric endoscopic ultrasound left lobe liver biopsy was performed and revealed normal hepatic tissue with no evidence of cirrhosis or inflammation. Lymphangiography was not performed but there was no history of prior thoracic trauma or external beam radiotherapy.

The patient was started on a low triglyceride diet by dietitians and continued to have regular paracentesis for symptomatic relief. In 2022, he was admitted with spontaneous bacterial peritonitis (ascitic WCC 10×10^9/L). There was no perforation on plain radiography or CT imaging. Despite brief improvement with broad spectrum antibiotics, repeat ascitic fluid cultures returned multi-resistant *Escherichia coli* and *Enterococcus faecium*. Despite

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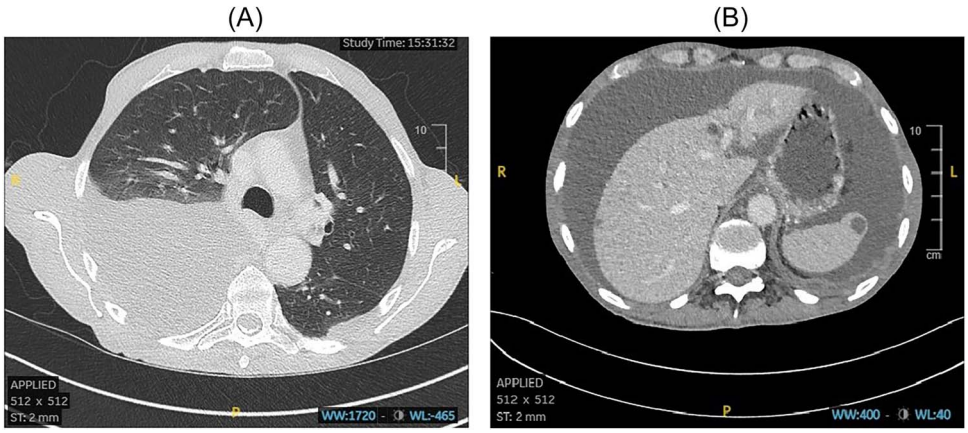

Figure 1. Computed tomography demonstrating (a) large right-sided pleural effusion with surrounding atelectasis and (b) significant abdominal ascites with benign liver and splenic cysts.

high leukocytosis and mixed bacterial ascitic culture, repeat CT imaging demonstrated no viscus perforation or abscess, and the patient was started on nasogastric feeding and therapeutic paracentesis when symptomatic from tense ascites.

Despite regular paracentesis, there was rapid re-accumulation of ascites and further deterioration with splinting of the diaphragm and respiratory compromise. After further clinical deterioration, repeat CT scanning of the abdomen was performed which demonstrated pneumoperitoneum and a possible perforation and the hepatic flexure of the colon. Further ascitic cultures grew *Candida albicans* and *E. faecium*. The patient died as a result of acute peritonitis despite conservative medical management as he was not fit for surgical intervention. A post-mortem was not undertaken after the case was reviewed by the medical examiner as the cause of death was peritonitis secondary to bowel perforation.

DISCUSSION

Chylous ascites is rare and usually has a poor outcome due to its frequent association with malignancy [1–3]. Other contributing factors to morbidity are the loss of a large volume of fluid, calorie-rich lipids and protein in the ascitic fluid. The mainstay of treatment is management of the underlying cause, but as this case shows, this is not always possible when the underlying cause is not identified.

In such situations, symptomatic management is required. Changes in oral diet ensure reduction in lymph flow—low lipid and high medium chain triglyceride diets are the first step, but total parenteral nutrition (TPN) can be started if adapted diets fail [2–4]. Medium-chain triglycerides are absorbed and transported to the liver as free fatty acids and glycerol, while long-chain triglycerides are converted to chylomicrons, thereby increasing lymph production. TPN may be used in selected cases when enteral diet has failed to improve ascites, as reduction in enteral feeding can result in reduction of lymph flow [1, 4, 5].

Both somatostatin and its analogues use have been reported in literature. The mechanism of action is not fully understood but is believed to reduce portal pressure and decrease peristalsis, leading to a reduced absorption of triglycerides into the lymphatic system [3, 4]. This treatment was not used in our case.

Repeated therapeutic paracentesis or indwelling shunts (e.g. peritoneal-venous 'Denver' shunts) may be performed for symptomatic relief, but this comes with the added risk of infection or shunt occlusion [1, 6, 7]. No previous studies have identified or postulated whether chylous ascites increases the risk of secondary bacterial peritonitis, or whether this is more likely a secondary complication of repeated ascitic drainage, but chronic loss of chyle does result in immune system compromise and malnutrition [8]. It is therefore crucial to involve dietitians as part of a multidisciplinary approach to management to ensure adequate calorie intake.

CONFLICT OF INTEREST STATEMENT

There are no conflicts of interest to declare.

FUNDING

No funding was received or required in this case.

ETHICAL APPROVAL

There was no ethical approval required in this case.

DATA AVAILABILITY

The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials.

GUARANTOR

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