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Case Study

Chylous ascites and gastric adenocarcinoma: A case report and review of literature

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A B S T R A C T

Chylous ascites is an uncommon finding characterized by the presence of a milky-appearing peritoneal fluid. The accumulation of chyle in the peritoneum develops when a disruption of the lymphatic system exists. The origin of this disruption may be secondary to traumatic injury, obstruction or rupture of the peritoneal or retroperitoneal lymphatic glands and by the exudation of lymph through retroperitoneal vessels. We report the case of an 81-year-old anticoagulated man presented with constitutional syndrome, abdominal distension and severe edema in the lower limbs in the setting of a chylous ascites related to two very rare causes of it: tumoral portal vein thrombosis and gastric adenocarcinoma.

Introduction

Chylous ascites, or chyloperitoneum, is an uncommon finding characterized by the presence of a milky-appearing peritoneal fluid, rich in triglycerides. Currently, the estimated incidence in clinical practice is about 1 per 20,000 admissions (Press et al. 1982). Indeed, the triglyceride levels in ascitic fluid are the hallmark in the diagnosis of chylous ascites.

The accumulation of chyle in the peritoneum appears when a disruption of the lymphatic system occur. The origin of this disruption may be secondary to traumatic injury, the exudation of lymph through retroperitoneal vessels or to obstruction or rupture of the peritoneal or retroperitoneal lymphatic glands. In this way, portal vein thrombosis is an unusual cause of chyloperitoneum. The etiology of portal vein thrombosis includes prothrombotic states and invasion or constriction by a malignant tumor.

We describe the case of an 81-year-old man presented with constitutional syndrome, abdominal distension and severe edema in the lower limbs in the setting of a chylous ascites related due to a tumoral portal vein thrombosis secondary to an advanced gastric adenocarcinoma.

Case Presentation

An 81-year-old man was admitted to our

Internal Medicine Department because of constitutional syndrome and a rapidly evolving hydropic decompensation. His past medical history included hypertension, COPD and atrial fibrillation. He had no surgical history. He reported a 50 mg / day alcohol consumption. His current medications were losartan, aclidinium bromide and acenocoumarol.

He referred appetite loss, postprandial fullness, asthenia and anorexia lasting eight months accompanied by progressive painless abdominal distension for the last three weeks with resting dyspnea and edema in the lower limbs in the last 72 hours.

At admission, his temperature was 37 ºC, his blood pressure was 135/80 mmHg, and his heart rate was 95 b.p.m. On physical examination were evident a grade 3 ascites and collateral abdominal circulation. The remainder of the physical examination was unremarkable. Routine blood tests revealed a total leukocyte count of 5.7 x $10^3/cm^3$, Normal Range (NR) 4.8 - 10.8 x 10^3; 68 % neutrophils, NR 42.0 - 75.0%; Hb 13.2 g/dL , NR 13-18; platelet count 259 x 10^3 / L , NR 150 – 450 x 10^3; Na^+ 143 mEq /L, NR 135-145; K^+ 3.9 mEq/L, NR 3-5; creatinine 0.8 mg/dL, glucose 116 mg/dL. Glutamyl transpeptidase (GGT), aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (AP) and bilirubin were normal (GGT 45 U/L , AST 34 U/L, ALT 31 U/L, AP 75 U/L , bilirubin 0.8 mg/dL). Albumin level was 3.7 g/dL (NR: 3.4 -5.4). Lactate dehydrogenase was 446 U/L (NR:105 - 333 U/L). Total cholesterol and triglycerides were 212 and 134 (NR: 150-200 and < 200), respectively. Serum amylase was 78 (NR: 23- 85 U/L).

A paracenthesis was performed obtaining four liters of milky-appearing peritoneal fluid (Figure 1). Biochemical analysis of the ascitic fluid detected a level of trigyceriddes of 240 mg/dL. The serum to ascites albumin gradient (SAAG) was greater than 1.1 g / dl. Adenosine deaminase activity (ADA) was normal. Cytologic studies of the peritoneal fluid were negative.

An abdominal computed tomography revealed portal thrombosis from the intrahepatic portal vein and a generalized thickening of the gastric wall (Figure 2) without signs of an evolved hepatopathy. The esophagogastroduodenoscopy revealed an ulcerated area of 20 x 10 cm at the greater curvature of the gastric body (Figure 3). The anatomopathological study was consistent with a poorly differentiated adenocarcinoma (G3).

Discussion

The lymphatic system is an important pathway where fluids, proteins, lipids and lipid soluble vitamins flow from the intestinal spaces to the vascular system [2]. Lymph and interstitial fluid usually have the same osmotic pressure, so, molecules enter into the lymphatic vessels by the influence of hydrostatic and oncotic pressures (Griniatsos et al, 2010). The mechanisms by which chylous ascites is produced are related to obstruction or disruption of the lymphatic ducts. As discussed below, this obstruction may be due to a benign or a malignant cause.

Four pathophysiological mechanisms have been proposed for its development. Briefly, a) Primary lymphatic fibrosis, originated by malignant obstruction of the lymphatic or chyle cistern, which generates a subserosa dilation with extravasation into the peritoneal cavity; b) Lymph exudation through the lymphatic retroperitoneal wall vessels, which form a fistula into the

peritoneal cavity, as in congenital lymphangiectasia and c) Dilation of retroperitoneal lymph vessels secondary to obstruction of the thoracic duct or chyle cistern as in heart failure and constrictive pericarditis and d) Acquired thoracic duct obstruction from trauma or surgery, causing direct leakage of chyle through a lymphoperitoneal fistula (Cárdenas et al, 2002).

Chylous ascitesis is characterized by the presence of a milky fluid with a triglyceride concentration that exceeds the serum concentration is greater than 110-200 mg/dL (Runyon et al, 1988) and often greater than 1000 mg/dL.

There are multiple causes of chylous ascites (Table 1). During the last decades, malignancies, mainly lymphomas, have been the first cause of chyloperitoneum. Gastric carcinoma is an uncommon cause of this entity. A review of literature using The PubMed database of the U.S. National Library of Medicine showed 19 reported cases of gastric carcinoma and chyloperitoneum between years 1964 y 2014.

Other interesting question in our patient to discuss is the presence of a portal vein thrombosis despite oral anticoagulation for his atrial fibrillation. Cancer is a highly thrombophilic entity which origins a high number of symptomatic or asymptomatic venous thromboembolic events (VTE). (Gary et al, 2014). Cancer patients have a 6–7-fold higher risk of venous thromboembolism (VTE) as compared with non-cancer patients (Prandoni et al, 2014). VTE is a predictor of poor survival in these patients. In our case, portal vein thrombosis is probably related to the state of hpercoagulability derivated of gastric cancer. In the setting of our patient`s constitutional syndrome, chronic portal vein thrombosis would have induced an obstruction to flow within the portal venous system leading to a portal hypertension state which was an aggregating factor to increase the chylous ascites.

The role of anticoagulation in patients with chronic tumoral PVT is not clear. The goal of anticoagulation in this cases is to prevent recurrent thrombosis as well as the thrombus extension, and promote recanalization, if possible. Recanalization of portal vein with anticoagulant drugs is more feasible in cases of acute than chronical thrombosis. (Engman et al, 2008). The response to Vitamin-K-Antagonists (VKA) seems to differ between patients with or without cancer.

Many works have found an improved efficacy against recurrent VTE for Low-molecular-weight heparin (LMWH), situation that confirms the fact that cancer patients are less responsive to VKA, So, LMWHs are the cornerstone of VTE treatment in oncologic patients. Some studies suggest that LMWHs may also have direct antitumor effects and improve survival rates (Hull et al., 2006). Notwithstanding, further clinical research is needed to confirmate this effect.

Direct oral anticoagulants (DOACs) are emerging alternatives for VTE treatment especially in cases of cancer-associated VTE (Wharin et al, 2014). DOACs can be administered with a fixed-dose orally, have a predictable drug effect, does not need routine laboratory monitoring and they have a very few drug or food interactions.

On the other hand, anticoagulation therapy is associated with higher bleeding complications in cancer patients than in non-cancer patients so the decision to start anticoagulation must be made on a case-by-case basis.

Table.1 Causes of chylous ascites

Congenital
Primary lynfatic hypoplasia
Yellow nail syndrome
Klippel Trénaunay syndrome
Primary lynfatic hyperplasia
Acquired
Malignancies
Lymphoma
Kaposi's sarcoma
Peritoneal carcinomatosis (ovary, stomach, colon)
Enfermedad de Hodgkin, leucemia, linfoma, mieloma multiple
Post-surgical
Abdominal aneurysm repair
Retroperitoneal node dissection
Catheter placement for peritoneal dialysis
Inferior vena cava resection
Laparoscopic nissen fundoplication
Liver transplantation
Duodenopancreatectomy
Radical nephrectomy
Inflammatory
Radiation
Pancreatitis
Constrictive pericarditits
Retroperitoneal fibrosis
Sarcoidosis
Celiac sprue
Whipple's disease
Retractile mesenteritis
Infectious
Tuberculosis
Filariasis
Mycobacterium avium intracellulare
Abdominal trauma
Hemodynamic
Portal hypertension
Right heart failure
Dilated cardiomyopathy
Nephrotic syndrome

Figure.1 Chylous ascites: milky-appearing peritoneal fluid

Figure.2 Thrombosis from the intrahepatic portal vein (black arrow) and a generalized thickening of the gastric wall (white arrow)

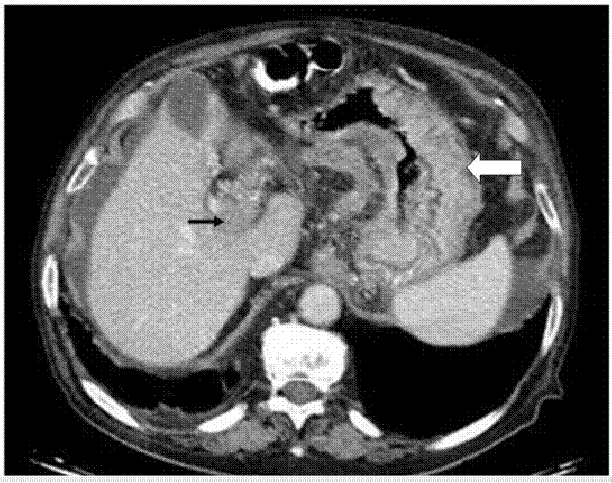
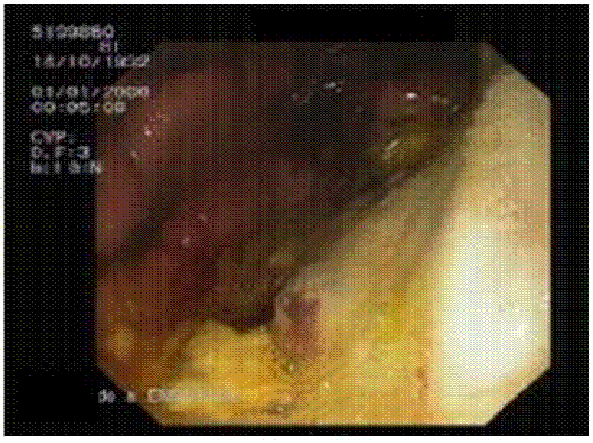


Figure.3 Ulcerated area of 20 x 10 cm at the greater curvature of the gastric body



Our patient was evaluated by the Oncology service, discarding the initiation of a chemotherapeutic treatment due to the advanced stage of the tumour, the age and the associated comorbidities. Only symptomatic relief measures were instaured, including multiple s drainage procedures and analgesic drugs. Acenocumarol treatment was discontinued. We started anticoagulation with enoxaparin, 80 mg subcutaneous bid. It is important to notice that the triglyceride concentration in peritoneal fluid started to decline since the initation of LMWH therapy as well as the edema of the lower limbs improved rapidely.

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