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Caso clínico

Chylous ascytes secondary to acute pancreatitis: a case report and review of literature

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Abstract

Chylous ascites is an uncommon finding which is due to the presence of thoracic or intestinal lymph in the abdominal cavity. It is usually caused by a chronic disruption of the lymphatic system. The present report is one of the rare cases in the literature of chylous ascites secondary to idiopathic acute pancreatitis, which showed a complete resolution with a combination of low fat enteral nutrition with MCT and somatostatin analogs.

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Key words: *Chylous ascites. Acute idiopathic pancreatitis. Medium chain triglycerides. Somastotin analogs.*

ASCITIS QUILOSA SECUNDARIA A PANCREATITIS: CASO CLÍNICO Y REVISIÓN DE LA BIBLIOGRAFÍA

Resumen

La ascitis quilosa es un hallazgo infrecuente producido por la presencia de linfa de origen torácico o intestinal en la cavidad abdominal. Normalmente es producido por la disfunción crónica del sistema linfático. El caso que presentamos es uno de los pocos casos descritos en la literatura de ascitis quilosa secundaria a una pancreatitis aguda idiopática, que se resolvió completamente con una combinacion de dieta enteral baja en grasas con triglicéridos de cadena media y análogos de somatostatina.

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Palabras clave: *Ascitis quilosa. Pancreatitis aguda idiopática. Triglicéridos de cadena media. Análogos de somatostatina.*

Introduction

Chylous ascites is an uncommon finding[1] which is due to the presence of thoracic or intestinal lymph in the abdominal cavity.[2] It is usually caused by a chronic disruption of the lymphatic system. The proposed pathophysiological mechanism includes the obstruction of the lymph flow through the dilated subserosal lymphatics into the peritoneal cavity which produces collagen deposition, fibrosis, and protein-losing enteropathy. Other mechanisms that have been involved are the exudation of lymph through the walls of congenital or acquired dilated retroperitoneal vessels into the abdominal cavity, and the obstruction from direct trauma of the thoracic duct[3]. Its diagnosis is based on the biochemical study of the ascitic fluid. The most important and extended diagnostic criterion is the presence of > 2.3 mM (> 200 mg/dl) of triglycerides in ascitic fluid.[1,2,4,5].

Acute and chronic pancreatitis are known causes of chylous ascites. By causing inflammation of the abdominal structures, with compression of the lymphatic vessels, lymph effuses into the abdominal cavity. Besides it can also produce pleural lymphatic effusion and consequent chylothorax.[6]

Eight cases of chylous ascites associated with acute pancreatitis have been reported so far. Three of them occurred in alcohol drinkers, two associated with enolic acute pancreatitis[7,8] and the other one in acute on chronic pancreatitis.[9] Three other cases took place in hyperlipidemia-associated acute pancreatitis, two during pregnancy,[10,11] and the other one in a familiar hyperlipidemia context.[12] The remaining two cases occurred

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in patients with gallbladder stone disease.[13,14] Here we report an uncommon case of insidious chylous ascites following a severe acute idiopathic pancreatitis, in a 68y old woman, which was resolved with somatostatin analogs and artificial nutritional support.

Case report

A 68y old woman came to the emergency room suffering from upper abdominal pain, nausea, and occasional vomiting which started several hours before. The patient had not any relevant medical history, she was not an alcohol drinker, nor had she suffered from pancreatitis before. At admission she had an arterial pressure of 112/62 mmHg, a heart rate of 80 beats per minute, and a temperature of 36º C. Physical examination revealed only an epigastric pain irradiated to both left and right hypochondria without peritoneal irritation.

The first blood test showed a serum amylase of 5,257 U/L, lypase of 8,392 U/L, leucocytes of 17,700 per μL, with transaminases, and other analytical parameters within normal ranges. Radiological images of thorax and abdomen did not show any pathological findings, and abdominal ultrasound showed a normal biliary duct diameter without gallstones, and a hypogenic, globulous, and increased in size pancreas. The pancreatic tail was not well defined in ultrasound, which suggested an incipient pancreatic collection.

The patient was diagnosed of acute pancreatitis, and admitted to the Gastroenterology ward, with intravenous fluid therapy, antibiotics and analgesic drugs. Four days later, an abdominal CT scan was performed. It revealed multiple mesenteric collections, a pancreatic cyst, and fluid in the abdominal cavity, all these findings compatible with an E Balthazar's degree pancreatitis. The following day the patient developed fever of 38º C, and progressive anemia with dyspnea. She moved to the Intensive Care Unit, where she received intravenous antibiotic therapy, blood transfusions, and total parenteral nutrition support. After six days in the Intensive Care Unit she improved, and moved again to the Gastroenterology ward with the following diagnoses: Systemic Inflammatory Response Syndrome (SIRS) possibly triggered by acute pancreatitis, with a severity index of 4.

One week later a study of the ascitic fluid was performed showing a pancreatic amylase > 900 U/L, which confirmed the pancreatic origin of the ascites. That day multiple abdominal drainages were placed in the abdominal collections, somatostatin analogs were started and a nasojejunal tube was placed in order to start enteral nutrition. The patient progressively improved as shown by her clinical and analytical parameters (amylase 44 U/L, lipase 24 U/L, leucocytes 6,000 per μL, Hb 9.1 g/dL). Also the abdominal collections and ascites were reduced and an oral diet was started. Several days later, the patient worsened again with fever of 39º C and abdominal pain. A CT scan was performed showing more abdominal collections and a greater amount of ascitic fluid (fig. 1 and 2).

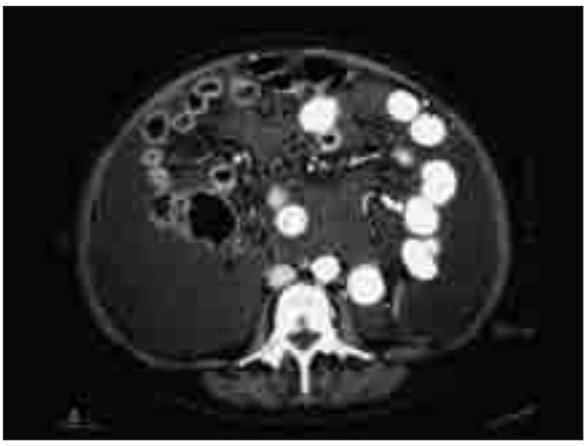

Fig. 1.—Contrast-enhanced venous phase CT scans with oral contrast. Axial image showing abundant abdominal ascites.

The patient moved to the General Surgery ward in order to drain abdominal collections. Four days later, a new ascitic fluid analysis showed > 4,000 mg/dL of triglycerides, > 200 mg/dL of cholesterol, no microbiologic findings, and pancreatic amylase in normal range. After the confirmation of chylous ascites, total parenteral nutrition through a Peripheral Inserted Central Catheter (PICC) and somatostatin analogs were started.

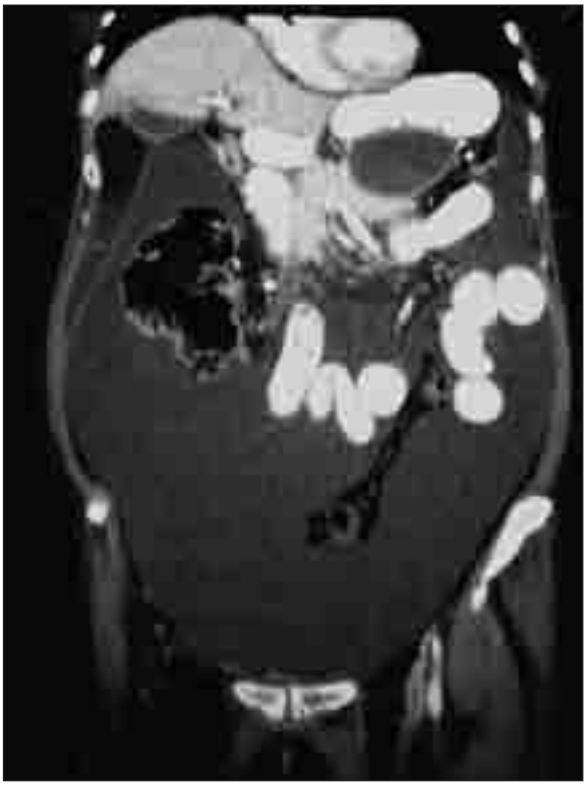

Fig. 2.—CT scan coronal image shows abundant abdominal ascites with septa (arrow) and fluid-fluid level of differential density due to layering of the chylous fluid.

Table I
Causes of chylous ascites

Malignancy	Lymphoma
	Linfangioleiomyomatosis
	Carcinoid tumor
	Kaposi's sarcoma
	Other malignancies
Congenital	Primary lymphatic hipoplasia
	Yellow nails syndrome
	Klippel-Trenaunay syndrome
	Primary bilateral lymphatic hyperplasia
	Primary intestinal lymphangiectasia
Surgical	Aorta aneurism reparation
	Lower cava resection
	Laparoscopic Nissen's procedure
	Peritoneal dialysis catheter
	Retroperitoneal lymph nodes resection
Inflamatory	Sarcoidosis
	Celiac disease
	Whipple's disease
	Retractile mesenterithis
	Retroperitoneal fibrosis
	Radiotherapy
	Pancreatitis
	Constrictive pericarditis
Traumatism	Battered child syndrome
	Abdominal contusion
Infectious	Tuberculosis
	Filariasis (Wuchereria bancrofti)
	Mycobacterium avium intracelulare
Cirrhosis	Enolic, biliary…
Miscelanea	Right Heart Failure
	Dilated myocardiopathy
	Nephrotic syndrome

Ten days later this catheter was replaced with a jugular central line because of local phlebitis. After twenty one days of total parenteral nutrition, the jugular line had to be removed due to candidemia, and appropriate therapy was started. Culture of the line tip was positive for *Candida parapsilosis*. At this time enteral nutrition support was progressively introduced. The chosen enteral nutrition formula (Monogen™, Nutricia Advanced Medical Nutrition-Danone Group) had a 25% of total calories as lipid, and 90% of these lipids were medium chained triglycerides (MCT).

Radiological tests showed a progressive decrease of the intra-abdominal collections and ascitic fluid. The patient was discharged after three months with a low fat diet and oral enteral nutrition (1,500 mL/day of Monogen™) and subsequently followed-up monthly. Two years later, with radiological evidence of no abdominal collections not any remnant ascitic fluid, she went on a free diet. Her weight increased 8.5 kg (from 45.5 kg to 54 kg) with an increase of body mass index (BMI) of 3.5 kg/m^2 (from 18.7 kg/m^2 to 22.2 kg/m^2) (table I). The patient is now, three years after hospital discharge, on a free diet with a normal body weight and no signs or symptoms of ascites.

Discussion

The incidence of chylous ascites in developed countries is approximately one case per 20,000 admissions, although large epidemiological studies are lacking.[1] Among known causes of chylous ascites there are many pathological processes such as cirrhosis, infections, malignancy, congenital defects, traumatism, inflammatory processes, nephropathies and cardiopathies (table II). Two-thirds of all chylous ascites present in developed countries as a consequence of abdominal malignancy[1,15] and cirrhosis.[16-19] The incidence has increased in the past years due to longer survival of oncologic patients. In contrast infectious etiologies such as tuberculosis[20] or filariasis,[21] are responsible for the majority of the cases in developing countries.

After extensive medical database search on Medline and Embase, only eight cases of chylous ascites secondary to acute pancreatitis have been reported in english literature before, with different etiologies (table III). All these cases occurred in the context of alcohol abuse, gallbladder stone disease, or lipids disorders.

Treatment of the underlying cause is a cornerstone in this entity whenever possible.[22-24] The resolution of chylous ascites usually takes several weeks,[25,26] but in some cases like ours, it may take months, or even some years after resolution of the underlying cause. In our case, we found no specific cause for the acute pancreatitis, so it was regarded as idiopathic. Therefore, the development and resolution of chylous ascites was more insidious than in previous reported cases, and the time between pancreatitis and chylous ascites onset was also longer.[7-14] Our case shows for the first time a case of idiopathic acute pancreatitis-associated chylous ascites.

When ascites persists after the resolution of the underlying cause, it is recommended a high protein and low lipid diet, the latter in the form of medium chain triglycerides (MCT). This type of triglycerides is directly absorbed from the bowel to the portal circulation, without passing through the lymph vessels, and it reduces the production and flow of lymph. On the other hand, long chain triglycerides in diet must be avoided, as these are converted into free fatty acids and monoglycerides which are transported through the lymphatic system.[22,23,25] There are some enteral nutrition products with a high percentage of MCT. We chose one which provides only a 25% of total fat,

Table II
Cases of chylous ascites secondary to acute pancreatitis

Summary of case report	*Authors*
A case of acute chylous peritonitis mimicking acute appendicitis in a 38y old man with acute on chronic alcoholic pancreatitis, which resolved in 4 days.	Smith EK et al.[9]
A case of chylous ascites in a 50y old man 4 weeks after an acute alcoholic pancreatitis, which resolved in two months with a combination of octreotide and total parenteral nutrition.	Al-Ghamdi MY et al.[7]
A case of chylous ascites secondary to acute hiperlipidemic pancreatitis in a 54y old man with family history of hyperlipidemia, managed with gemfibrozil and low fat diet which resolved in less than 3 weeks.	Khan FY et al.[12]
A case of a 28y old woman with hypertriglyceridemia, acute pancreatitis, and chylous ascites during the third trimester of pregnancy. It resolved in 43 days treated with cesarean, antibiotics and parenteral nutrition.	Chuang SC et al.[10]
A case of chylous ascites secondary to acute pancreatitis during the third trimester of pregnancy in a 24y old woman. It was managed by cesarean, section, antibiotics and parenteral nutrition for 2 months.	Liu CJ et al.[11]
A case of chylous ascites secondary to acute biliary pancreatitis in a 68y old woman, treated by cholecystectomy, IV hydratation and antibiotics, which resolved in one week.	Ben-Ami H et al.[13]
A case of chylous ascites associated with acute pancreatitis secondary to gallbladder stone disease in a 66y old woman undergoing continuous ambulatory peritoneal dialysis. It resolved in 2 months with cholecystectomy, parenteral nutrition and antibiotics.	Pérez Fontán M et al.[14]
A case of chylothorax and chyloperitoneum associated with alcoholic acute pancreatitis in a 43y old man which resolved in 12 days by chest and peritoneal drainage.	Goldfarb JP et al.[8]

with 90% as MCT, and also additional supplements of vitamins, minerals and oligoelements. This composition is specially designed for the treatment of chylothorax, chylous ascites, and other processes with impaired lymph circulation.

Somatostatin analogs have been successfully used in different forms of chylous ascites. Somatostatin is known to inhibit a variety of gastrointestinal processes, hormones and secretions. Although they have been used successfully in the treatment of chylous ascites, the mechanism of action in these disorders remains unclear, and need further research.[8,25,27-30] Patients who do not improve with enteral nutrition and somatostatin analogs can be managed with home parenteral nutrition with variable results.[26,31-33]

The present report is one of the rare cases in the literature of chylous ascites seco*ndary to idiopathic acute pancreatitis, which showed a complete resolution with a combination of low fat enteral nutrition with MCT and somatostatin analogs.

Table III
Serum proteins levels in relation to clinical evolution and nutritional support

Time	*Albumin (g/dl)*	*Transferrin (mg/dl)*	*Prealbumin (mg/dl)*	*RBP (mg/dl)*	*Nutritional support and clinical events*
In-hospital					
1 week	3.05	110	16.5	2.04	Intensive care unit, parenteral nutrition
2 week	2.58	125	7.94	< 1.05	Enteral nutrition then oral diet
5 week	1.69	93.1	8.72	1.14	Octreotide analogs started
6 week	2.03	117	8.53	1.09	Clinical improvement
8 week	1.42	76.1	6.25	< 1.05	Clinical worsening, parenteral nutrition
11 week	2.12	138	16.2	1.62	Surgery ward, Monogen™
17 week	2.69	163	16.9	2.69	Hospital discharge, low fat diet, Monogen™
Home					
2 month	3.94	180	17.5	2.26	Monogen™ withdrawal
3 month	4.64	222	22.6	2.79	Low fat diet
9 month	4.30	182	19.4	2.43	Total resolution of chylous ascites
21 month	3.87	202	18.8	2.57	Free diet
26 month	4.44	186	20.3	2.55	

Laboratory normal ranges: Albumin: 3.3-5.2 g/dl; Transferrin: 200-360 mg/dl; Prealbumin: 20-40 mg/dl; RBP: Retinol binding protein 3-6 mg/dl.

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