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Chylous ascites secondary to pancreatic pseudocyst: A case report

Ascitis quilosa secundaria a seudoquiste pancreático: reporte de un caso

The abnormal intraperitoneal accumulation of lymph resulting from extravasation into the abdominal cavity is defined as ''chylous ascites'' (CA) or ''chyloperitoneum''.[1] It is produced after the disruption or obstruction of thoracic or abdominal lymphatic circulation. Considered a rare entity, it has an incidence of approximately one case per 20,000 patients and is related to malignant (lymphomas), congenital, and inflammatory (acute pancreatitis) diseases.

A 61-year-old man had presented with severe acute pancreatitis complicated with a pancreatic pseudocyst one year earlier. He sought medical attention due to nausea and vomiting 5-6 times per day, increase in abdominal perimeter, and weight loss of 12 kg, of 4-month progression. He stated he had no dyspnea, orthopnea, or previous surgeries. Physical examination revealed poor general status; the patient was wasting (weight: 50 kg, height: 1.65 m, BMI: 18.3), dehydrated, with mild pallor, a nonpainful distended abdomen, a positive fluid wave, and no hepatic stigmata.

Laboratory tests showed mild anemia, hypoalbuminemia, and no alterations in blood chemistry, electrolytes, or liver function tests. In addition, fecal elastase was measured and found to be low (130 μg/g), and so treatment with pancreatic enzymes and a proton pump inhibitor was started in the context of a patient with exocrine pancreatic insufficiency.

Paracentesis revealed a whitish, milky fluid (Fig. 1) and its analysis (Table 1) showed a hyperintense serum ascites albumin gradient, with elevated proteins and triglycerides of more than 200 mg/dL, confirming the diagnosis of chylous ascites. Elastography was not carried out due to the presence of ascites, and the echocardiogram showed no alterations.

Magnetic resonance imaging of the abdomen identified a 95 x 110 x 95 mm collection (volume: 496 mL), dependent on the body and tail of the pancreas, and abundant free fluid. The liver and portal venous system appeared normal, and there was no adenomegaly (Fig. 2).

Parenteral nutrition was started with a special regimen and enteral tube feeding with a low-fat diet and medium-chain triglyceride supplementation. Octreotide 100 mcg was administered subcutaneously every 8 h, and the milky peritoneal fluid became clearer, turning to citrine yellow.

The patient underwent percutaneous drainage of the pancreatic pseudocyst due to the lack of access to endoscopic ultrasound (EUS) at our hospital. A total of 500 mL of the pancreatic collection was extracted, leaving catheters in the pancreatic bed and peritoneal cavity, with progressively descending flows. The patient did not undergo surgery. His evolution was favorable, with improvement in tolerating oral intake and weight gain (a current IBM of 22), as well as tomographic evidence of reduced volume of the pancreatic collection and free fluid (Fig. 3).

The diagnosis of CA was made by consensus, with a concentration of triglycerides > 200 mg/dL in the ascitic fluid.[2] Its origin is more frequently associated with portal hypertension, endothelial involvement, and rupture of dilated lymph channels. In causes not associated with portal hypertension (congenital, inflammatory, infectious, neoplastic, postoperative, and traumatic), lymphatic fluid is released by the dilated retroperitoneal vessels into the abdominal cavity through a fistula with the peritoneum.[2,3]

Inflammatory causes are reported to be associated with acute and chronic pancreatitis. Two mechanisms are reported in both: filtration through the lymph vessels damaged by pancreatic enzymes or lymph exudation due to flow obstruction secondary to inflammatory changes in the retroperitoneum adjacent to the pancreas. This

Figure 1 Milky-appearing ascitic fluid.

Table 1 Ascitic fluid study.

Ascitic fluid	Value	Ascitic fluid	Value
Leukocytes	300	SAAG	1.2
PMNs	5%	Amylase	252
Red blood cells	200	Triglycerides	900
Glucose	111	Culture	Negative
LDH	130	ADA	7.7
Proteins	3.4	PAP	Negative
Albumin	1.4	Cell block	Negative

ADA: adenosine deaminase; LDH: lactate dehydrogenase; PAP: prostate acid phosphatase; PMN: polymorphonuclear leukocytes.

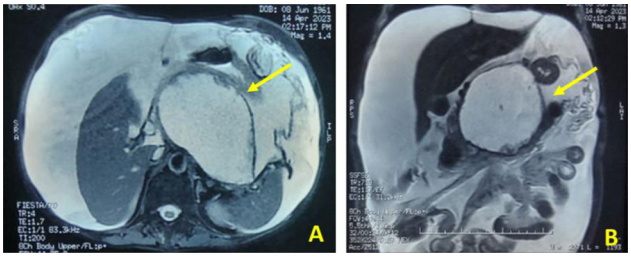

Figure 2 A–B: Capsular hyperintense image, with partial septa in the body and tail of the pancreas, associated with abundant free fluid.

usually occurs after days or weeks of presenting with pancreatitis.[4,5]

CA management depends on the etiology. Dietary measures include a high-protein low-fat (triglycerides) diet to prevent conversion into monoglycerides and free fatty acids, reducing lymph flow.[2,6] For nonresponders, bowel rest is suggested to reduce the lymph flow and establish total parenteral nutrition.[7]

Concerning drugs, orlistat prevents the conversion of triglycerides into fatty acids in the intestinal lumen, whereas somatostatin and octreotide aid in inhibiting lymph fluid excretion.[3,8]

The invasive procedures include large-volume paracentesis, transjugular intrahepatic portosystemic shunt (TIPS) placement, and peritoneovenous shunt (PVS) placement. Paracentesis is carried out using pharmacologic and drug measures in patients with symptomatic ascites. PVS is an option for cases that are refractory to medical treatment, in which surgery is not indicated, albeit it has potential complications.[9,10]

In the case reported herein, multiple large-volume paracenteses were carried out, with partial symptom alleviation. Due to the recurrence of the ascites, percutaneous drainage of the pancreatic pseudocyst was performed. After this, and in addition to octreotide use and the dietary measures put in place, the patient progressed favorably, with significant improvement in oral intake, abdominal perimeter reduction, ascites clarification, and weight gain.

Identifying CA is important for providing specific and definitive treatment, including dietary, nutritional, and pharmacologic management, and correcting the underlying cause.

Ethical considerations

The authors declare that no experiments on humans were conducted for this study. We employed the patient data collection format of our work center, maintaining patient anonymity and obtaining a signed statement of informed consent.

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Conflict of interest

The authors declare that there is no conflict of interest.

Author contribution

The authors participated in the concept and design of the article, as well as the drafting and approval of the final version to be published.

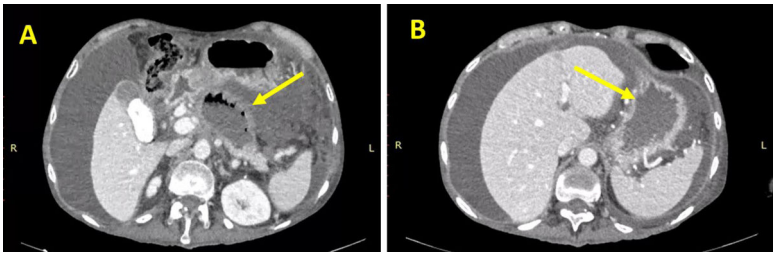

Figure 3 Multi-slice spiral computed tomography of the abdomen, with contrast. A) Pancreatic collection showing less volume than in previous images. B) Gastric chamber with adequate distension.

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Cricopharyngeal achalasia: A rare cause of dysphagia in infancy

Acalasia cricofaríngea: una causa infrecuente de disfagia en la infancia

Cricopharyngeal achalasia (CA) is a motor disorder caused by the lack of relaxation of the cricopharyngeus muscle during swallowing.[1] It is rare in pediatrics and its etiology is multifactorial, related to intramural nerve plexus alterations, central nervous system disorders, and infections.[2] It can present in the first months of life but diagnosis tends to be delayed due to nonspecific symptoms and a low level of clinical suspicion. It manifests as dysphagia, regurgitation, nasopharyngeal reflux, cough, sialorrhea, recurrent pneumonia, bronchoaspiration, and failure to thrive.[3,4]

A male patient seen since he was 7 months of age at the pediatric gastroenterology service, with a history of upper respiratory symptoms since the first day, predominantly nocturnal nasopharyngeal reflux, recurrent broncho-obstructive episodes, episodes of apnea, cough during breastfeeding, sialorrhea, and poor secretion management. With complementary feeding, he presented with choking, the passage of food content through the nose, vomiting, and occasional regurgitation. At the first consultation, the patient's anthropometric measurements were weight: 7.65 kg (−0.87 SD), height: 70 cm (0.08 SD), and weight-for-height: (−1.18 SD). Physical examination revealed no alterations. Due to clinical oropharyngeal dysphagia, an esophagogastroduodenoscopy (EGD) was performed that revealed a 60% reduction of the cervical esophageal lumen between C3 and C4 (Fig. 1), suggesting a cricopharyngeal spasm vs a vascular ring. In the EGD, a 60% reduction of the cricopharyngeal lumen was observed that impeded the passage of the endoscope. At 8 months of life, the first session of endoscopic dilation was carried out, improving the dysphagia and respiratory symptoms. Cricopharyngeal achalasia was suspected. High-resolution esophageal manometry, with a solid-state probe, using a 4.2 mm external probe and 36 sensors, showed an abnormal pressurization pattern in the cricopharyngeus muscle consistent with CA (Fig. 2). The patient was symptom-free up to 28 months of life, after which he presented with recurrence of the dysphagia. At present, the patient has required two endoscopic dilations, at 30 and 46 months. In the first dilation, an 8 mm balloon was employed and progressively advanced to 10 mm, 12 mm, and 15 mm. The patient currently has no dysphagia at 56 months of life, no new bronco-obstructive events, and weight gain is adequate.

Organic diseases, such as esophageal stricture, tracheoesophageal fistula, or tracheoesophageal cleft, must be ruled out in cases of dysphagia in infants.[5] CA is included within the differential diagnosis and a high level of suspicion is required. Its delayed diagnosis can lead to serious complications, such as bronchoaspiration, recurrent pneumonia, dehydration, malnutrition, and even death.[4,6]