

Upper gastrointestinal lesions secondary to Crohn's disease become resistant to treatment in up to 21% of the cases, have complete clinical and endoscopic remission in 57%, and present at least one relapse of symptoms and lesions in 21%.<sup>10</sup> In our case, medical treatment was established after the procedure, resulting in lesion and symptom remission.

And finally, we can conclude that oropharyngeal and esophageal involvement in Crohn's disease is rare and represents a diagnostic challenge due to the scant specificity of the clinical manifestations (aphthous ulcers), as well as the histologic findings (absence of granulomas), and also to the limited value of endoscopy and biopsy in that location, leading to late diagnosis with the consequent therapeutic and prognostic implications.

In cases such as ours, with oral lesions that do not respond to symptomatic treatment, with no accurate diagnosis, and with a biopsy excluding malignancy, the possibility of inflammatory bowel disease should be considered. Endoscopic and colonoscopic studies directed at discovering associated lesions primarily in the terminal ileum should be performed to make a correct diagnosis and begin early medical treatment. In this manner, remission is achieved and the progression into more advanced forms of the disease or the appearance of complications that require surgical treatment in a clinical situation of greater morbidity and mortality for the patient are prevented.

### Financial disclosure

No financial support was received in relation to this article.

### Conflict of interest

The authors declare that there is no conflict of interest.

### References

1. Sakuraba A, Iwao Y, Matsuoka K, et al. Endoscopic and pathologic changes of the upper gastrointestinal tract in Crohn's disease. *Biomed Res Int.* 2014;2014:610767.

2. Ohta M, Konno H, Kamiya K, et al. Crohn's disease of the esophagus: Report of a case. *Surg Today.* 2000;30:262-7.
3. Quezada S, Turner P, Alexiev B, et al. Severe refractory orofacial Crohn's disease: Report of a case. *Dig Dis Sci.* 2009;54:2290-5.
4. Wang W, Ni Y, Ke C, et al. Isolated Crohn's disease of the esophagus with esophago-mediastinal fistula formation. *World J Surg Oncol.* 2012;10:208.
5. Ferrer-Bradley I, Hinojosa del Val J. Definiciones, manifestaciones clínicas y diagnóstico de la enfermedad de Crohn. *Medicine.* 2012;11:257-65.
6. Lazarev M, Huang C, Bitton A, et al. Relationship between proximal Crohn's disease location and disease behavior and surgery: A cross-sectional study of the IBD Genetics Consortium. *Am J Gastroenterol.* 2013;108:106-12.
7. Mejía-Rivas M, Yamamoto-Furusho J, Remes-Troche J, Uscanga L. Enfermedad de Crohn del tubo digestivo proximal. Una presentación rara de una enfermedad poco común en México. *Rev Gastroenterol Mex.* 2009;74:110-4.
8. Mohamed R, Schultz R, Fedorak R. Oropharyngeal Crohn's disease. *Clin Exp Gastroenterol.* 2008;1:15-8.
9. Kim-Doan K, Alexis R, Schwarz S, Nguyen KD, Alexis R, Schwarz SM. Oropharyngeal and proximal esophageal involvement during adalimumab treatment of Crohn disease. *J Pediatr Gastroenterol Nutr.* 2010;50:225-6.
10. Feagans J, Victor D, Joshi V. Crohn disease of the esophagus: A review of the literature. *South Med J.* 2008;101:927-30.

M. Loreto-Brand<sup>a,\*</sup>, A. Fernández-Pérez<sup>a</sup>,  
C. Celeiro-Muñoz<sup>b</sup>, A. Álvarez-Castro<sup>c</sup>,  
M. Bustamante-Montalvo<sup>a</sup>

<sup>a</sup> *General and Gastrointestinal Surgery, Hospital Clínico Universitario de Santiago, Santiago de Compostela, La Coruña, Spain*

<sup>b</sup> *Pathologic Anatomy Service, Hospital Clínico Universitario de Santiago, Santiago de Compostela, La Coruña, Spain*

<sup>c</sup> *Gastrointestinal Endoscopy Service, Hospital Clínico Universitario de Santiago, Santiago de Compostela, La Coruña, Spain*

\* Corresponding author.

E-mail address: [m3lbita@hotmail.com](mailto:m3lbita@hotmail.com) (M. Loreto-Brand).

## Chylous ascites secondary to cirrhosis of the liver: A case report<sup>☆</sup>



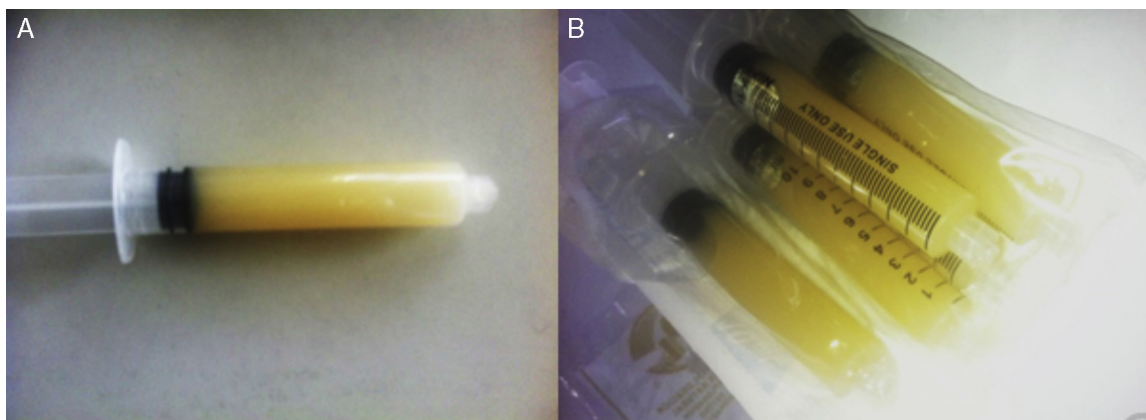
### Ascitis quilosa secundaria a cirrosis hepática. Reporte de un caso

Chylous ascites is a rare cause of ascites resulting from the accumulation of lymph in the abdominal cavity. It has different etiologies that interrupt the lymphatic flow. Diagnosis

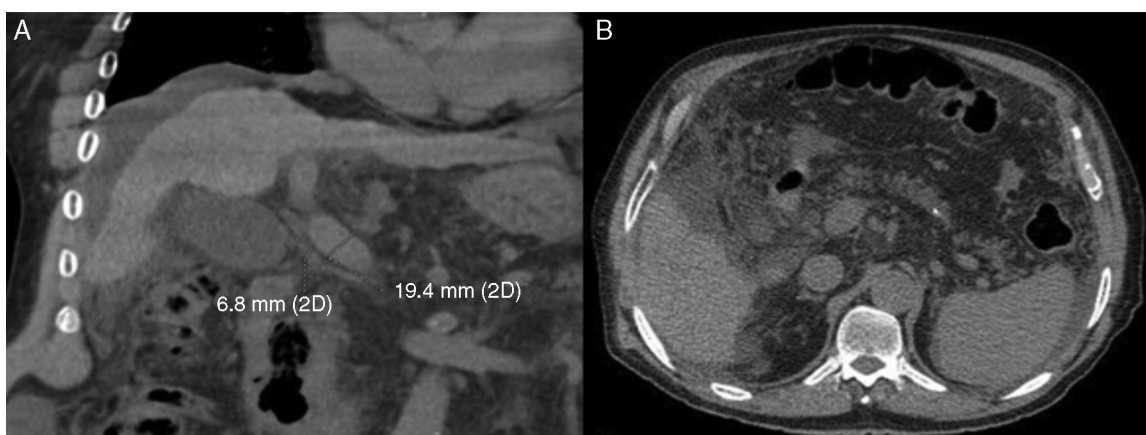
is made when a milky or turbid fluid is observed, with a triglyceride concentration  $\geq 110$  mg/dL.<sup>1,2</sup> The diagnostic criterion for some authors is a serum triglyceride to fluid ratio  $> 1.0$ , a cholesterol ratio  $< 1.0$ , a leukocyte count  $\geq 300$  cells/mm<sup>3</sup>, and/or a predominance of lymphocytes with negative culture and cytology.<sup>3</sup> Its incidence varies from 1 in 20,000 to 1 in 187,000 referral hospital admissions.<sup>2,4</sup> Its causes are varied and cirrhosis is responsible for 0.5% of cases.<sup>5</sup>

We present herein the case of an 84-year-old man with a past history of diabetes and high blood pressure. Five years prior he was diagnosed with cirrhosis of the liver due to alcohol. His current illness began 5 months before his hospital admission, characterized by lower limb edema. During the last 2 weeks he presented with a progressively increasing abdominal perimeter that resulted in dyspnea, and was the

<sup>☆</sup> Please cite this article as: Piña-Pedraza JP, Álvarez-Avalos L, Vargas-Espinosa JM, Salcedo-Gómez A, Carranza-Madriral J. Ascitis quilosa secundaria a cirrosis hepática. Reporte de un caso. *Revista de Gastroenterología de México.* 2015;80:285-287.



**Figure 1** A) Ascitic fluid with a turbid aspect, obtained from the first paracentesis. B) Ascitic fluid that is slightly less turbid, obtained from the second paracentesis.



**Figure 2** A) Abdominal tomography scan showing: free fluid, small liver, vena porta with a 19.4 mm diameter, and no thrombi. B) A normal pancreas and no retroperitoneal adenomegalies.

reason he sought medical attention. He had not consumed alcohol for the last 4 years and had not experienced abdominal pain. Physical examination revealed muscle atrophy with no jugular plethora. He had rhythmic heart sounds and there were no pleuropulmonary alterations. The patient's abdomen was prominent with water dullness, with no visceromegaly. Lower limb edema reached the thighs. Upon his admission, paracentesis was carried out, obtaining a milky fluid (Fig. 1).

Laboratory work-up upon admission showed: hemoglobin 15.2 g/dl, platelets 133,000, leukocytes 5,700 with 80% neutrophils, 14% lymphocytes, and 6% monocytes, glucose 379 mg/dl, creatinine 0.86 mg/dl, BUN 21 mg/dl, urea 44.9 mg/dl, total serum cholesterol 151 mg/dl, serum triglycerides 111 mg/dl, amylase 42 IU/l, and lipase 12 IU/l. LFT with AST 41 U/l, ALT 21 U/l, alkaline phosphatase 116 U/l, albumin 2.8 g/dl, and globulins 2.9 g/dl. Alpha-fetoprotein 3.65  $\mu$ g/l (normal value: 0-5), carcinoembryonic antigen 4.4  $\mu$ g/L (normal value: 0-3), Ca 19-9 of 23 IU/ml (normal value: 0-37), and adenosine deaminase 7.6 IU/l (normal value: 0-6.7). Ascitic fluid cytochemistry with a pH of 6.5, cells 768 with 95% lymphocytes, glucose 225 mg/dl, proteins 1,264 mg/dl, and triglycerides 305 mg/dl. Ascitic

fluid cytology was negative for neoplastic cells. Blood culture and ascitic fluid culture were negative.

Ultrasound imaging revealed a small liver with irregular edges, vena porta with a 13 mm diameter, and abundant ascitic fluid. Tomography scan identified abundant ascitic fluid, a small and irregular liver, vena porta with a 19.4 mm diameter with no thrombi, normal pancreas, and no adenomegalies (Fig. 2).

Endoscopy found large esophageal varices in the distal two-thirds, slight hypertensive portal gastropathy changes in the fundus and body and erosive gastropathy in the antrum. Colonoscopy identified telangiectasia, diverticular disease of the left colon, and internal hemorrhoids.

The patient was treated with a normal protein and low-fat diet, paracentesis with the administration of albumin, and later with diuretics. At present his response is unsatisfactory, given that moderate lower limb edema persists, as well as the ascites, requiring repeat paracentesis.

There are multiple causes of chylous ascites, and the most frequent are malignant neoplasias, especially lymphoma. Others include breast and pancreatic neoplasia. The condition can have an inflammatory disease origin, such as pancreatitis, or a traumatic origin, such as constrictive

pericarditis, observed after abdominal surgery or blunt trauma. These causes were satisfactorily ruled out in our patient, and only liver cirrhosis was documented as the cause of chylous ascites, which is seen in 0.5-1% of cases. Due to the poor response in our patient, other measures will be carried out, such as middle-chain triglyceride or octreotide use.<sup>6,7</sup> Another therapeutic option is the administration of orlistat, which has been reported to reduce the quantity of triglycerides in the ascitic fluid in patients with cirrhosis.<sup>8</sup>

### Financial disclosure

No financial support was received in relation to this article.

### Conflict of interest

The authors declare that there is no conflict of interest.

### References

1. Lazarus DL, Al-Busafi SA, Hilzenrat N. Chylous ascitis secondary to giant liver hemangioma. *Case Rep Gastroenterol.* 2012;6:478–83.
2. Almakdisi T, Massoud S, Makdisi G. Lymphomas and chylous ascitis: Review of the literature. *Oncologist.* 2005;10:632–5.
3. Yilmaz M, Akbulut S, Isik B, et al. Chylous ascitis after liver transplantation: Incidence and risk factors. *Liver Transpl.* 2012;18:1046–52.
4. Steinemenn DC, Dindo D, Clavien PA, et al. Atraumatic chylous ascitis: Systematic review on symptoms and causes. *J Am Coll Surg.* 2011;212:899–905.
5. Rector WG. Spontaneous chylous ascites of cirrhosis. *J Clin Gastroenterol.* 1984;6:369–72.
6. Huang Q, Jiang ZW, Jiang J, et al. Chylous ascites: Treated with total parenteral nutrition and somatostatin. *World J Gastroenterol.* 2004;10:2588–91.
7. Leong RW, House AK, Jeffrey GP. Chylous ascites caused by portal vein thrombosis treated with octreotide. *J Gastroenterol Hepatol.* 2003;18:1211–3.
8. Chen J, Lin RK, Hassanein T. Use of orlistat (xenical) to treat chylous ascites. *J Clin Gastroenterol.* 2005;39:831.

J.P. Piña-Pedraza<sup>a</sup>, L. Álvarez-Avalos<sup>a,b,\*</sup>,  
J.M. Vargas-Espinosa<sup>a</sup>, A. Salcedo-Gómez<sup>a</sup>,  
J. Carranza-Madrigal<sup>a</sup>

<sup>a</sup> *Internal Medicine Service, Hospital General  
"Dr. Miguel Silva" Secretaría de Salud, Morelia,  
Michoacán, Mexico*

<sup>b</sup> *Universidad Michoacana de San Nicolás de Hidalgo,  
Morelia, Michoacán, Mexico*

\* Corresponding author. Diego José Abad 193 – 3 Colonia Centro, C.P. 58000 Morelia, Michoacán, México.  
Tel.: +443 313 33 35.  
E-mail address: [drlaa@prodigy.net.mx](mailto:drlaa@prodigy.net.mx) (L. Álvarez-Avalos).

## Multiple scalp metastases as a first manifestation of pancreatic adenocarcinoma<sup>☆</sup>



### Metástasis múltiples en cuero cabelludo como primera manifestación de adenocarcinoma de páncreas

An 81-year-old woman had a past history of well-controlled high blood pressure and essential tremor treated with propranolol. She consulted her family physician after noticing the appearance of 8-10 painful lesions on her scalp over the last 3 weeks. The lesions (fig. 1) consisted of firm nodules, adhered to deep layers, between 1 and 2 cm in size, and the majority had an ulcerated surface covered with small blood clots. She was referred to the dermatology service for consultation. Two weeks later, before said consultation, she presented with marked mucocutaneous jaundice, choloria, and acholia, for which she came to the emergency department of our center and was admitted to complete her evaluation. A cutaneous biopsy was ordered from the dermatology service. Blood test results were bilirubin 10.2 mg/dl, AST 111 IU/ml, ALT 227 IU/ml, GGT 3300 IU/ml,

and CA-19-9 436 IU/ml (normal limit < 37 IU/ml). An abdominal CT scan showed a mass located in the uncinate process of the pancreas that conditioned dilation of the common bile duct and duct of Wirsung. Peritoneal, pulmonary, and paravertebral muscle lesions suggestive of metastasis were also observed. Due to these findings, endoscopic ultrasound fine needle aspiration of the pancreatic mass was performed. The scalp biopsy (fig. 2) revealed the presence of cutaneous metastases with morphologic and immunohistochemical characteristics consistent with a pancreatic origin (positive for cytokeratins 7 and 19 and negative for



Figure 1 Scalp nodule.

<sup>☆</sup> Please cite this article as: Tavío-Hernández E, Cañete-Ruiz Á, Moreno C, Urech M, Cano-Ruiz A. Metástasis múltiples en cuero cabelludo como primera manifestación de adenocarcinoma de páncreas. *Revista de Gastroenterología de México.* 2015;80:287–289.