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CASE REPORT | LIVER

Successful Treatment of Refractory Chylous Ascites With Octreotide in a Patient With Decompensated Cirrhosis

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ABSTRACT

Chylous ascites is a rare manifestation of decompensated cirrhosis that is associated with increased short-term mortality. Exclusion of other etiologies must be performed to allow for appropriate management, which itself can be a challenge in the setting of decompensated cirrhosis. We report a case of chylous ascites in a patient with decompensated cirrhosis that was successfully managed with octreotide before liver transplantation.

KEYWORDS: Chylous ascites; cirrhosis; portal hypertension; octreotide

INTRODUCTION

Chylous ascites is a rare manifestation of decompensated cirrhosis that accounts for <1% of cirrhosis-related ascites and is associated with increased short-term mortality.[1] There are other secondary etiologies, such as postsurgical, radiation-induced, infectious, autoimmune, and malignant, so new-onset chylous ascites warrants further diagnostic workup.[2] In addition, there is no consensus for optimal treatment.[3] This diagnostic and therapeutic ambiguity leads to difficulties in management of chylous ascites and may delay definitive treatment, such as liver transplantation (LT). We describe a case of a patient with cirrhosis and refractory chylous ascites of initially unclear etiology who was successfully treated with octreotide before LT.

CASE REPORT

A 63-year-old man with a history of metabolic dysfunction-associated steatohepatitis cirrhosis complicated by esophageal varices, ascites, and hepatocellular carcinoma (HCC) status-post transarterial radioembolization (TARE) was admitted to the hospital for management of diuretic-resistant ascites requiring serial large-volume paracentesis (LVP).

He was diagnosed with cirrhosis 7 years prior to admission (PTA) and remained well-compensated until 1-year PTA, when he was found to have a 2.5-cm HCC. The HCC was treated with a segmental TARE as a bridge to LT, which was complicated by the development of ascites that was initially managed with diuretics. During the LT evaluation, a coronary angiogram demonstrated severe multivessel disease, and an echocardiogram showed normal biventricular function, so a coronary artery bypass graft was to be performed concomitantly at the time of LT. While on the LT waitlist, he had recurrent HCC requiring transarterial chemoembolization and progressive portal hypertension with a variceal bleed requiring band ligation and worsening ascites refractory to low sodium diet and diuretic therapy with furosemide and spironolactone, which were limited because of hyponatremia and hyperkalemia, respectively. He required increasingly frequent LVP, prompting the current admission.

On admission, an LVP was performed, and the fluid had a "milky" fluid appearance. The fluid analysis showed a high-serum ascites albumin gradient (3.2), low protein (<2.0 g/dL), 15.2 polymorphonuclear leukocyte/μL, and markedly elevated triglycerides (698 mg/dL), consistent with chylous ascites and portal hypertension. Repeat LVP corroborated the diagnosis of primary chylous ascites with triglyceride 494 mg/dL. Ascitic bacterial, fungal, and acid-fast cultures had no growth, and cytology was benign. Cross-sectional

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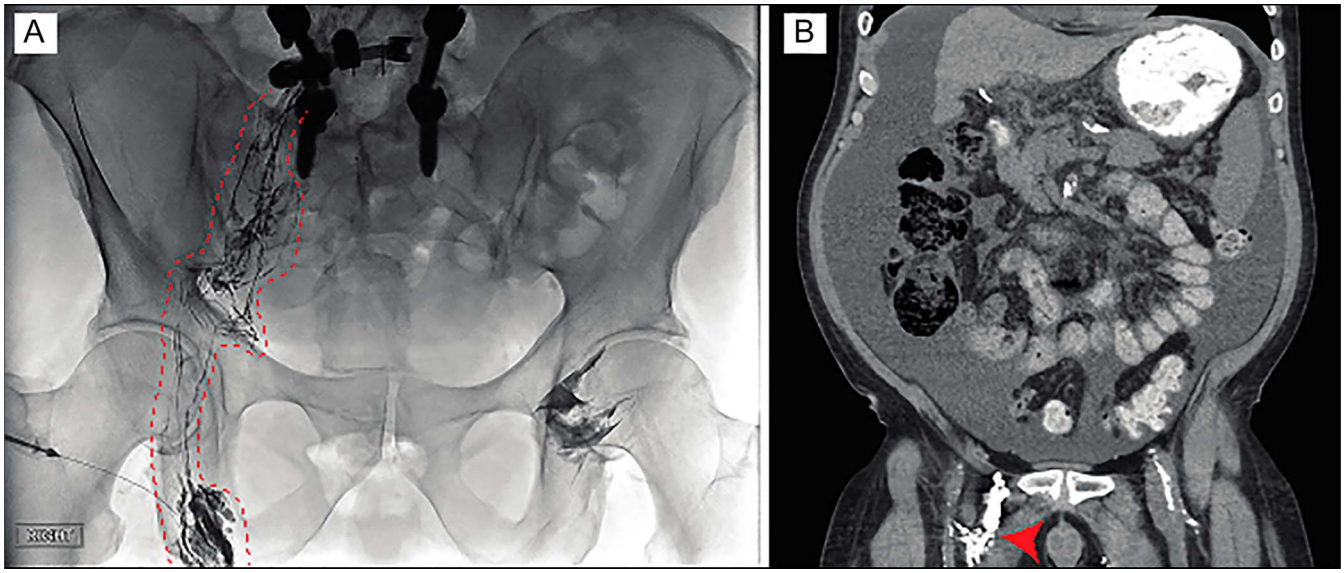

Figure 1. (A) The patient underwent a lymphangiogram in which lipiodol was injected into the inguinal lymphatics. The flow of the lipiodol is outlined in dotted red lines. No immediate extravasation was detected. (B) Approximately 24 hours after the lymphangiogram, our patient underwent a delayed computed tomography scan. Again, no extravasation of lipiodol was seen. Red arrowheads depict lipiodol within the inguinal lymphatics. Additional white areas are vascular calcifications.

imaging demonstrated stable recurrent HCC without evidence of progression, local invasion, or distant metastasis. Ultimately, a lymphangiogram was performed that did not demonstrate contrast extravasation on immediate fluoroscopic images (Figure 1A) or subsequent computed tomography (Figure 1B).

During admission, he continued to require LVP every 24–48 hours with 6–8 L of chylous fluid removed each time. He was started on a medium-chain triglyceride diet with aid of a dietitian without noticeable improvement. Total parenteral nutrition was deferred, given risk of potential complications. Given the absence of a readily reversible cause, subcutaneous octreotide 200 mg 3 times daily was started. After 1–2 weeks of octreotide and PTA dose of diuretics, his ascites improved, and the LVP frequency decreased to every 2–3 weeks after his discharge. In addition, the fluid appearance changed from milky to straw-colored. Several months later, although still on octreotide, he underwent a successful LT-coronary artery bypass graft and remains well postoperatively without recurrence of chylous ascites while off octreotide.

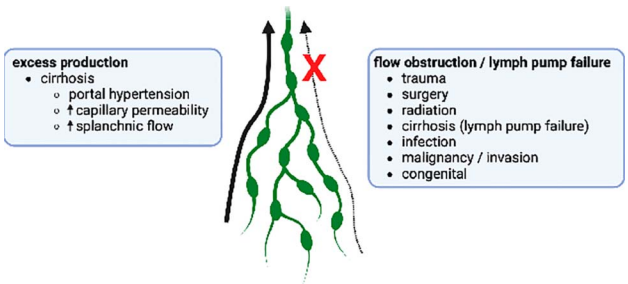

Figure 2. Cartoon depicting multiple etiologies of chylous ascites. Chylous ascites results from either excess lymph production or poor lymph flow, either from lymphatic obstruction or lymph pump failure. In cirrhosis, portal hypertension and increased capillary permeability combined with increased splanchnic blood flow result in excess production of lymph. However, neurohormonal mechanisms also result in lymphatic pump failure in cirrhosis. Together, these mechanisms put patients with cirrhosis at increased risk of chylous ascites. Additional causes of mechanical flow obstruction are listed. Figure was generated with biorender.com.

DISCUSSION

There are limited case reports and case series demonstrating successful treatment of primary chylous ascites in patients with portal hypertension using octreotide.[4–7] Our case provides further evidence for this treatment modality in a similar patient.

Chylous ascites, defined as ascitic fluid triglycerides greater than 200 mg/dL, results from disruption of the lymphatic system, which travels from the periphery to the thoracic duct. Lymph accumulation can result from either excessive lymph production or flow obstruction (Figure 2).[8] There are many possible etiologies, including cirrhosis with portal hypertension. In cirrhosis, multiple mechanisms increase lymph production and decrease lymphatic drainage. Elevated caval and hepatic venous pressures increase hepatic lymph production. Portal hypertension leads to interstitial fluid and lymph overload and a compensatory dilation of lymphatic vessels. Cirrhosis is also associated with a vasodilated state with hyperdynamic circulation that results in lymphatic pump failure.[2] Cumulatively, this can result in chylous ascites. However, it is unclear why this only occurs in $<1\%$ of patients with cirrhosis and ascites.

Our patient had additional confounding factors that may have caused chylous ascites. These include malignancy that can invade the lymphatics and radiation therapy that can cause lymphatic

fibrosis.[3] Determining the most probable etiology is important because it affects treatment and eligibility for LT. Cross-sectional imaging did not show extensive tumor involvement or fibrosis from previous TARE. In adition, new-onset chylous ascites after TARE has not been reported. Lymphangiogram did not show evidence of lymphatic injury. It is important to note that inguinal lymphangiogram may be unable to visualize lymph extravasation within intestinal and hepatic lymphatics, and intrahepatic and mesenteric lymphangiograms have low success rates.[9] As such, pinning our patient's chylous ascites on cirrhosis was a diagnosis of exclusion, and determining the etiology was necessary for both current treatment but also determining his transplant candidacy.

When a readily reversible anatomic cause of chylous ascites is not identified, in addition to diuretic therapy and intermittent LVP, management ranges from (1) dietary modifications (eg, medium chain triglycerides) or even total parenteral nutrition to decrease lymph production, (2) medications (eg, octreotide) to decrease chyle production, (3) procedures including lymphangiogram with percutaneous embolization or a transjugular intrahepatic portosystemic shunt for portal venous decompression, and (4) peritoneal drains for palliation.[3] There is no consensus on an optimal management approach. Successful usage of octreotide is limited to case reports.[5–7] The somatostatin receptor analog is believed to decrease portal pressures, intestinal absorption of triglycerides, and lymph flow to reduce chylous ascites. There is no guidance on dosing and therapy duration, and, in our case, it was continued until the time of LT. Finally, despite being used as a diagnostic test, there are rare reports of nondiagnostic lymphangiograms improving chylous ascites, presumably because of a local inflammatory reaction caused by lipiodol adjacent to the site of leak.[9,10] Notably, these results from lymphangiograms have been reported in patients with chylous ascites from etiologies, other than cirrhosis. This may be due to the drainage pattern of inguinal lipiodol into the cisterna chyli and subsequently thoracic duct without reaching the intestinal or hepatic lymphatics in a retrograde path. Thus, it is unlikely that the lymphangiogram provided therapeutic benefit in our patient even in the absence of an observed leak.

In summary, this report describes successful treatment of refractory chylous ascites with octreotide in a patient with cirrhosis. Octreotide should be considered for treatment of primary chylous ascites, but more robust data and clinical trials are needed to determine its true efficacy in this rare condition that is associated with high mortality.

DISCLOSURES

Author contributions: All authors made substantial contributions to the work including acquisition and interpretation of data. All authors drafted the work and reviewed critically and approved the final manuscript before submission. MA Odenwald is the article guarantor and will accept full responsibility for the conduct of the study.

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REFERENCES

1. Cardenas A, Chopra S. Chylous ascites. *Am J Gastroenterol.* 2002;97(8):1896–900.
2. Bhardwaj R, Vaziri H, Gautam A, Ballesteros E, Karimeddini D, Wu GY. Chylous ascites: A review of pathogenesis, diagnosis and treatment. *J Clin Transl Hepatol.* 2018;6(1):105–13.
3. Lizaola B, Bonder A, Trivedi HD, Tapper EB, Cardenas A. Review article: The diagnostic approach and current management of chylous ascites. *Aliment Pharmacol Ther.* 2017;46(9):816–24.
4. Leong RW, House AK, Jeffrey GP. Chylous ascites caused by portal vein thrombosis treated with octreotide. *J Gastroenterol Hepatol.* 2003;18(10):1211–3.
5. Berzigotti A, Magalotti D, Cocci C, Angeloni L, Pironi L, Zoli M. Octreotide in the outpatient therapy of cirrhotic chylous ascites: A case report. *Dig Liver Dis.* 2006;38(2):138–42.
6. Zhou DX, Zhou HB, Wang Q, Zou SS, Wang H, Hu HP. The effectiveness of the treatment of octreotide on chylous ascites after liver cirrhosis. *Dig Dis Sci.* 2009;54(8):1783–8.
7. Ghimire S, Shah H, Paudel S, Yang TJ, Khan HM, Khan HMA. Chylous ascites and pleural effusion treated with intravenous octreotide. *Cureus.* 2020;12(6):e8669.
8. Kumar R, Anand U, Priyadarshi RN. Lymphatic dysfunction in advanced cirrhosis: Contextual perspective and clinical implications. *World J Hepatol.* 2021;13(3):300–14.
9. Nadolski GJ, Chauhan NR, Itkin M. Lymphangiography and lymphatic embolization for the treatment of refractory chylous ascites. *Cardiovasc Interv Radiol.* 2018;41(3):415–23.
10. Kitagawa S, Sakai W, Hasegawa T. Ultrasound-guided intranodal lymphangiography with ethiodized oil to treat chylous ascites. *ACG Case Rep J.* 2016;3(4):e95.