Ischemic liver lesions mimicking neoplasm in a patient with severe chronic mesenteric ischemia

Bernardo C. Mendes, MD,[a] Gustavo S. Oderich, MD,[a] Thanila A. Macedo, MD,[b] and Roger K. Moreira, MD,[c] *Rochester, Minn*

Chronic mesenteric ischemia most frequently presents with abdominal pain, weight loss, and food fear. Ischemic involvement of the liver is infrequent because of the dual blood supply via the portal vein and hepatic artery. Hepatic infarction has been associated with embolization, thrombosis, arterial injury, prothrombotic states, and impairment of portal venous flow. We report a patient with chronic mesenteric ischemia and severe mesenteric arterial disease who presented with large liver masses suspicious for neoplasm. Tissue samples from two hepatic biopsies confirmed ischemic lesions. After open surgical mesenteric revascularization, the patient had complete symptom improvement and nearly complete regression of the liver lesions. (J Vasc Surg Cases 2015;1:144-7.)

Chronic mesenteric ischemia (CMI) most often presents with abdominal pain, weight loss, and food fear.[1] Because of extensive collaterals, most patients with symptoms of CMI have stenosis or occlusion of at least two of the three mesenteric arteries.[2,3] Diagnostic imaging is recommended to confirm the diagnosis, plan an intervention, and evaluate other potential causes of abdominal pain such as malignancy, inflammatory bowel disease, or motility disorders. Liver infarction as a presentation of CMI has not been described, possibly due to dual blood supply via the hepatic artery and portal vein.[4] We report a patient who presented with CMI associated with severe three-vessel mesenteric artery disease and two large ischemic liver lesions. The patient consented with the operation and with publication of this manuscript.

CASE REPORT

A 76-year-old man presented with an 8-month history of postprandial abdominal pain, food fear, and 40-pound weight loss. The patient reported significant worsening of abdominal pain over 5 weeks requiring total parenteral nutrition due to inability to have any oral intake. His medical history was notable for prior attempted mesenteric stenting at another institution 2 months before presentation, which was unsuccessful, coronary artery disease, coronary artery bypass grafting, peripheral artery disease, bilateral iliac artery stents, and Barrett esophagus. Computed tomography angiography (CTA) demonstrated heavy calcification and occlusion of the celiac artery, long-segment occlusion of the superior mesenteric artery (SMA), and a small, diseased inferior mesenteric artery (Fig 1). Both internal iliac arteries were occluded with distal collateral reconstitution.

There were two large heterogeneous liver lesions, including a 6-cm mass in the inferior aspect of the right hepatic lobe (segment VI) and 4-cm poorly marginated mass with enhancing and cystic components in the periphery of the right hepatic lobe (segment VIII). Both lesions had associated segmental bile duct dilatation (Fig 2). There was thickening of the ascending colon wall. Concentrations of serum alanine and aspartate aminotransferases, alkaline phosphatase, and albumin, and prothrombin time were within normal limits.

A gastroenterology consultation was obtained to evaluate possible metastatic malignant disease. An esophagogastroduodenoscopy demonstrated Barrett esophagus, with no evidence of malignancy. A colonoscopy was negative for neoplasm but suggestive of ischemic-type injury. Carcinoembryonic antigen was minimally elevated at 4.9 ng/mL.

A specimen obtained from an ultrasound-guided biopsy of the liver masses demonstrated necrotic liver tissue that could potentially correspond to malignancy, with no definite neoplasia identified. A specimen from a repeat biopsy in a different lesion showed isolated hepatic necrosis with no evidence of malignancy (Fig 2). These findings led to a proposed diagnosis of hepatic infarction secondary to chronic atherosclerotic mesenteric ischemia, and open surgical mesenteric revascularization was recommended.

A midline transperitoneal abdominal exploration revealed no evidence of palpable abdominal masses involving the liver, small intestine, colon, or peritoneum. There was extensive calcification of the infrarenal aorta, iliac arteries, and proximal SMA. A bifurcated supraceliac aorta-to-common hepatic artery and SMA graft was performed using a 14-mm × 7-mm polyester graft. The left side limb was tunneled in a retropancreatic position and anastomosed end-to-side to the distal SMA, and the right limb was anastomosed end-to-side to the common hepatic artery. Intraoperative ultrasound imaging revealed a widely patent graft with no evidence of technical defects.

The patient had an uneventful postoperative course, with adequate tolerance to oral diet, discontinuation of the total parenteral nutrition, and hospital discharge on postoperative day 8. A CTA repeated 4 days after the operation demonstrated widely

From the Division of Vascular and Endovascular Surgery,[a] the Department of Radiology,[b] and the Department of Pathology,[c] Mayo Clinic.

Author conflict of interest: none.

Reprint requests: Gustavo S. Oderich, MD, Mayo Clinic, 200 First St SW, Rochester, MN 55905 (e-mail: oderich.gustavo@mayo.edu).

The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

2352-667X

http://dx.doi.org/10.1016/j.jvsc.2015.03.020

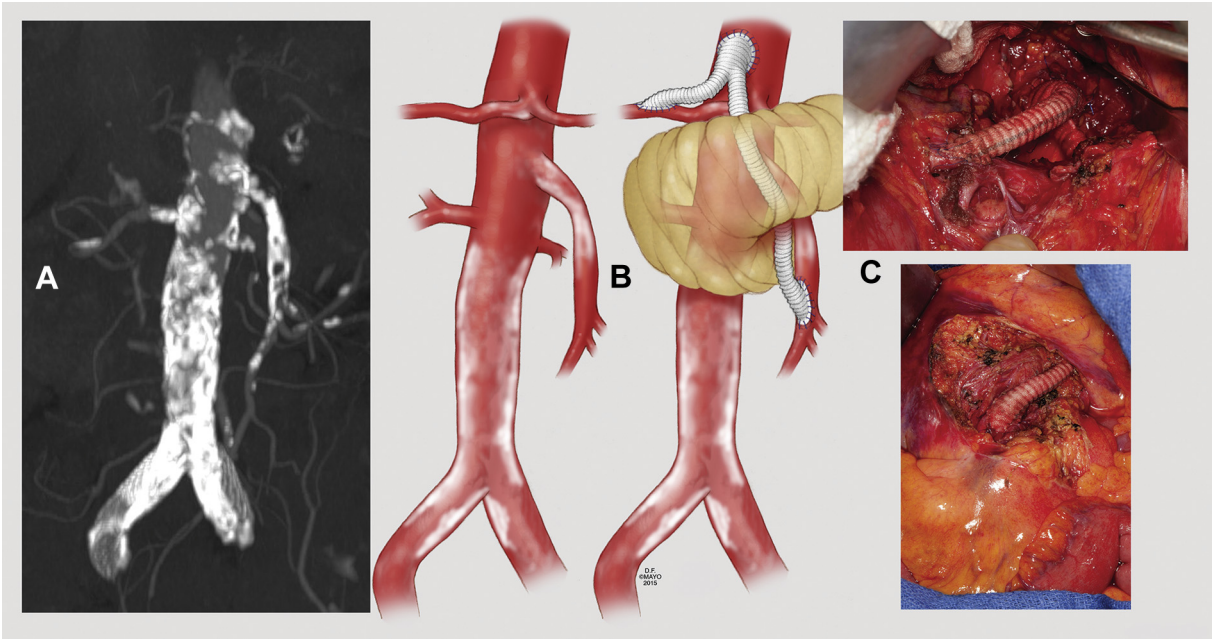

Fig 1. A, Computed tomography angiography (CTA) of the abdominal aorta demonstrates a highly calcified aorta and bilateral iliac vessels, with occlusion of the celiac axis and a long segment of the superior mesenteric artery (SMA). **B,** Schematic drawing and **(C)** intraoperative photograph show a 14-mm × 7-mm bifurcated polyester graft originating from the supraceliac aorta, with one limb anastomosed end-to-side to the common hepatic artery and one limb tunnelled retropancreatic and anastomosed end-to-side to the distal SMA. Reprinted by permission of the Mayo Foundation for Medical Education and Research. All rights reserved.

patent bypass limbs and a decrease in the size of both ischemic lesions. At 12 months, the patient had gained 40 pounds and was free of recurrent symptoms. Repeat CTA demonstrated a widely patent bifurcated graft and nearly complete regression of both hepatic lesions, thus confirming its ischemic nature (Fig 2, *B*).

DISCUSSION

CMI rarely involves solid organs, such as the liver or spleen, unless there is distal embolization from a proximal source. Hepatic infarction has been associated with surgical or interventional procedures, liver transplantation, arterial embolization, and prothrombotic states.[5,6] The vascular anatomy of the upper abdomen contains extensive collaterals that compensate chronic occlusion of the celiac axis or proximal segments of the hepatic artery, including the left gastric, gastroduodenal, pancreatoduodenal, and parietal vessels such as the intercostal and phrenic arteries.[4,7] Because of its dual blood supply via the hepatic artery and portal vein and the extensive collateral network between the mesenteric arteries, the liver is resistant to chronic ischemic injury.[8] Provided there is normal portal venous flow, hepatic necrosis is uncommon with isolated hepatic artery occlusion, with the exception of bile duct and gallbladder ischemia that occurs with injury or thrombosis of the right hepatic artery. Isolated portal venous obstruction or thrombosis with normal arterial inflow will rarely lead to hepatic necrosis.[4,9]

In the patient presented here, there was extensive arterial disease involving the celiac axis, SMA, and inferior mesenteric artery, with no evidence of any abnormality affecting the portal venous system. The hepatic infarction was likely due to significant ischemia from the poor collateral arterial network, although distal arterial embolization could not be ruled out as a potential cause.

The radiologic appearance of hepatic infarction varies widely. Prior descriptions include peripheral wedge-shaped to central, round, nonhomogeneous lesions, with or without bile duct necrosis.[5,10,11] Holbert et al[5] reviewed CT studies from 18 patients who had 55 specimen-proven ischemic hepatic lesions. In 83% of the patients, hepatic infarction resulted from complications of liver transplantation. At presentation, all hepatic lesions were associated with lower attenuation in unenhanced and enhanced studies compared with surrounding hepatic parenchyma. Round or oval lesions were more common, but wedge-shaped lesions with bile duct necrosis occurred in few patients. One of four wedge-shaped lesions evolved into round lesions, which are more difficult to differentiate from abscess or metastatic disease on the basis of CT appearance. In these cases, a biopsy specimen was recommended to rule out or confirm malignancy. The authors concluded that hepatic infarction can present with varied appearance reflecting the evolutionary process of ischemic injury.[5] In the presented case, hepatic lesions had a round-oval pattern rather than wedge-shaped, which led to needle aspiration due to the indeterminate appearance and malignant potential.

The importance of cross-sectional imaging in the evaluation of patients with CMI cannot be overemphasized.

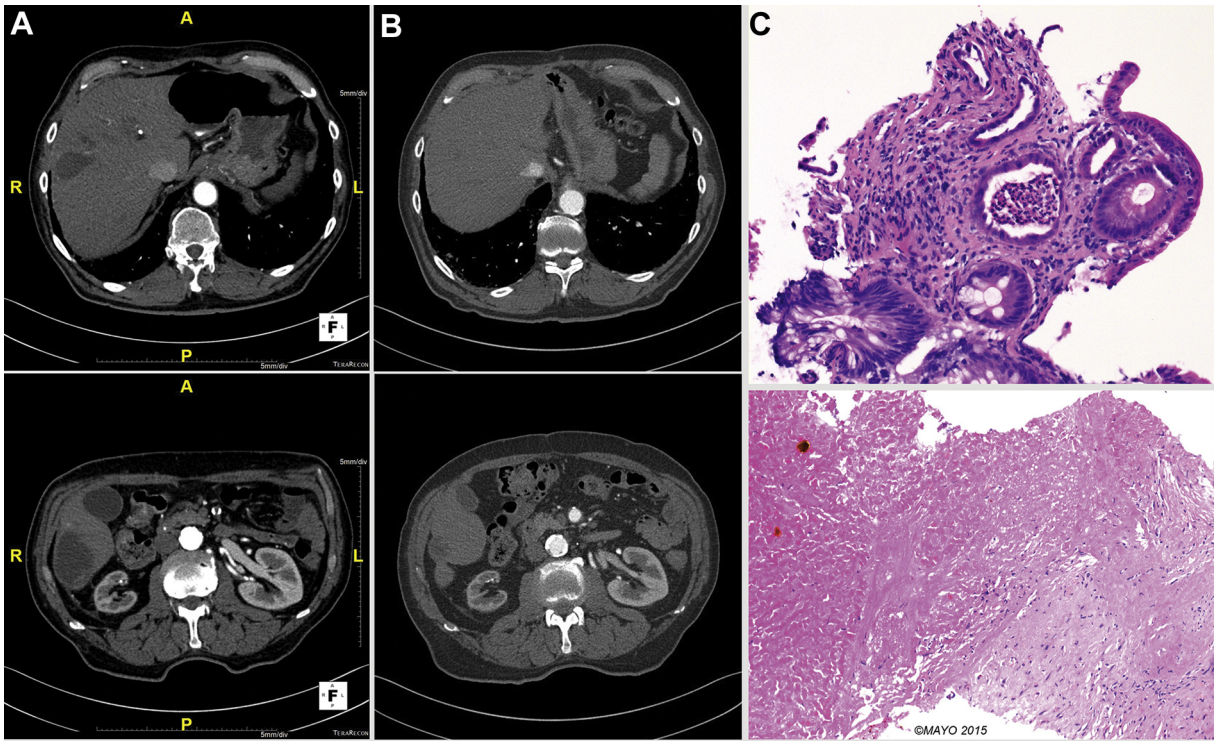

Fig 2. A, Portal venous-phase computed tomography angiography (CTA) demonstrates two hypodense, hepatic lesions measuring 4 cm and presenting with enhancing and cystic components in the segment VIII, with associated segmental bile duct dilation, and a nonhomogeneous mass measuring 6 cm in the segment VI. **B,** Postoperative CTA demonstrates nearly complete resolution of hepatic lesions after hepatic and mesenteric revascularization. **C,** ***Top,*** Colon biopsy specimen shows colonic mucosa with focal areas of crypt "withering" and associated hyalinization of the lamina propria, consistent with ischemic-type injury. A focus of acute inflammation (crypt abscess) is also present (hematoxylin and eosin, original magnification ×400. ***Bottom,*** Needle core biopsy sample shows extensive coagulative necrosis of the liver parenchyma with surrounding fibrous tissue and minimal reactive inflammatory infiltrate, without evidence of malignancy (hematoxylin and eosin, original magnification ×200). Reprinted by permission of the Mayo Foundation for Medical Education and Research. All rights reserved.

Because symptoms are nonspecific, other causes of chronic abdominal pain and weight loss should be evaluated, including malignant and inflammatory bowel disease. Imaging is useful not only to confirm mesenteric artery disease but also to plan mesenteric revascularization. In this case, the extensive involvement of the SMA by long-segment occlusion and calcification rendered percutaneous stenting technically more difficult and prone to a suboptimal result. Recanalization would require stenting of at least 7 cm of SMA and re-entrance in the very distal SMA. Although stenting is possible in select cases of long occlusion, these patients have higher rates of restenosis and late failure. Therefore, we opted for an open surgical revascularization.

Albeit rare, this case should raise the awareness that liver lesions in patients with classic symptoms of CMI can be associated with ischemia. Although angioplasty and stenting is the first treatment option in most patients with CMI,[12,13] a subset of patients will still benefit from conventional open revascularization due to extensive occlusion, failed stents, or unusual etiology.[2,14,15] This case illustrates an example of long-segment occlusion of the SMA and celiac artery with heavy calcification with previous failed attempted endovascular revascularization, rendering open revascularization a reasonable option. The choice of two-vessel reconstruction is controversial but made logical sense in this patient with ischemic liver lesions revascularization of the hepatic artery.[1,16]

CONCLUSIONS

This case illustrates an unusual clinical presentation of CMI with ischemic damage to the liver mimicking neoplasm. The long-segment occlusion and heavy calcification of the mesenteric arteries rendered this patient not an ideal candidate for an endovascular intervention. Open revascularization in these patients remains a valuable option and is associated with excellent long-term symptom improvement and high patency rates.

REFERENCES

1. Oderich GS, Bower TC, Sullivan TM, Bjarnason H, Cha S, Gloviczki P. Open versus endovascular revascularization for chronic mesenteric ischemia: risk-stratified outcomes. J Vasc Surg 2009;49:1472-9 e3.

2. Oderich GS, Gloviczki P, Bower TC. Open surgical treatment for chronic mesenteric ischemia in the endovascular era: when it is necessary and what is the preferred technique? Semin Vasc Surg 2010;23:36-46.
3. Wildermuth S, Leschka S, Alkadhi H, Marincek B. Multislice CT in the pre- and postinterventional evaluation of mesenteric perfusion. Eur Radiol 2005;15:1203-10.
4. Carroll R. Infarction of the human liver. J Clin Pathol 1963;16:133-6.
5. Holbert BL, Baron RL, Dodd GD. Hepatic infarction caused by arterial insufficiency: spectrum and evolution of CT findings. AJR Am J Roentgenol 1996;166:815-20.
6. Miura F, Asano T, Amano H, Yoshida M, Toyota N, Wada K, et al. Eleven cases of postoperative hepatic infarction following pancreatobiliary surgery. J Gastrointest Surg 2010;14:352-8.
7. Takeuchi Y, Arai Y, Inaba Y, Ohno K, Maeda T, Itai Y. Extrahepatic arterial supply to the liver: observation with a unified CT and angiography system during temporary balloon occlusion of the proper hepatic artery. Radiology 1998;209:121-8.
8. Mays ET, Wheeler CS. Demonstration of collateral arterial flow after interruption of hepatic arteries in man. N Engl J Med 1974;290:993-6.
9. Yamashita K, Tsukuda H, Mizukami Y, Ito J, Ikuta S, Kondo Y, et al. Hepatic infarction with portal thrombosis. J Gastroenterol 1997;32:684-8.
10. Giovine S, Pinto A, Crispano S, Lassandro F, Romano L. Retrospective study of 23 cases of hepatic infarction: CT findings and pathological correlations. Radiol Med 2006;111:11-21.
11. Smith GS, Birnbaum BA, Jacobs JE. Hepatic infarction secondary to arterial insufficiency in native livers: CT findings in 10 patients. Radiology 1998;208:223-9.
12. Schermerhorn ML, Giles KA, Hamdan AD, Wyers MC, Pomposelli FB. Mesenteric revascularization: management and outcomes in the United States, 1988-2006. J Vasc Surg 2009;50:341-8 e1.
13. Oderich GS, Erdoes LS, LeSar C, Mendes BC, Gloviczki P, Cha S, et al. Comparison of covered stents versus bare metal stents for treatment of chronic atherosclerotic mesenteric arterial disease. J Vasc Surg 2013;58:1316-23.
14. Tallarita T, Oderich GS, Macedo TA, Gloviczki P, Misra S, Duncan AA, et al. Reinterventions for stent restenosis in patients treated for atherosclerotic mesenteric artery disease. J Vasc Surg 2011;54:1422-9 e1.
15. Oderich GS, Tallarita T, Gloviczki P, Duncan AA, Kalra M, Misra S, et al. Mesenteric artery complications during angioplasty and stent placement for atherosclerotic chronic mesenteric ischemia. J Vasc Surg 2012;55:1063-71.
16. Park WM, Cherry KJ Jr, Chua HK, Clark RC, Jenkins G, Harmsen WS, et al. Current results of open revascularization for chronic mesenteric ischemia: a standard for comparison. J Vasc Surg 2002;35:853-9.

Submitted Feb 5, 2015; accepted Mar 17, 2015.