International Journal of Gastrointestinal Intervention

journal homepage: www.ijgii.org

Case Report

Interventional treatment of a huge hepatic artery aneurysm as an unusual cause of upper gastrointestinal bleeding

Se Hwan Kwon^{1,*}, Jehong Yoon¹, Seung Yeon Noh^{1,2}, Hyung Joon Ahn³, and Joo Hyeong Oh¹

ABSTRACT

A 61-year-old man presented to our hospital with abdominal pain and melena. He showed no obvious source of bleeding on gastroduodenoscopy. Computed tomography and conventional angiography revealed a huge aneurysm arising from the common hepatic artery. The aneurysm was embolized using coils. The procedure was uncomplicated, and the patient was discharged from the hospital 5 days after admission. Herein, we present the case of a rare huge hepatic artery aneurysm (5.7 × 6.0 cm in size) that caused abdominal pain and upper gastrointestinal bleeding.

Copyright © 2020, Society of Gastrointestinal Intervention.

Keywords: Aneurysm; Embolization, therapeutic; Hepatic artery; Methods

Introduction

Hepatic artery aneurysm (HAA) is a rare lesion, with an incidence of approximately 0.002% (approximately 20% of all visceral aneurysms), that is difficult to diagnose clinically.^{1,2} The causes of HAA include atherosclerosis, vasculitis, fibromuscular dysplasia, cystic medial necrosis, surrounding inflammation, or trauma.^{3,4} Intrahepatic aneurysms are most frequently the results of trauma, iatrogenic injury from biopsy or intervention, infection, or vasculitis. By contrast, extrahepatic aneurysms are most often degenerative or dysplastic.^{5,6} The presentation of HAA ranges from an incidental finding to an acute life-threatening hemorrhage caused by rupture.⁷ In the past, many HAA cases were diagnosed after rupture, but recently, advances in rapid cross-sectional imaging have enabled earlier diagnosis of HAA.³

Herein, we present the case of a rare huge HAA (5.7 × 6.0 cm in size) that caused abdominal pain and upper gastrointestinal (UGI) bleeding. The patient was successfully treated with interventional coil embolization.

Case Report

A 61-year-old man was admitted to Kyung Hee University Hospital because of a recent upper abdominal pain and melena. He had no history of trauma, diabetes, or surgery. The patient had no connective tissue disorders or other systemic anomalies, and no significant family history of disease. On admission, he was hemodynamically stable. However, his initial hemoglobin and hematocrit levels were mildly decreased (11.9 g/dL and 35.2%, respectively). UGI endoscopy and flexible colonoscopy did not reveal any significant abnormalities, except transported dark blood in the colon. A computed tomography (CT) scan showed a 5.7 × 6.0-cm huge aneurysm with a mural thrombus at the common hepatic artery (Fig. 1). The duodenal bulb, proximal duodenal second portion, and portal veins were contacted with and compressed by a huge aneurysm. We thought that the huge HAA was the underlying cause of UGI bleeding and the hidden concealed small rupture or leaking portion was missed during the endoscopy examination. The patient was felt to be at high risk for rebleeding. After consultation with vascular surgeons and interventional radiologists, interventional treatment (transarterial coil embolization or stent-graft placement) was considered primary treatment op-

¹Department of Radiology, College of Medicine, Kyung Hee University, Seoul, Korea²Department of Radiology, Soonchunhyang University Seoul Hospital, Seoul, Korea³Department of Surgery, College of Medicine, Kyung Hee University, Seoul, Korea
Received December 2, 2019; Revised January 13, 2020; Accepted January 13, 2020

* Corresponding author. Department of Radiology, Kyung Hee University Medical Center, College of Medicine, Kyung Hee University, 23 Kyungheedaero, Dongdaemun-gu, Seoul 02447, Korea.

E-mail address: Kwon98@khu.ac.kr (S.H. Kwon).

Fig. 1. Axial (A), coronal (B) and three-dimensional (C) computed tomography images showing the huge hepatic artery aneurysm (arrows) with the mural thrombus arising from the common hepatic artery. GDA, gastroduodenal artery; PHA, proper hepatic artery; RHA, right hepatic artery; LHA, left hepatic artery.

Fig. 2. Celiac angiogram showing the huge common hepatic artery aneurysm.

tion and transarterial coil embolization was chosen as the therapeutic method because we did not have available stent-graft due to small artery diameter and a very short out-flow arterial tract.

In the angiography room, a right femoral artery approach was used under local anesthesia. After placement of a 5-Fr introducer sheath, a 0.035-inch guidewire (Radiofocus M; Terumo, Tokyo, Japan) and 5-Fr angiography catheter (Cook Medical, Bloomington, IN, USA) were inserted through the introducer sheath. Conventional celiac and hepatic angiographies confirmed the diagnosis (Fig. 2). The common hepatic artery and aneurysm were cannulated using a 5-Fr angiography catheter and 1.98-Fr micro-catheter system (Asahi Intecc, Nagoya, Japan). Then, transarterial coil embolization of the aneurysm was performed, including the in- and out-flow arterial tracts (afferent and efferent arteries). Two 0.018-inch micro-coils (3 mm in diameter, Tornado embolization coil; Cook Medical) and twenty-two 0.035-inch coils (12 mm coil [$n = 8$], 10 mm coil [$n = 9$], 8 mm coil [$n = 1$], 6 mm coil [$n = 1$], 4 mm coil [$n = 3$]; Nester embolization coil; Cook Medical) were used.

The final angiography (Fig. 3) revealed a complete embolization of the aneurysm and preserved proper hepatic artery flow through the gastroduodenal artery. The clinical course of the patient after the embolization was uneventful. Three days after

the treatment, his blood laboratory test results were normal, and his abdominal pain and melena had resolved. He was discharged in good condition from the hospital 5 days after admission. The follow-up CT scan obtained 6 months later showed the complete thrombosis of the previous huge HAA and good flow into the proper hepatic artery through the gastroduodenal artery (Fig. 4).

Discussion

HAAs are usually asymptomatic and diagnosed either incidentally or on autopsy.^{5,6} Recently, the recent advances in and rapid proliferation of the use of cross-sectional imaging have made early detection of HAA possible. However, selective angiographies of the celiac axis and superior mesenteric artery are essential not only to confirm the diagnosis but also to supply important information about the related vascular anatomy. When symptomatic, the most common presentation of patients with intact HAAs is continuous right upper quadrant or epigastric pain.^{8,9} However, they may present with severe abdominal pain, a mass, hypovolemia secondary to rupture or gastrointestinal bleeding with normal endoscopy.¹⁰ Delay in diagnosis may lead to life-threatening hemorrhage. Rupture has been reported in 14%–80% of cases, with mortality rates ranging from 21% to 35%.^{1,5} In 80% of patients with HAA, rupture of the aneurysm is the initial clinical event.⁸ HAA may rupture into the adjacent hepatic venous, portal, gastrointestinal system, or biliary tract, or directly into the abdominal cavity.¹¹ UGI bleeding caused by HAA rupture is rare, but might be a clinically significant cause of UGI bleedings and has been reported.^{3,7,8,10} In the previous case reports, HAA was diagnosed on ultrasonography, CT scan, or angiography and endoscopy revealed no significant abnormality, as was the case in our patient. We think that the hidden concealed small rupture or leaking portion can be missed during the endoscopy examination. Therefore, if the cause of UGI bleeding remains obscure after routine endoscopic evaluations, hidden visceral artery aneurysm should be considered because delayed diagnosis of aneurysm rupture may lead to a life-threatening condition.

HAAs can be treated with surgical or endovascular approaches. While no definite consensus exists regarding the aneurysm size criteria, treatment is usually recommended for visceral artery aneurysms > 2 cm in diameter.^{1,3,12} However, treatment decisions are difficult in some patients and must be made on an individual basis because anatomic suitability, clinical presentation, underlying etiology, general health status, and comorbidity factors can affect the treatment method.^{1,3} Before using endovascular management, for many years, the open surgery was the only treat-

Fig. 3. Post-embolization angiogram of the celiac trunk (A) and superior mesenteric artery (B), showing complete coil embolization of the aneurysm and preservation of the proper hepatic artery flow (arrow) through the gastroduodenal artery.

Fig. 4. Follow-up axial computed tomography image showing the coil embolization and complete thrombosis of the huge aneurysm (arrows).

ment method. Recently, the mainstay of treatment is endovascular techniques such as coil embolization or covered stent placement.⁵ Endovascular management should also achieve isolation of the aneurysm from the normal arterial circulation. This isolation can be accomplished in several ways. In coil embolization, care must be taken to ensure that any collateral arteries that might support the continued flow of the aneurysm are occluded. However, this technique may be associated with potential early complications such as hepatic ischemia, hepatic abscess, infection, sepsis, and cholecystitis.^{5,8} Covered stent or stent-graft placement provide another means of excluding visceral artery aneurysm from the normal arterial circulation. Limitations of covered stents include delivery systems whose size and rigidity preclude stent placement in distal tortuous branches. Covered stents are usually reserved for arteries 6 mm or larger in diameter because of the risk of thrombosis of smaller arteries.^{3,5} These endovascular techniques play an important role, especially in the management of surgical candi-

dates at high risk.

In conclusion, we present an unusual case of huge HAA manifested by UGI bleeding, and the UGI bleeding was successfully managed with interventional coil embolization. Safety and effectiveness of the endovascular treatment for HAA warrant further treatment and investigations in the future.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

ORCID

Se Hwan Kwon, <https://orcid.org/0000-0002-7777-8422>

References

1. Abbas MA, Fowl RJ, Stone WM, Panneton JM, Oldenburg WA, Bower TC, et al. Hepatic artery aneurysm: factors that predict complications. *J Vasc Surg.* 2003;38: 41–5.
2. Pilleul F, Valette PJ. [Management of aneurysms of the hepatic artery. 15 patients]. *Presse Med.* 2001;30:1139–42. French.
3. Hemmati H, Karimian M, Moradi H, Farid Marandi K, Haghdoust A. Endovascular treatment of a huge hepatic artery aneurysm by coil embolization method: a case report. *Iran J Radiol.* 2015;12:e5200.
4. Parmar H, Shah J, Shah B, Patkar D, Varma R. Imaging findings in a giant hepatic artery aneurysm. *J Postgrad Med.* 2000;46:104–5.
5. Noshier JL, Chung J, Brevetti LS, Graham AM, Siegel RL. Visceral and renal artery aneurysms: a pictorial essay on endovascular therapy. *Radiographics.* 2006;26: 1687–704; quiz 1687.
6. Shanley CJ, Shah NL, Messina LM. Common splanchnic artery aneurysms: splenic, hepatic, and celiac. *Ann Vasc Surg.* 1996;10:315–22.
7. Narula HS, Kotru A, Nejim A. Hepatic artery aneurysm: an unusual cause for gastrointestinal haemorrhage. *Emerg Med J.* 2005;22:302.
8. Doğan R, Yildirim E, Gökürk S. Gastrointestinal hemorrhage caused by rupture of a pseudoaneurysm of the hepatic artery. *Turk J Gastroenterol.* 2012;23:160–4.
9. Riesenman PJ, Bower TC, Oderich GS, Bjarnason H. Multiple hepatic artery aneurysms: use of transcatheter embolization for rupture. *Ann Vasc Surg.* 2006;20:399–404.
10. Frank M, Phillips R, Aldin Z, Ghosh D. Hepatic artery aneurysm: an unusual cause of upper gastrointestinal bleed. *BMJ Case Rep.* 2017;2017:bcr-2017-219865.
11. Finley DS, Hinojosa MW, Paya M, Imagawa DK. Hepatic artery pseudoaneurysm: a report of seven cases and a review of the literature. *Surg Today.* 2005;35:543–7.
12. de Ruiter-Derksen GL, Bruijnen RC, Joosten F, Reijnen MM. Endovascular treatment of a hepatic artery aneurysm causing chronic abdominal pain; a case report. *Ann Hepatol.* 2010;9:104–6.