Laparoscopic Management of Massive Hemobilia From an Intrahepatic Aneurysm

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ABSTRACT

Background: Intrahepatic arterial aneurysms are rare and typically related to trauma, transplantation, iatrogenic injury, or infection. They account for approximately 10% of clinically significant hemobilia.

Case Report: We present the case of a 49-year-old man with an intraparenchymal hepatic artery aneurysm that presented as massive hemobilia following a laparoscopic cholecystectomy. The aneurysm could not be managed by interventional embolization and required a left hepatic lobectomy, which was performed laparoscopically.

Discussion: Evaluation of hemobilia requires a multidisciplinary team approach. The diagnosis of hepatic artery aneurysm can be most readily made by MRI or CT scan. Interventional embolization of the aneurysm may be effective treatment but is not always possible due to anatomic considerations. Where indicated, surgical resection in a manner that preserves a maximal amount of normal hepatic parenchyma is the treatment of choice.

Conclusion: This is the first report of laparoscopic liver resection performed for bleeding from a hepatic artery aneurysm and adds an effective treatment modality to the surgical armamentarium.

Key Words: Hepatic parenchyma, Liver resection, Hepatic artery aneurysm, Hemobilia.

INTRODUCTION

Hemobilia, the phenomenon of bleeding into the biliary tree, from an intrahepatic aneurysm is an extremely rare occurrence. Hepatic artery aneurysms account for approximately 10% of hemobilia cases, and most are related to trauma, transplantation, surgical injury, or infection. Hemobilia caused by an intrahepatic arterial aneurysm is rare and presents a challenging medical diagnosis. Currently, several potential therapeutic options are available, including embolization, stenting, and resection. The choice of treatment must consider the acuity of the bleeding and the anatomic location of the aneurysm as well as the underlying cause. The role of laparoscopic liver resection in the treatment of a bleeding hepatic artery aneurysm has not been evaluated previously.

We report on a 49-year-old male with massive hemobilia, a recent laparoscopic cholecystectomy, a past medical history of hypertension, and a newly diagnosed aortic dissection. We present the diagnostic approach, a review of the literature, and treatments for hemobilia caused by an intrahepatic aneurysm. In this case, laparoscopic left hepatic lobectomy was performed for definitive treatment.

CASE REPORT

A 49-year-old African-American male with abdominal pain and dark tarry stools was seen at a community hospital. He had a past medical history significant only for hypertension. He had developed right upper quadrant pain and had passed several dark melanotic stools over 2 days. Hospital admission laboratory tests showed his hemoglobin and hematocrit were 5.3 and 16.8, respectively. He required transfusion of several units of packed red blood cells but remained hemodynamically stable. An ultrasound demonstrated gallbladder sludge, stones, and thickening consistent with cholecystitis. Within 72 hours of admission, the patient’s hematocrit became stable, and he did not require further transfusion. A laparoscopic cholecystectomy was performed. During back-table dissection of the gallbladder, coagulated blood was found. Postoperatively, the patient had further gastrointestinal bleeding with significant drops in hemoglobin and hematocrit requiring repeated transfusions. He received 7 units...
of blood within 48 hours after cholecystectomy and was transferred to our hepatobiliary service for further treatment.

A CT scan with intravenous contrast was obtained. The CT scan revealed a previously undiagnosed aortic dissection beginning just distal to the left subclavian artery and terminating at the level of the left renal artery. The scan also showed intrahepatic biliary dilatation and an accompanying left hepatic artery aneurysm with associated outpouching of the right portal vein that likely represented a venous varix (Figure 1a). An MRA was obtained to further define the hepatic vascular abnormalities. The MRA showed a lobulated area in the left lobe of the liver, which measured 3cm by 3cm, highly suggestive of a portal venous varix (Figure 1b).

The aortic dissection was thought to be stable, not requiring intervention. The options for treatment of the hepatic vascular/biliary abnormality included embolization by interventional techniques or hepatic resection.

As an initial treatment approach, an arteriogram with embolization was attempted. The arteriogram and subsequent catheter placement were technically challenging, secondary to the aortic dissection with prominent false and true lumens. The catheter was guided to the common hepatic artery and further to the left hepatic artery; however, secondary to high-grade stenosis at the origin of the left hepatic artery aneurysm, the catheter could not be advanced, preventing embolization.

Definitive treatment required a left hepatic lobectomy. The laparoscopic approach consisted of four 12-mm ports. Intraoperative ultrasound duplex was used to identify the location of the aneurysm with the left lobe of the liver. A plane of resection that maximized preservation of normal parenchyma was marked using cautery. The hepatic parenchyma was divided using the LigaSure Atlas. The left portal vein and left hepatic artery were divided intrahepatically by using a 45-mm laparoscopic linear stapler, with 2.5-mm staples. Ultimately, the left hepatic vein was isolated and similarly divided. A 5-cm left subcostal incision was made to remove the specimen. The specimen contained a large aneurysm with connection to the biliary system (Figure 2). The final pathology was consistent

**Figure 1.** A. CT scan demonstrating left lobe hepatic artery aneurysm and aortic dissection. B. Sagittal MRI with lobular vascular abnormality in left hepatic lobe.

**Figure 2.** Specimen after resection with 2-cm aneurysm.
with an aneurysm that communicated with the biliary system and also demonstrated thrombi in adjacent portal vein tributaries. The patient had an uncomplicated hospital course without further bleeding. He was discharged on postoperative day 3.

**DISCUSSION**

A hepatic artery aneurysm is very rare but potentially life-threatening. Of the visceral arteries most prone to true aneurysmal formation, the splenic and hepatic arteries are most common. Splenic and hepatic aneurysms account for 60% and 20% of all visceral artery aneurysms, respectively. Hepatic artery aneurysms may occur anywhere along the hepatic arterial system, both external to the liver and within the liver parenchyma. Seventy-seven percent of hepatic aneurysms are confined to the segment proximal to the liver, 20% have combined intra- and extraparenchymal involvement, and 3% are exclusively within the liver. Excluding traumatic aneurysms, patients most commonly present within the sixth decade of life. There is a male predominance (approximately 3:2).

Historically, most hepatic artery aneurysms have been mycotic in origin. Today, nearly 50% of hepatic aneurysms are traumatic in nature. Traumatic hepatic aneurysms typically result from either trauma (car accidents) or are iatrogenic, resulting from a diagnostic procedure. The remainder is associated with medial degeneration and secondary atherosclerosis, with hypertension as the predominant comorbid condition. Interestingly, the populations most at risk for developing hepatic aneurysms are patients with fibromuscular dysplasia or polyarteritis nodosa.

Most hepatic aneurysms are found incidentally on CT scan or angiography. Patients with symptoms often present with hemobilia and melena. Abdominal pain is associated with rapidly expanding aneurysms. Decompression into the biliary tree with resulting hemobilia is more common than free rupture is into the abdominal cavity.

Hemobilia is an unusual cause of upper gastrointestinal bleeding. In recent years, hemobilia has increased in incidence, presumably secondary to increasing numbers of surgical and interventional radiologic procedures involving the liver. Approximately 10% of hemobilia cases are associated with a hepatic artery aneurysm. The most common symptoms associated with hemobilia are jaundice (30%), biliary colic (52%), and gastrointestinal hemorrhage (73%). These 3 symptoms are referred to as

Quincke’s classic triad of hemobilia. The complete triad occurs in approximately 20% of symptomatic patients. Once an upper gastrointestinal (UGI) bleed is suspected, endoscopy is the first-line diagnostic modality. If blood or clot is seen at the ampulla of Vater, hemobilia is highly likely. However, only 12% of the upper endoscopies done to evaluate acute UGI bleeding are diagnostic. The choice between other diagnostic modalities depends on patient history and clinical examination. Radiologic studies, such as computed tomography, abdominal sonography, magnetic resonance, and angiography, are all potentially useful in identifying hepatic arterial abnormalities. Angiography can detect the source of significant hemobilia in over 90% of patients, allowing for localization of the vascular abnormality and possible therapeutic stenting or embolization.

Treatment options for intrahepatic versus extrahepatic aneurysms are quite different. The ultimate goal is to prevent or stop active hemorrhage and relieve any associated biliary obstruction. For extrahepatic aneurysm excision, arterial reconstruction or intraarterial stenting to affect aneurysm exclusion are the most effective options. Ligation is considered as definitive treatment where no other options exist.

If the aneurysm is intrahepatic, transarterial embolization is the first-line treatment. Embolization has a success rate of between 80% and 100% in some reports and a lower morbidity and mortality rate compared with that in open surgery. Embolization can maximize preservation of hepatic parenchyma. If embolization fails or is contraindicated, surgical intervention, such as ligation of the artery or aneurysm excision, should be considered.

Laparoscopic surgery for hemobilia caused by a hepatic artery aneurysm has not been described previously in the literature. In centers experienced in laparoscopic liver resection, this approach adds an additional treatment modality. In our patient, laparoscopic resection allowed for complete removal of the aneurysm, preventing further bleeding or other potential complications that could result from a vascular-biliary fistula.

**CONCLUSION**

Hemobilia secondary to intrahepatic aneurysm in a patient with newly diagnosed aortic dissection is a rare finding. A systematic approach to hemobilia should be followed to decrease morbidity and mortality. Embolization is a first-line therapy for intrahepatic aneurysms but could not be accomplished in this case secondary to
stenosis and anatomic limitations imposed by aortic dissection. In this case, laparoscopic left lobe resection provided definitive treatment without complications. Increasing familiarity with laparoscopic liver resection in centers familiar with the techniques involved provides additional options for patient treatment that may expedite treatment and enhance patient recovery.

References:
1. Harlaftis NN, Akin JT. Hemobilia from ruptured hepatic aneurysm. Report of a case and review of the literature. *Am J Surg*. 1977;133:229–232.
2. Abbas MA, Stone WM, Fowl RJ, et al. Splenic artery aneurysms. Two decades experience at Mayo clinic. *Ann Vasc Surg*. 2002;16:442–449.
3. Stanley JC, Thompson NW, Fry WJ. Splanchnic artery aneurysms. *Arch Surg*. 1970;101:689–697.
4. Berceli SA. Hepatic and splenic artery aneurysms. *Semin Vasc Surg*. 2005;18(4):196–201.
5. Lumsden AB, Mattar SG, Allen RC, et al. Hepatic artery aneurysms: the management of 22 patients. *J Surg Res*. 1996;60:345–350.
6. Porter L, Houston M, Kadir S. Mycotic aneurysms of the hepatic artery: treatment with arterial embolization. *Am J Med*. 1979;67:697–701.
7. Sidhu M, Shaw D, Daly C, et al. Post-traumatic hepatic pseudoaneurysm in children. *Pediatr Radiol*. 1999;29:46–52.
8. Green MH, Duell RM, Johnson CD, Jamieson NV. *Haemobilia Br J Surg*. 2001;88:773–786.
9. Yoshida J, Donahue PE, Nyhus LM. Hemobilia: review of recent experience with a worldwide problem. *Am J Gastroenterol*. 1987;82:448–455.
10. Merrell SW, Schneider PD. Hemobilia – evolution of current diagnosis and treatment. *West J Med*. 1991;155:621–625.
11. Liu TT, Hou MC, Lin HC, et al. Life-threatening hemobilia caused by hepatic artery pseudoaneurysm: A rare complication of chronic cholangitis. *World J Gastroenterol*. 2003;9:2883–2884.