Transarterial Embolization for Hemobilia Caused by Aneurysm of Hepatic Artery: Report of a case

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Aneurysm of the hepatic artery can rupture into biliary system and cause hemobilia that may be lethal. We here report a case of massive hemobilia caused by a sacular aneurysm of the right hepatic artery that was successfully treated by transarterial embolization. In order to prevent recurrence of the aneurysm, we not only emblolized completely the chamber of aneurysm but also the hepatic arterial lumen around and proximal to the orifice of the aneurysm simultaneously.

Key words: Bile duct; Hepatic arteries, Aneurysm; Therapeutic Embolization

Spontaneous hepatic artery aneurysms are rare. Histologically, mycotic aneurysms were the most common cause, with atherosclerosis being the second [1]. Once ruptured, spontaneous hepatic artery aneurysms can cause massive hemorrhage. They may rupture into the biliary tract and result in hemobilia, presenting with the triad of right upper quadrant colicky pain, obstructive jaundice, and gastrointestinal bleeding. This situation is potentially life-threatening [1-3]. In the last decade, 65% of the reported hepatic artery aneurysms had ruptured, and the mortality rate associated with rupture was 21% [1], hence an aggressive approach to manage these lesions once identified is recommended. We here present an experience using transarterial embolization (TAE) to control hemobilia caused by a hepatic artery aneurysm.

CASE REPORT

An 80-year-old female patient suffered from hematemesis and melena several times in one day and was admitted into our hospital. Hypotension and tachycardia were identified on the arrival at our emergency room. Fluid supplement and blood transfusion were administered immediately.

Tracing the past history, this patient had received operations for dissecting aneurysm of the aortic arch and mycotic aneurysms of jejunum and ileum one and a half year before this admission, respectively. The abnormal laboratory data were hemoglobin 7.3 g/dl, hematocrit 21.9 %, serum albumin 2.4 g/dl, alkaline phosphotase 196 IU/L, GPT 53.3 IU/L and total bilirubin 2.84 mg/dl. The panendoscopic examination on admission revealed blood emanating from the ampula of Vater, and then hemobilia was diagnosed. Ultrasound examination of the liver disclosed a cystic lesion measuring about 2.5 centimeter in diameter in the right lobe of liver, and the Doppler study showed arterial flow in this lesion. Computed tomography (CT) scan revealed dilatation of intrahepatic bile ducts,
and a 3 cm intensely enhanced nodular structure was identified in the right hepatic lobe near the hilar region (Fig 1) corresponding to the lesion noted in the abdominal sonography. According to the ultrasound and CT findings, aneurysm of hepatic artery causing hemobilia was impressed.

Celiac arteriography was performed and a sacular aneurysm arising from one branch of the right hepatic artery was demonstrated (Fig 2a). TAE was requested by the referring physician on account of the high surgical risk. The arterial lumen distal to the orifice of the aneurysm could not be clearly identified due to distortion and vascular spasm of the arteries around the aneurysm. TAE was executed immediately when the informed consent was obtained. After superselective catheterization into the chamber of the sacular aneurysm through its narrow neck arising from superior branch of the right hepatic artery by a 4 French angiocatheter (Cordis, Miami, Fla), multiple metallic coils (Cook, Bloomington, Ind) were deployed into the lumen. When the majority of the chamber of the aneurysm was occupied by the metallic coils, about 1 ml of tissue adhesive (Histoacryl [B Braun, Tuttingen, Germany], 1:1 dilution with lipiodol [Guerbet, Aulnay-sous-Bois, France]) was

Figure 1. The contrast-enhanced CT scan of the liver reveals an intensely enhanced vascular lesion (arrow) measuring about 3 cm in diameter over the central portion of the right lobe of the liver and dilatation of the contiguous intrahepatic bile ducts.

Figure 2. a. The selective common hepatic arteriography discloses an aneurysm (arrow) arising from one branch of the right hepatic artery. b. TAE of the aneurysm itself is performed firstly with multiple metallic coils (arrow) and then injection of tissue adhesive (arrowhead) into the residual lumen between coils for complete obliteration of the aneurysmal lumen. Thereafter, embolization of the hepatic artery around (curved arrow) and proximal (open arrow) to the orifice of the aneurysm is also performed with metallic coils. c. Reconstitution of the hepatic artery distal to the embolization by the intrahepatic collaterals (arrow) can be identified in the follow-up arteriography.
injected into the aneurysm to achieve complete obliteration of the residual lumen of the aneurysm (Fig 2b). Additional TAE of the branch of right hepatic artery where the aneurysm originated was applied in the arterial lumen around and proximal to the orifice of aneurysm with two metallic coils (Fig 2b) for prevention of reconstitution of the aneurysm via the collaterals. The follow-up hepatic arteriography revealed complete obliteration of the lumen of the aneurysm and reconstitution of the distal arterial lumen of the embolized right hepatic artery by several intrahepatic collaterals (Fig 2c).

The hemobilia ceased after TAE treatment with no significant complication. This patient was discharged in stable condition 11 days after TAE. The liver enzyme returned to normal range about one month after treatment. No evidence of dilatation of the intrahepatic ducts was found in the follow-up CT scan performed about four months after TAE (Fig 3). Liver sonography with color Doppler study performed about four years after TAE revealed complete obliteration of the aneurysmal lumen by the echogenic embolizers without detectable color flow signal. During the follow-up period of six years, the patient survived well and did not encounter any recurrent hemorrhage.

**DISCUSSION**

Hemobilia is one of the causes of gastrointestinal bleeding that is often ignored because of its scarcity. Common causes of severe hemobilia are trauma, either iatrogenic or in related to accident, inflammatory processes, gallstones, tumors, and vascular disorders [4]. Vascular disease, which used to be a common cause of hemobilia, is today responsible for only 10% of causes of gross hemobilia [5]. Usually, the bleeding is of arterial nature; a venobiliary communication is an exception [5]. The origins of hemobilia may be diverse and include the cystic artery, anomalous hepatic artery, and hepatic artery to portal vein fistulas [6, 7]. Hepatic artery aneurysm was first reported at autopsy in 1809 [8]. Hepatic artery aneurysms traditionally represent 20% of all visceral aneurysms, and 20% of them are intrahepatic [9].

Endoscopy is the major tool for diagnosis of gastrointestinal hemorrhage, but failure to identify the source of upper gastrointestinal bleeding at endoscopy results in a clinical dilemma. Hence, the endoscopist should pay attention to the papilla of Vater to verify the diagnosis of hemobilia when the bleeding source arising from esophagus, stomach, or duodenum is excluded. Unfortunately, only about 30% of hemobilia could be observed by endoscopic examinations [10].

Ultrasound and CT may contribute to the diagnosis by identification of blood clots in the dilated common bile duct and/or gallbladder [11]. Vascular lesion in the hepatic parenchyma or along the course of common bile duct can also be depicted by Doppler sonography and contrast-enhanced CT as in our case. Angiography is necessary to clarify the nature and location of the source of hemobilia and has the capability of therapeutic intervention [12].

The management of hemobilia is directed at stopping bleeding and relieving biliary obstruction. Surgery for bleeding involves ligation of the bleeding vessel or excision of the aneurysm. When this cannot be achieved, non-selective ligation of the right or left hepatic artery may be performed. If there is concern that the blood supply to the liver will be compromised, arterial reconstruction is indicated, bypassing the affected section of blood vessel using venous autograft or splenohepatic anastomosis [13, 14]. If hemorrhage from collateral vessels continues even after ligation of the common hepatic artery, resection of the affected liver segment may be necessary [15]. These surgical interventions all are associated with high incidence of morbidity or even mortality for patients with unpredictable surgical risk as in our case, so surgery is indicated when other non-operative treatment has failed [3].

In 1976, Walter et al. [16] reported the first successful TAE of a hepatic artery. With recent improvements in selective catheters, TAE is now the first line of intervention to stop bleeding for most causes of hemobilia. TAE involves the selective catheterization of a hepatic artery and embolic occlusion by autologous blood, detachable balloon, metallic coils, tissue
adhesive or Gelfoam. Previous reviews and retrospective series have shown the success rate of TAE to be 80-100 percent [3, 12, 17, 18]. The reported mortality and morbidity rates are lower than those after surgery [3, 18]. Failure may be technical or due to the extensive collateral hepatic blood vessels [19, 20]. Embolization of the whole aneurysmal lumen can get rid of the possibility of refilling of the aneurysm by these intrahepatic collaterals [12]. Nevertheless, the aneurysm will reappear, even partially, on account of insufficient occupation of the aneurysmal lumen by the embolic materials, as was reported by O’Connor et al. [21]. Therefore, highly selective catheterization of the vascular lumen distal to the orifice of the vascular lesion and then embolization from this region to the arterial segment proximal to the orifice of vascular lesion is necessary to prevent the reconstitution of the vascular lesion via the feeding artery antegrade or the abundant collaterals retrogradely. If the aneurysm is refilled by the retrogradely flowing collaterals due to inadequate distal blockage, the hemobilia will recur. In this situation, the next TAE will be technically difficult and associate with increased risk of complication on account of possible damage of more hepatic parenchyma because blockage all of the visible collaterals is inevitable. Consequently, blocking the arterial lumen distal and proximal to the identified orifice of vascular lesion simultaneously is mandatory to secure the result of TAE. However, if the arterial lumen distal to the aneurysm cannot be identified or reached, embolization of the aneurysm itself with additional blockage of the arterial lumen about and proximal to the orifice of the vascular lesion, just as our case, is necessary to prevent refilling of the aneurysmal lumen by the unrecognized terminal artery distal to the aneurysm reconstituted by the intrahepatic collaterals. In our case, this method of TAE secures the effect of hemostasis and prevents recurrent hemorrhage, and no complication occurs after TAE.

Many embolic materials have been used in TAE for vascular lesions. Permanent embolizers are recommended for secure obliteration and avoidance of recanalization of the vascular lesion. Metallic coils are designed in different sizes and shapes for usage in diverse situations. Nevertheless, residual vascular chamber that is not completely occupied by the metallic coils, particularly in a huge aneurysm with usage of multiple coils, will appear. As mentioned before, hemobilia may recur from this residual vascular lesion. In order to block the aneurysmal lumen as completely as possible, we introduced tissue adhesive, a kind of fluid embolizer, into the residual chamber of the vascular lesion when multiple metallic coils were deployed and the risk of rupture of aneurysmal wall increased with insertion of additional coils. However, additional embolization of the hepatic artery around and proximal to the neck of the aneurysm by metallic coils was also performed in our case for eradication of any possibility of recurrence of the aneurysm. Using metallic coils for blockage of the hepatic artery, the arterial lumen distal to the placed metallic coils can be reconstituted by the abundant collaterals, and this may obviate the ischemia of hepatic parenchyma in the territory of the embolized hepatic artery.

In conclusion, transarterial embolization is an effective way to control hemobilia caused by aneurysm of hepatic artery. Embolization of the aneurysmal lumen as completely as possible and blockage of the hepatic arterial channel from the neck of the vascular lesion are mandatory to prevent recurrent hemorrhage.

**REFERENCES**

8. Guida PM, Moore SW. Aneurysm of the hepatic artery: report of five cases with a brief review of previously reported cases. Surgery 1966; 60: 299-310.
13. Goodnight JE, Blaisdell FW. Hemobilia. Surg Clin...


以經動脈栓塞術治療因肝動脈瘤引起之膽道出血：
一病例報告

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肝動脈瘤可能會破裂至膽道系統而引起致命的膽道出血。我們在此報告一成功地以經動脈栓塞術治療因囊狀右肝動脈瘤造成大量膽道出血的病例；為了避免動脈瘤復發，我們不僅栓塞動脈瘤本身，同時也將支配動脈瘤的肝動脈從動脈瘤入口處至其近端栓塞住。

關鍵詞：膽道；動脈瘤；栓塞術