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RAPID COMMUNICATION

Extrahepatic collaterals and liver damage in embolotherapy for ruptured hepatic artery pseudoaneurysm following hepatobiliary pancreatic surgery

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Abstract

AIM: To evaluate the effects of extrahepatic collaterals to the liver on liver damage and patient outcome after embolotherapy for the ruptured hepatic artery pseudoaneurysm following hepatobiliary pancreatic surgery.

METHODS: We reviewed 9 patients who underwent transcatheter arterial embolization (TAE) for the ruptured hepatic artery pseudoaneurysm following major hepatobiliary pancreatic surgery between June 1992 and April 2006. We paid special attention to the extrahepatic arterial collaterals to the liver which may affect post-TAE liver damage and patient outcome.

RESULTS: The underlying diseases were all malignancies, and the surgical procedures included hepatopancreatoduodenectomy in 2 patients, hepatic resection with removal of the bile duct in 5, and pancreaticoduodenectomy in 2. A total of 11 pseudoaneurysm developed: 4 in the common hepatic artery, 4 in the proper hepatic artery, and 3 in the right hepatic artery. Successful hemostasis was accomplished with the initial TAE in all patients, except for 1. Extrahepatic arterial pathways to the liver, including the right inferior phrenic artery, the jejunal branches, and the aberrant left hepatic artery, were identified in 8 of the 9 patients after the completion of TAE. The development of collaterals depended on the extent of liver mobilization during the hepatic resection, the postoperative period, the presence or absence of an aberrant left hepatic artery, and the concomitant arterial stenosis adjacent to the pseudoaneurysm. The liver tolerated TAE without significant consequences when at least one of the collaterals from the inferior phrenic artery or the aberrant left hepatic artery was present. One patient, however, with no extrahepatic collaterals died of liver failure due to total liver necrosis 9 d after TAE.

CONCLUSION: When TAE is performed on ruptured hepatic artery pseudoaneurysm, reduced collateral pathways to the liver created by the primary surgical procedure and a short postoperative interval may lead to an unfavorable outcome.

Key words: Hepatic artery pseudoaneurysm; Transcatheter arterial embolization; Extrahepatic collateral pathways; Liver damage; Hepatobiliary pancreatic surgery

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INTRODUCTION

Delayed massive arterial bleeding in the postoperative period is a rare complication for patients with hepatobiliary pancreatic diseases, but often results in a lethal outcome due to the abrupt onset of massive hemorrhage. Bleeding occurs mainly from erosion or pseudoaneurysm of major visceral arteries as a result of an adjacent septic condition or an intraoperative arterial injury[1-3].

Transcatheter arterial embolization (TAE) has been proposed as the first-line treatment to control massive bleeding associated with a ruptured hepatic artery pseudoaneurysm after hepatobiliary pancreatic surgery because of its advantages over surgery. Such advantages include an easier approach, precise localization of the pseudoaneurysm, assessment of collateral pathways to the liver, less chance of re-bleeding, and low mortality rates[1-7]. Meanwhile, TAE on the hepatic artery may lead to liver abscesses, gallbladder necrosis, biliary stricture, intrahepatic biloma, and embolization of normal vessels[8-10]. Furthermore, hepatic TAE may cause a total interruption of the arterial blood supply to the liver, thus

presenting a risk for liver infarction, especially in patients after right or left hepatic lobectomy[11-14].

In this study, we retrospectively reviewed the patients who underwent TAE for a ruptured hepatic artery pseudoaneurysm following major hepatobiliary pancreatic surgery, paying special attention to the extrahepatic collateral pathways which may be associated with post-TAE liver damage and a negative patient outcome.

MATERIALS AND METHODS

Between June 1992 and April 2006, 10 patients developed a rupture of the hepatic artery pseudoaneurysm following major hepatobiliary pancreatic surgery in our department. This included 1 patient who suddenly died due to abrupt massive hemorrhaging. The remaining 9 patients who underwent a diagnostic angiography and subsequent transcatheter hemostasis treatment were reviewed in this study. The clinical characteristics of these 9 patients are summarized in Table 1. These patients included 6 men and 3 women with a mean age of 70 years (range 63-75). A total of 11 pseudoaneurysms developed: 4 in the common hepatic artery, 4 in the proper hepatic artery, and 3 in the right hepatic artery. Subsequent ruptures of the portal vein were seen in 2 patients. The underlying diseases included hilar bile duct carcinoma, ampullary carcinoma, gallbladder carcinoma, intrahepatic cholangiocarcinoma, and metastatic liver cancer from colon cancer.

The surgical procedures performed were hepatopancreatoduodenectomy in 2 patients, left hepatic lobectomy with a resection of the caudate lobe and bile duct in 4, a segmental resection of the liver with removal of the bile duct in 1, and pylorus-preserving or conventional pancreaticoduodenectomy in 2. All patients received lymphadenectomy around the hepatic artery. During the hepatic resection, the whole liver was mobilized from the diaphragm for the surgical procedure in 2 patients, and a unilateral mobilization of the liver was done in 4 patients. The postoperative complications included hepaticojejunal or pancreaticojejunal anastomotic leakage in 6 patients. The bleeding from ruptured pseudoaneurysm occurred 12-256 d following the primary operation. During the follow-up, 2 patients (cases no.1 and 2) had melena or hematemesis. Of the remaining 7 patients, 6 presented bleeding from the drain in the abdominal cavity, which was preceded by an intermittent minor hemorrhage or "sentinel bleed" in 5 of the cases. A contrast-enhanced abdominal CT scan was carried out immediately after the initial "sentinel bleed" in 2 patients, and both depicted pseudoaneurysm in the hepatic artery (Figure 1). In addition, hemorrhagic shock (systolic blood pressure < 90 mmHg, pulse rate > 100/min) was presented in 6 of the patients.

The diagnostic angiography was performed using a 4-Fr or 5-Fr angiographic catheter (Clinical Supply, Tokyo, Japan) through a transfemoral approach. In all cases, celiac, common hepatic, and superior mesenteric angiographies and arterial portographies, were routinely taken to detect the bleeding points. Immediately after identifying pseudoaneurysms, with or without extravasation of the contrast madia, TAE was performed with distal and proximal placement of embolic coils to the pseudoaneurysm for the hemostasis. The embolic coils used were 0.035-inch stainless-steel coils, 3-8 mm in diameter, or 0.018-inch platinum coils, and 3-6 mm in diameter (Cook, Bloomington, IN).

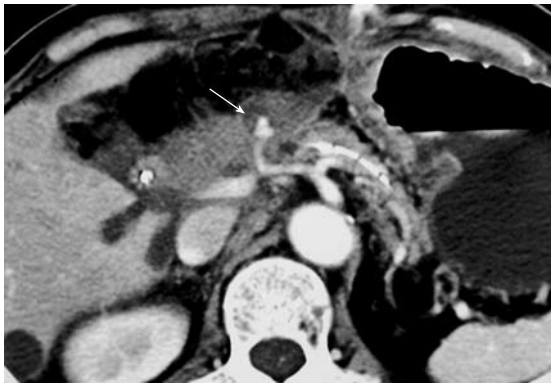

Figure 1 A 66-year-old female with pylorus-preserving pancreaticoduodenectomy (12 d after surgery). A contrast-enhanced CT scan presents a pseudoaneurysm on the distal side of the common hepatic artery (arrow).

The procedural success of TAE and the presence of collateral arterial blood flow to the liver were confirmed at the end of the procedure by performing angiographies of the celiac, superior mesenteric, and inferior phrenic arteries. The hepatic function tests evaluated before and after TAE included total bilirubin (T-Bil), aspartate aminotransferase (AST), and alanine aminotranferease (ALT). We retrospectively reviewed the 9 patients with special attention paid to the impact of collateral pathways to the liver on post-TAE complications and patient outcome.

RESULTS

Embolization of the hepatic artery was done in the extrahepatic level in all patients (Table 2). However, the embolic coils migrated to the intrahepatic level of the left hepatic artery in 2 patients. An immediate successful hemostasis was accomplished during the initial TAE in all cases, except for 1 patient (case no. 3) who required a second TAE because of re-bleeding 1 hour after the first procedure. The first TAE was performed at the proximal side of the right hepatic artery where pseudoaneurysm and extravasation were observed. The second TAE was performed by adding the coils distally and proximally.

Angiographies after the completion of the TAE showed extrahepatic collateral pathways to the liver in 8 of 9 patients. This consisted of the right inferior phrenic artery in 6 cases (Figure 2), the jejunal branches in 2 (Figure 3), and the aberrant left hepatic artery branched from the left gastric artery in the other 2. These collateral pathways supply the arterial flow throughout the hepatic lobes via intrahepatic communicating branches (Figure 4). Among these extrahepatic collaterals, the main collateral pathways to the liver were either the right inferior phrenic artery or the aberrant left hepatic artery. In 5 patients who underwent a hepatic resection without whole liver mobilization, the collateral pathways from the right inferior phrenic artery were well developed. In 2

Table 1 Clinical characteristics of patients with ruptured hepatic artery pseudoaneurysms after hepatobiliary pancreatic surgery

Case no.	Age/ sex	Underlying diseases	Surgical procedures	Hepatic lobes mobilized	Postoperative complications	Postoperative interval (d)	Clinical presentation	Sentinel bleed	Pseudoaneurysm		
									Location	size (cm)	Extra-vasation
1	75/M	Metastatic liver cancer	HTX (S7 + 8), BDR	R	None	256	Melena	No	PHA	2.7	Yes
2	74/M	Intrahepatic cholangiocarcinoma	HTX (S1 + 2 + 3 + 4), BDR	R&L	HJ leak	105	Hematemesis, shock	No	PHA	0.7	Yes
3	66/M	Hilar bile duct carcinoma	HTX (S1 + 2 + 3 + 4), PD	R&L	HJ leak	28	BFD, shock	Yes	PHA, RHA	2.2, 3.1	Yes
4	71/M	Hilar bile duct carcinoma	HTX (S1 + 2 + 3 + 4), BDR	L	HJ leak	19	BFD, shock	Yes	RHA	2.8	Yes
5	76/F	Intrahepatic cholangiocarcinoma	HTX (S1 + 2 + 3 + 4), BDR	L	None	17	Hematemesis, shock	No	PHA	1.5	Yes
6	72/M	Hilar bile duct carcinoma	HTX (S1 + 2 + 3 + 4), BDR	L	HJ leak	15	BFD	Yes	CHA	1.3	No
7	67/F	Gallbladder carcinoma	HTX (S4a + 5), PD	None	PJ leak	35	BFD	No	CHA	1.2	Yes
8	63/M	Ampullary carcinoma	PD	None	PJ leak	23	BFD, shock	Yes	CHA, RHA	1.5, 2.3	Yes
9	66/F	Ampullary carcinoma	PPPD	None	None	12	BFD, shock	Yes	CHA	0.8	No

HTX: hepatectomy; BDR: bile duct resection; S7 + 8: hepatic posterior inferior and superior segments; S1 + 2 + 3 + 4: caudate lobe and hepatic lateral and medial segments; S4a + 5: hepatic medial inferior and anteror inferior segments; PD: pancreaticoduodenectomy; PPPD: pylorus-preserving pancreaticoduodenectomy; R: right; L: left; HJ: hepaticojejunal anastomosis; PJ: pancreaticojejunal anastomosis; BFD: bleeding from drain; PHA: proper hepatic artery; RHA: right hepatic artery; CHA: common hepatic artery.

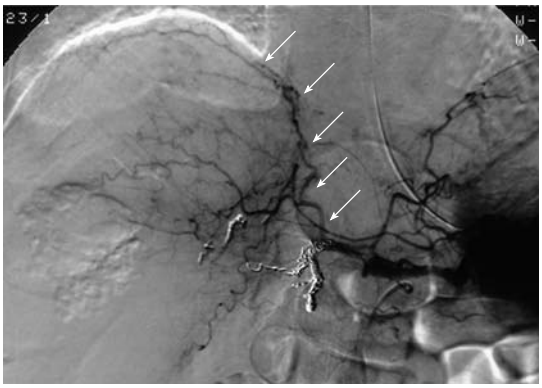

Figure 2 A 74-year-old male with left hepatic lobectomy (105 d after surgery). The right inferior phrenic artery (arrows) is presented as a collateral pathway to the liver on an angiography after TAE for the proper hepatic and gastroduodenal arteries.

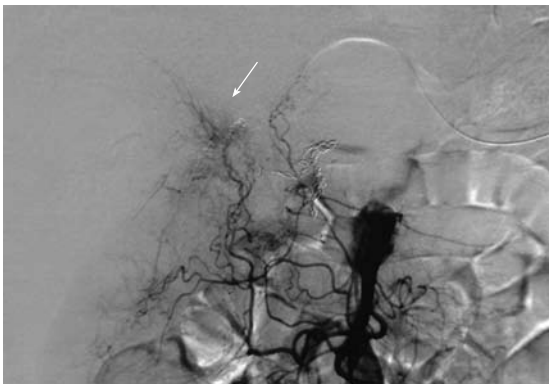

Figure 3 The jejunal branches (arrow) are seen as a collateral pathway to the liver in the same patient as in Figure 2.

patients who received whole liver mobilization from the diaphragm during the hepatic resection, 1 patient (case no. 2) presented a well-developed collateral pathway from the right inferior phrenic artery, but the other (case no. 3) demonstrated no collateral pathways. The postoperative interval was 105 d and 28 d, respectively. Collateral pathways from the jejunal branches were seen in 2 patients who had a long postoperative course (cases no. 1 and 2), meanwhile, arterial stenosis or spasm adjacent to the pseudoaneurysm were identified in 6 patients. The extra- and intrahepatic arterial pathways to the liver developed in accordance with the degree of arterial stenosis (Figure 5).

One patient with no extrahepatic collateral pathways to the liver died of liver failure due to total liver necrosis 9 d after TAE. However, 8 patients with extrahepatic collateral pathways tolerated TAE. Six of these 8 patients showed mild to moderate elevations of T-Bil, AST, and ALT after TAE, and liver functions returned to normal within 10 d without any complication. The remaining 2 patients (cases no. 7 and 8) showed a considerable liver dysfunction likely due to a chance migration of embolic coils to the left hepatic artery during TAE. They were subsequently cured with a conservative treatment, although 1 patient developed a liver abscess in the left hepatic lobe.

Two patients who developed a rupture of the portal vein after TAE were managed with re-laparotomy (case no. 2) and interventional radiologic technique (case no. 6), respectively. In the former, the rupture of the portal vein occurred 37 d after TAE, in which the embolic coils in the proper hepatic artery migrated to the adjacent portal vein, resulting in disruption of the vein. Although this patient underwent re-laparotomy, the ruptured portal vein could not be repaired due to huge adhesions around the portal system and the fragility of the portal vein. He died of multiple organ failure (MOF) 14 d after re-laparotomy. In the latter patient, the bleeding from the portal vein

Table 2 Results of embolotherapy for ruptured hepatic artery pseudoaneurysms after hepatobiliary pancreatic surgery

Case no.	Arteries embolized	No.of coils placed	Migration of coils	TAE success	Collateral pathways				Hepatic arterial stenosis	Changes in liver function tests: pre-TAE→post-TAE (days after TAE)			Follow-up interval after TAE	Outcome
					RIPA	Jejunal branches	aberrant LHA	intrahepatic branches		T-Bil (mg/dL)	AST (IU/L)	ALT (IU/L)		
1	PHA	17	No	Complete	Good	Good	None	Fair	Yes	1.2→3.2 (3)	29→131 (3)	22→112 (3)	34 mo	Alive
2	PHA, GDA	18	No	Complete	Good	Good	None	Good	Yes	1.1→1.3 (5)	26→41 (1)	12→16 (1)	52 d	Died of MOF
3	PHA, RHA	13	No	Complete	Poor	Poor	None	?	No	1.3→19.4 (3)	65→9750 (2)	79→7865 (3)	9 d	Died of liver failure
4	RHA	10	No	Complete	Good	Poor	None	Good	Yes	1.7→5.6 (1)	46→214 (1)	34→132 (1)	7 mo	Died of local recurrence
5	PHA	9	No	Complete	Fair	Poor	None	Fair	No	0.9→2.5 (5)	20→141 (4)	24→289 (4)	6 mo	Alive
6	CHA	12	No	Complete	Good	Poor	None	Good	Yes	1.6→6.2 (3)	22→118 (3)	21→95 (4)	54 d	Died of MOF
7	CHA	6	Yes	Complete	Fair	Poor	None	Fair	No	1.2→3.2 (3)	29→965 (3)	22→947 (3)	9 mo	Died of liver metastasis
8	CHA, RHA	13	Yes	Complete	Poor	Poor	Good	Good	Yes	0.6→1.1 (5)	38→439 (1)	57→700 (2)	48 mo	Alive
9	CHA	22	No	Complete	Poor	Poor	Good	Good	Yes	0.5→2.2 (3)	20→144 (1)	48→241 (1)	58 mo	Alive

PHA: proper hepatic artery; DGA: gastroduodenal artery; RHA: right hepatic artery; CHA: common hepatic artery; RIPA: right inferior phrenic artery; LHA: left hepatic artery; T-Bil: total bilirubin; AST: aspartate aminotransferase; ALT: alanine aminotranferease; MOF: multiple organ failure.

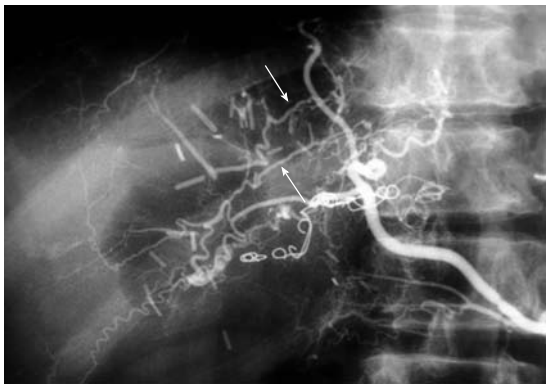

Figure 4 A 71-year-old male with left hepatic lobectomy (41 d after surgery). The intrahepatic communicating branches (arrows) are visualized via the branches of the right inferior phrenic artery on an angiography after TAE for the right hepatic artery.

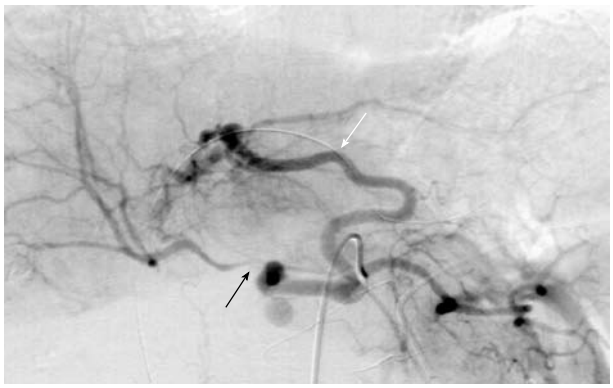

Figure 5 A diagnostic celiac angiography showing a stenosis of the right hepatic artery (black arrow) distal to the ruptured hepatic artery pseudoaneurysm in the same patient as in Figure 1. The aberrant left hepatic artery (white arrow) originating from the left gastric artery was dilated and supplied arterial blood flow to the right hepatic artery through the intrahepatic communicating branches before TAE.

occurred 3 d after TAE. A Gianturco expandable metallic stent (Z-stent; Cook, Bloomington, IN) was placed in the portal vein to bridge the site of venous disruption via a peripheral branch of the ileocecal vein under a small laparotomy in the right lower quadrant of the abdomen. The extra-portal cavity was then packed with 30 microcoils through a slit in the Z-stent. This patient died of MOF induced by sepsis originating from the abscess around the microcoils packed in the extra-portal cavity 51 d after placement of the stent.

DISCUSSION

Massive arterial bleeding can occur early or late in the postoperative course of patients undergoing hepatobiliary pancreatic surgery. Early bleeding within 24 h after operation usually originates from vessels at the anastomotic suture-line or in the intra-abdominal cavity due to inadequate hemostasis[15,16]. On the other hand, delayed bleeding occurs mainly due to erosion or pseudoaneurysm of major visceral arteries and has a higher mortality rate than those with suture-line bleeding[16].

Three major predisposing factors for delayed arterial bleeding after hepatobiliary pancreatic surgery have been suggested: (1) digestion of arterial walls due to activated pancreatic juice or infectious bile from anastomotic insufficiency, (2) arterial irritation by localized abscess in the intra-abdominal space, and (3) a mechanical injury of the artery during operation, particularly due to lymph node dissection for malignancy[1-3,14,17,18]. In the present study, lymphadenectomy around the hepatic artery was performed on all 9 patients. In addition, postoperative hepaticojejunal or pancreaticojejunal anastomotic leakage was identified in 6 cases. Although the rupture of visceral arteries after surgery may be a complex process and actual pathogenesis remains obscure, histopathology of autopsy

of our case no. 3 demonstrated that infectious bile invaded the hepatic arterial wall and completely destroyed the media consisting of elastic fibers[11].

The compensatory arterial circulation to the liver after TAE for the treatment of ruptured hepatic artery pseudoaneurysm was recognized in 8 of the 9 patients who underwent major hepatobiliary pancreatic surgery in this study. Michels has described the details of extrahepatic arterial collaterals to the liver after ligation of the hepatic artery in a series of 200 cases of hepatic resection, in which at least 26 possible routes of collateral arterial blood supply to the liver from the common hepatic trunk were noted[19]. Among them, the following are clinically important: (1) the subphrenic branch of the phrenic artery or internal mammary artery, (2) the aberrant hepatic artery, (3) the periductal arterial plexus, (4) other collaterals into the hepatic hilum from the superior mesenteric artery, and (5) the intrahepatic branches[19,20]. Although the significance of extrahepatic collateral pathways in association with liver damage after hepatic TAE has been reported[2,21-23], the relationship between the primary surgical procedure and the development of extrahepatic collaterals following hepatobiliary pancreatic surgery has not been described in detail.

In this study, the main collateral pathways to the liver after TAE in patients with major hepatobiliary pancreatic surgery included the right inferior phrenic artery, the aberrant left hepatic artery, and the intrahepatic communicating branches. The development of these collaterals depended on the primary operative procedure, postoperative duration, and concurrent arterial stenosis adjacent to the pseudoaneurysm. They subsequently affected post-TAE liver damage and patient outcome. Following TAE, total liver necrosis occurred in 1 patient who had no extrahepatic collateral pathways. This was probably due to whole liver mobilization during hepat opancreatoduodenectomy 28 d prior to TAE. Another patient received whole liver mobilization along with left hepatic lobectomy 105 d prior to TAE, and showed a well-developed collateral pathway from the right inferior phrenic artery. Conversely, the inferior phrenic artery was maintained in 5 patients who underwent a hepatic resection without whole liver mobilization. The hepatic arterial stenosis adjacent to the pseudoaneurysm was also associated with the development of extra- and intrahepatic collateral pathways. As a result, the liver tolerated TAE without significant consequences when one of the collaterals from the inferior phrenic artery or the aberrant left hepatic artery was present. Therefore, when performing hepatic TAE, all the information mentioned above should be considered in individual patients who have undergone hepatobiliary pancreatic surgery. TAE on the hepatic artery has generally been considered to be relatively safe if the portal blood flow is maintained[12,13,24]. However, it could result in an unfavorable outcome when the development of extrahepatic pathways to the liver is poor. In such cases, endovascular repair of hepatic artery pseudoaneurysms using a stent-graft with preservation of the arterial blood flow to the liver[25], may be an alternative treatment to TAE.

Brodsky and Turnbull[26] emphasize a "sentinel bleeding" as a precursor of vessel erosion after pancreatic surgery. In our series, episodes of sentinel bleeding from the drainage tube prior to massive hemorrhage were recognized in 5 patients. Interestingly, an urgent contrast-enhanced CT scan successfully depicted hepatic artery pseudoaneurysm in 2 patients when the abdominal CT scan was carried out immediately after the initial sentinel bleeding. The significance of a CT scan in the detection of visceral pseudoaneurysm following pancreaticoduodenectomy has previously been reported[1,27]. Moreover, a dilated right inferior phrenic artery representing an extrahepatic collateral after TAE for hepatocellular carcinoma has been demonstrated by using biphasic helical CT scans[28]. Early diagnosis and management of an arterial pseudoaneurysm before it ruptures is essential to improve the prognosis of this condition. Accordingly, early CT scan should be required in patients associated with postoperative leakage of enteric anastomosis or intra-abdominal abscess, as well as in cases with a sentinel bleeding after surgery.

Subsequent ruptures of the portal vein were seen in 2 patients. Although the Z-stent placement in the portal vein was effective in the hemostasis of 1 case, the patient finally died of MOF induced by sepsis originating from abscess around the microcoils packed in the extra-portal cavity. In such a case, a "second-look" operation for the abscess should be performed after the hemodynamic condition of the patient becomes stable.

In conclusion, TAE should be the first choice of treatment for ruptured hepatic artery pseudoaneurysms after hepatobiliary pancreatic surgery. In performing hepatic TAE, however, information on liver mobilization, the postoperative period, the presence or absence of an aberrant left hepatic artery, and concomitant arterial stenosis adjacent to the pseudoaneurysm is of prime importance due to these factors affecting the development of extrahepatic collateral pathways to the liver and the post-TAE patient outcome.

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