Table 1 Pancreatitis bundles

1. When a diagnosis of acute pancreatitis has been made, repeated severity assessment should be carried out within 24 h, and 24–48 h after diagnosis on the basis of estimating the severity assessment criteria of acute pancreatitis prepared by the Ministry of Health, Labour and Welfare.
2. For patients with severe acute pancreatitis, transference to an appropriate medical facility should be considered within 3 h after a diagnosis has been made.
3. For patients with acute pancreatitis, causes of pancreatitis should be differentiated using medical records, hematological examination and imaging studies.
4. For gallstone-induced pancreatitis, early ERC + ES should be considered in those patients in whom cholangitis is accompanied and/or prolonged passage disorder of the biliary tract including occurrence or aggravation of jaundice are suspected.
5. At a medical facility where treatment for severe acute pancreatitis is performed, abdominal enhanced CT studies should be performed within 3 h after initial treatment. An unenhanced area and the extent of the disease should be examined and the severity should be assessed on the basis of the CT grade of acute pancreatitis by the Ministry of Health, Labour and Welfare.
6. For acute pancreatitis, sufficient amount of fluid replacement and monitoring should be performed, and mean arterial pressure (MAP)[a] should be maintained >65 mmHg and urinary output >0.5 ml/kg/h, respectively.
7. Pain control should be conducted for acute pancreatitis.
8. Prophylactic wide-spectrum antibiotics should be administered for severe acute pancreatitis within 24 h after onset.
9. In the case of severe acute pancreatitis, doctors should inscribe an application form for public payment should be given to the proxy of the patient as soon as possible (within 2 days) after a diagnosis of acute pancreatitis has been made.
10. Cholecystectomy should be performed after resolution of symptoms of pancreatitis for gallstone-induced pancreatitis accompanied by cholecystolithiasis.

[a] Mean arterial pressure (MAP) = diastolic pressure + (systolic pressure − diastolic pressure)/3

they can be altered to some extent so that the contents may be adjusted to the conditions of the individual institutions, but all the items listed below should be included. Except in special situations, it is desirable that all the items are carried out and recorded in medical records.

The efficacy and significance of achieving these items should be eventually validated, but assessment of the compliance rate with the guidelines as well as with the guidelines and with the pancreatitis bundles themselves becomes possible through pancreatitis bundles.

Items of pancreatitis bundles are described in Table 1.

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8TH WORLD CONGRESS ON TRAUMA, SHOCK, INFLAMMATION AND SEPSIS TSIS 2010

SELECTED PAPERS

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Difference between Tokyo Guidelines and Japanese Guidelines of acute cholangitis and cholecystitis

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Summary

Japanese Guidelines (JGL) and International Guidelines (Tokyo Guidelines: TGL) for the management of acute cholangitis and cholecystitis were published in September 2005, and in January 2007, respectively. Diagnosis and severity criteria of acute cholangitis and cholecystitis are defined for the first time in the world by these two Guidelines. Since there are many differences between the two Guidelines, these should be validated using retrospective and prospective clinical data.

Introduction

Japanese Guidelines (JGL) for the management of acute cholangitis and cholecystitis were published in September 2005[1)], and International Guidelines (Tokyo Guidelines: TGL) for the management of the diseases were published in January 2007[2-4)]. Diagnosis and severity criteria of acute cholangitis and cholecystitis are defined for the first time in the world. But there are many differences between the two guidelines.

Code: **292**

Materials and methods

Here, we elucidate the differences of the diagnosis and severity criteria of acute cholangitis and cholecystitis of these two Guidelines.

Results

Many differences exist between TGL and JGL. Diagnosis criteria of both acute cholangitis and cholecystitis of the two Guidelines are different (Table 1-4) [1-4].Moderate acute cholangitis and cholecystitis of TGL is defined as the disease that does

A. Clinical context and clinical manifestations	1	History of biliary disease
	2	Fever and/or chills
	3	Jaundice
	4	Abdominal pain (RUQ or upper abdominal)
B. Laboratory data	5	Evidence of inflammatory response [a]
	6	Abnormal liver function tests [b]
C. Imaging findings	7	Biliary dilatation, or evidence of an etiology (stricture, stone, stent, etc)
Suspected diagnosis	Two or more items in A	
Definite diagnosis	Charcot's triad (2+3+4)	
	Two or more items in A + both items in B and item C	

[a] Abnormal WBC count, increased of serum CRP level, and other changes indicating inflammation, [b] Increased serum ALP, γ-GTP(GGT) ,AST, and ALT levels

Tab. 1 - Tokyo Guidelines (TGL) diagnostic criteria for Acute Cholangitis.

A	1	Fever
	2	Abdominal pain (RUQ pain and upper abdominal)
	3	Jaundice
B	4	Increased serum ALP, γ-GTP
	5	Increased WBC count, serum CRP level
	6	Imaging findings (Biliary dilatation, stricture and stone)
Suspected diagnosis	one item in A + two or more items in B	
Definite diagnosis	Charcot's triad (1+2+3)	
	one item in A + all items in B	

Tab. 2 - Japanese Guidelines (JGL) diagnostic criteria for Acute Cholangitis.

A. Local signs of inflammation etc.	1	Murphy's sign
	2	RUQ mass/pain/tenderness
B. Systemic signs of inflammation etc.	1	Fever
	2	Elevated CRP
	3	Elevated WBC count
C. Imaging findings	Imaging findings characteristic of acute cholecystitis	
Definite diagnosis	One item in A and one item in B are positive	
	C confirms the diagnosis when acute cholecystitis is suspected clinically	

Note: acute hepatitis, other acute abdominal disease, and chronic cholecystitis should be excluded

Tab. 3 - Tokyo Guidelines (TGL) diagnostic criteria for Acute Cholecystitis.

A.	RUQ pain and epigastralgia
	Tenderness
	Muscle Defense
	Murphy sign
B	Fever
	Increased WBC count or serum CRP level
C	Imaging findings [a]
Suspected diagnosis	one item in A + one item in B
Definite diagnosis	Suspected diagnosis (one item in A + one item in B) + C

[a] US: sonographic Murphy's sign, thickened GB wall, enlarged GB, Incarcerated gallstone, Sonolucent layer in GB wall etc)

Tab. 4 - Japanese Guidelines (JGL) diagnostic criteria for Acute Cholecystitis.

not respond to the initial medical treatment, whereas, that of TGL is defined as abnormality of laboratory data and image findings. Therefore, treatment strategies of the diseases are also different, although both Guidelines recommend that therapies of the diseases depend on the severity of the diseases.

Conclusions

The diagnosis and severity criteria are defined based on expert's opinions. Therefore, we need verify these Guidelines using retrospective and prospective clinical data. To evaluate these Guidelines, CLASS Tokyo Study (prospective registry of acute cholangitis) has been began (http://class.umin.jp/english/index.html).

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SELECTED PAPERS

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The Clinical Evaluation of the Tokyo Guidelines 2007 Based on Clinical Cases

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Summary

"Tokyo Guidelines (TGL)", the world's first guidelines for biliary tract inflammation, was published in 2007. We have to evaluate TGL in order to ensure the accuracy on actual cases. Using TGL and Japanese Domestic Guidelines (JGL), we reviewed 74 cases of cholangitis, and 81 cases of cholecystitis that were clinically diagnosed by physician as an initial diagnosis on admission. On this article, we especially focused on 4 cases that had different diagnosis between TGL and JGL. On acute cholangitis, the difference of both guidelines is value of Charcot's triad. And on acute cholecystitis, the difference of both guidelines is based on the range of abdominal pain (ex. RUQ only or not)

Introduction

There were no evidenced-based-criteria for the diagnosis, severity assessment, of treatment of acute cholecystitis or acute cholangitis[1)], before publication of JGL and TGL. 2 years have passed after publication of TGL, we have to evaluate TGL to ensure the accuracy on actual cases. We retrospectively reviewed our biliary tract inflammation cases.

Materials and methods

74 Acute cholangitis cases and 81 Acute cholecystitis cases that were clinically

Code: 261

diagnosed as an initial diagnosis on admission during the period from Nov.2004 to Nov. 2005. We applied TGL & JGL diagnostic criterion.

Results

Acute cholangitis (M:F = 39:35, 69.2±15.2yo) TGL: Suspected diagnosis = 9cases(12.2%), Definite diagnosis = 43cases(58.1%). JGL: Suspected diagnosis = 30cases(40.5%), Definite diagnosis = 30cases(40.5%).

Case1:TGL = Definite, JGL = Suspected 74y.o. Male. Only epigastralgia(no RUQ pain) and 5 times vomit. He went to clinic and took CT scan and EGD, but origin wasn't clear. He was transferred. No fever. WBC13400, ALT64, ALP314, GGT92, T-Bil 2.02, CRP1.08, GB stone only.

Case2:TGL = not match, JGL = Definite 81y.o. Female. Vomited several times, and abdominal distention. She came to our ER. No abdominal pain and high fever. WBC13300, ALT250, ALP1468, GGT330, T-Bil5.98, CRP23.61, GB stones and CBD stones were pointed out.

Acute cholecystitis (M:F = 49:32, 69.0±15.0yo) TGL: Definite diagnosis = 66cases(81.5%). JGL: Suspected diagnosis = 14cases(17.3%), Definite diagnosis = 56cases(69.1%).

Case3: TGL = definite, JGL = Suspected 66y.o. Female. 10years ago, GB stone was pointed out. In July, after dinner, she had RUQ pain and came to ER. RUQ tenderness(+), no fever. WBC2100, AST594, ALT336, ALP813, GGT361, T-bil 1.80, CRP1.32, GB stone & CBD stones: not detected.

Case4: TGL = not match, JGL = Definite 67y.o. Male. He had fever and epigas-

TGL : Acute Cholangitis Diagnostic Criteria			case1	case2
A. Clinical context and clinical manifestations	1	History of biliary disease	No	No
	2	Fever and/or chills	No	No
	3	Jaundice	Yes	Yes
	4	Abdominal pain (RUQ or upper abdominal)	Yes	No
B. Laboratory data	5	Evidence of inflammatory response [a]	Yes	Yes
	6	Abnormal liver function tests [b]	Yes	Yes
C. Imaging findings	7	Biliary dilatation, or evidence of an etiology	No	Yes
Suspected diagnosis	Two or more items in A			
Definite diagnosis	Charcot's triad (2+3+4)			
	2 or more items in A + both items in B and item C		Yes	

Tab. 1 - TGL diagnostic criteria for Acute Cholangitis (Diagnosis of Case 1&2).

JGL: Acute Cholangitis Diagnostic Criteria			case1	case2
A	1	Fever	No	No
	2	Abdominal pain (RUQ pain and upper abdominal)	Yes	No
	3	Jaundice	Yes	Yes
B	4	Increased serum ALP, γ-GTP	Half	Yes
	5	Increased WBC count, serum CRP level	Yes	Yes
	6	Imaging findings (Biliary dilatation, stricture and stone)	No	Yes
Suspected		one item in A + two or more items in B	Yes	
Definite diagnosis		Charcot's triad (1+2+3)		
		one item in A + all items in B		Yes

Tab. 2 - JGL diagnostic criteria for Acute Cholangitis (Diagnosis of Case 1&2).

TGL : Acute Cholecystitis Diagnostic Criteria			Case3	Case4
A. Local signs of inflammation etc.	1	Murphy's sign	No	No
	2	RUQ mass/pain/tenderness	Yes	No
B. Systemic signs of inflammation etc.	1	Fever	No	Yes
	2	Elevated CRP	Yes	Yes
	3	Elevated WBC count	No	Yes
C. Imaging findings		Imaging findings characteristic of acute cholecystitis	No	Yes
Definite diagnosis		One item in A and one item in B are positive	Yes	
		C confirms the diagnosis when acute cholecystitis is suspected clinically		

Tab. 3 - TGLdiagnostic criteria for Acute Cholecystitis (Diagnosis of Case 3&4).

tralgia (not RUQ pain). WBC15600, ALP311, GGT620, T-Bil 6.23, CRP9.76, CT scan showed GB stone and GB wall thickness.

Conclusions

74 cholangitis and 81 cholecystitis cases were retrospectively reviwed. We introduced discrepancies of these diagnoses between the guidelines on actual clinical cases.

JGL: Acute Cholecystitis Diagnostic Criteria		case3	case4
A.	RUQ pain and epigastralgia, Tenderness, Muscle Defense, Murphy sign	Yes	Yes
B	Fever	No	Yes
	Increased WBC count or serum CRP level	Yes	Yes
C	Imaging findings [a]	No	Yes
Suspected diagnosis	one item in A + one item in B	Yes	
Definite diagnosis	Suspected diagnosis (one item in A + one item in B) + C		Yes

Tab. 4 - JGLdiagnostic criteria for Acute Cholecystitis (Diagnosis of Case 3&4).

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ORIGINAL ARTICLE

Management of postoperative arterial hemorrhage after pancreato-biliary surgery according to the site of bleeding: re-laparotomy or interventional radiology

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Abstract
Background/Purpose Intra-abdominal arterial hemorrhage is still one of the most serious complications after pancreato-biliary surgery. We retrospectively analyzed our experiences with 15 patients in order to establish a therapeutic strategy for postoperative arterial hemorrhage following pancreato-biliary surgery.
Methods Between August 1981 and November 2007, 15 patients developed massive intra-abdominal arterial bleeding after pancreato-biliary surgery. The initial surgery of these 15 patients were pylorus-preserving pancreatoduodenectomy (PPPD) (7 patients), hemihepatectomy and caudate lobectomy with extrahepatic bile duct resection or PPPD (4 patients), Whipple's pancreatoduodenectomy (PD) (3 patients), and total pancreatectomy (1 patient). Twelve patients were managed by transcatheter arterial embolization and three patients underwent re-laparotomy.
Results Patients were divided into two groups according to the site of bleeding: SMA group, superior mesenteric artery (4 patients); HA group, stump of gastroduodenal artery, right hepatic artery, common hepatic artery, or proper hepatic artery (11 patients). In the SMA group, re-laparotomy and coil embolization for pseudoaneurysm were performed in three and one patients, respectively, but none of the patients survived. In the HA group, all 11 patients were managed by transcatheter arterial embolization. None of four patients who had major hepatectomy with extrahepatic bile duct resection survived. Six of seven patients (85.7%) who had pancreatectomy survived, although hepatic infarction occurred in four.
Conclusions Management of postoperative arterial hemorrhage after pancreato-biliary surgery should be done according to the site of bleeding and the initial operative procedure. Careful consideration is required for indication of interventional radiology for bleeding from SMA after pancreatectomy and hepatic artery after major hepatectomy with bilioenteric anastomosis.

Keywords Complications · Postoperative bleeding · Pancreato-biliary surgery

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Introduction

Despite a declining mortality rate after pancreato-biliary surgery [1–4], intra-abdominal arterial hemorrhage is still one of the most serious complications [5–8]. Arterial bleeding has a disastrous clinical outcome, with a reported mortality rate of 20–50% [6, 9, 10]. With the recent advances in interventional radiology (IVR), transcatheter arterial embolization (TAE) has been widely used, gaining acceptance for the treatment of visceral aneurysm or pseudoaneurysm. Various articles regarding TAE for postoperative ruptured pseudoaneurysm have reported success rates of 50–100% [6, 11–13]. To our knowledge, management of postoperative arterial hemorrhage after pancreato-biliary surgery according to the site of bleeding and clinical condition including the type of operative procedure has not yet been advocated and, therefore, a detailed indication for TAE for this condition has still to be

established. In this article, we retrospectively analyzed our experiences with 15 patients in order to establish a therapeutic strategy for postoperative arterial hemorrhage following hepato-biliary-pancreatic surgery.

Patients and methods

Patients

Between August 1981 and November 2007, 708 patients underwent major pancreato-biliary surgery at the Department of Surgery, Teikyo University Hospital. Fifteen of these 708 patients (2.1%) (11 men and 4 women, 50–82 [mean 65.2] years old) developed massive intra-abdominal arterial bleeding after the surgery. These 15 patients were divided into two groups according to the site of bleeding: SMA group, bleeding from the superior mesenteric artery (SMA) (4 patients); HA group, bleeding from the stump of the gastroduodenal artery (GDA), right hepatic artery (RHA), common hepatic artery (CHA), or proper hepatic artery (PHA) (11 patients).

Patient characteristics are listed in Table 1. Diagnoses at initial surgery were pancreatic head carcinoma (4 patients), lower bile duct carcinoma (4 patients), perihilar cholangiocarcinoma (2 patients), ampullary adenocarcinoma (2 patients), intrahepatic cholangiocarcinoma (1 patient), gallbladder carcinoma (1 patient), and chronic pancreatitis (1 patient). The procedures of the initial surgery were pylorus-preserving pancreatoduodenectomy (PPPD) (7 patients), Whipple's pancreatoduodenectomy (PD) (3 patients), right hemihepatectomy and caudate lobectomy with extrahepatic bile duct resection (2 patients), right hemihepatectomy and caudate lobectomy with PPPD (1 patient), left hemihepatectomy and caudate lobectomy with extrahepatic bile duct resection (1 patient), and total pancreatectomy (1 patient). In the patients with malignant disease, skeletonization of the hepatic artery was routinely performed to remove neural and lymphatic tissue of the hepatoduodenal ligament. In patients with pancreatic head carcinoma, the SMA was right-semicircularly exposed to resect the surrounding right-sided nerve plexus. Sentinel bleeding was defined as minimal bleeding from either abdominal drains or the gastrointestinal tract without evidence of peripheral circulatory impairment or transfusion requirement.

TAE technique

Patients underwent emergency angiography using the standard Seldinger technique and an angiography catheter. At first, superior mesenteric arteriography, arterial portography, and celiac arteriography were performed with a 4 F preshaped catheter. When a pseudoaneurysm or extravasation of contrast media was identified, transcatheter microcoil embolotherapy was performed. A 2.9 F microcatheter (Sniper; Clinical Supply, Gifu, Japan, or Progreat; Terumo, Tokyo, Japan) was then inserted via the angiography catheter. Coil embolization, both proximal and distal of the

Table 1 Profiles of 15 patients encountering intra-abdominal arterial bleeding after pancreato-biliary surgery

Pt. no.	Age (year)/gender	Diagnosis	Initial operation
SMA group			
1	50/M	Ampullary carcinoma	PPPD
2	53/M	Pancreatic head carcinoma	PPPD
3	59/M	Pancreatic head carcinoma	PD
4	64/M	Pancreatic head carcinoma	PPPD
HA group			
5	78/M	Hilar bile duct carcinoma	Right hepatic lobectomy + S1 + BDR
6	73/F	Gall bladder carcinoma	Right hepatic lobectomy + S1 + PPPD + PVR
7	63/F	Hilar bile duct carcinoma	Right hepatic lobectomy + S1 + BDR
8	69/M	Intrahepatic cholangiocarcinoma	Left hepatic lobectomy + S1 + BDR + PVR
9	68/M	Pancreatic head carcinoma	PD
10	66/M	Ampullary carcinoma	PPPD
11	82/M	Lower bile duct carcinoma	PPPD
12	77/F	Lower bile duct carcinoma	PD
13	56/M	Lower bile duct carcinoma	PPPD
14	60/F	Chronic pancreatitis	TP
15	60/M	Lower bile duct carcinoma	PPPD

PPPD Pylorus preserving pancreatoduodenectomy; *PD* pancreatoduodenectomy; *S1* caudate lobectomy; *BDR* extrahepatic bile duct resection; *PVR* portal vein resection; *TP* total pancreatectomy

bleeding site, if possible, or pseudoaneurysm was performed with fibered platinum microcoils of various diameters and shapes (VortX; Boston Scientific, Natick, MA, USA, Trufill; Cordis, Miami Lakes, FL, USA; and Complex Helical Fibered Platinum Coil-18; Target Vascular, Fremont, CA, USA) and interlocking detachable coil (IDC) (Boston-Scientific, Natick, MA, USA) via the microcatheter.

Complication definitions

Leakage of a pancreatojejunostomy and pancreatic fistula was defined as amylase-rich exudate (amylase more than three times normal serum concentration) from the drainage tube placed at the pancreatojejunostomy and near the pancreas, respectively. Leakage of a hepaticojejunostomy was defined as the persistent presence of bile from the drainage tube placed at the hepaticojejunostomy. Diagnosis of hepatic infarction was based on the coexistence of CT findings (geographic, low attenuation perfusion defects without mass effect) seen in association with an acute increase in the serum alanine aminotransaminase level to more than 1,000 IU.

Results

The results are shown in Table 2. The interval between initial surgery and the first intervention in the 15 patients was 5–39 days (mean 22.6 days). All patients had intra-abdominal infections, the cause being leakage of pancreatojejunostomy in six patients, leakage of hepaticojejunostomy in five, and leakage of pancreatojejunostomy and hepaticojejunostomy, leakage of gastrojejunostomy, pancreatic fistula, and perforation of the jejunal limb in one patient each. Sentinel bleeding was noted in seven patients (46.7%) for 1–10 (mean 2.2) days before massive bleeding. Sentinel bleeding was identified as bleeding from drain in all seven patients. When sentinel bleeding occurred, contrast-enhanced CT and angiography were performed in five and four patients, respectively, but neither pseudoaneurysm nor extravasation was demonstrated on either examination. On the occurrence of massive bleeding, contrast-enhanced CT was performed before angiography in eight patients. Intra-abdominal pseudoaneurysms and hematomas, hematomas only, and fluid collection were demonstrated in five, two, and one patient, respectively. Angiography was performed in 14 patients, and pseudoaneurysms or extravasations were demonstrated in all of them.

SMA group

The SMA group consisted of four patients in whom bleeding occurred from SMA. Three patients required re-laparotomy, and coil embolization was performed in one. None of the patients in this group survived.

One patient (patient 2) was transferred to the operating room as soon as bleeding from drain was identified, without first performing angiography. The bleeding site could not be reached at re-laparotomy due to dense adhesion, serious inflammation, and massive bleeding; therefore ligation of the fistula was performed. Complete hemostasis was not obtained and he was died of bleeding shortly after the second surgery. The site of bleeding was detected at autopsy.

Angiography was performed in three patients. Extravasations from the right wall of SMA were demonstrated in two patients (patients 1 and 3) and a pseudoaneurysm of a tributary of SMA was identified in one patient (patient 4). Two patients (patients 1 and 3) were transferred to the operating room because TAE was avoided for fear of ischemia of the small intestine. In patient 1, SMA was cut both proximal and distal to the bleeding point, and re-arterialization was performed with a 6 mm polytetrafluoroethylene (Gore-Tex) graft. Because the jejunal limb lapsed into necrosis during the operation, the jejunal limb was resected with disassembling pancreatojejunostomy and hepaticojejunostomy. Tube hepaticostomy and pancreatostomy were performed. Although hemostasis was obtained, he did not recover from circulatory failure and died one day after the second operation. In patient 3, bleeding was stopped by suturing. The jejunal limb was resected disassembling pancreatojejunostomy and hepaticojejunostomy in order to detect the bleeding point by improving the operative field, although insufficiency of pancreatojejunostomy was not noted. Tube hepaticostomy and pancreatostomy were performed. The upper half of the midline incision was not closed, allowing open drainage. After the second operation, bleeding occurred several times and was stopped by suturing at bedside. The general state deteriorated gradually, and he died of hepatic failure 58 days after the initial surgery.

In one patient (patient 4), bleeding from CHA was also identified prior to bleeding from SMA. For the CHA bleeding, coil embolization was performed for CHA pseudoaneurysm using interlocking detachable coils 34 days after the initial surgery. Re-bleeding from CHA was not noted, but bleeding from a pseudoaneurysm of a tributary of SMA occurred 46 days after the surgery, and coil embolization was performed on the same day (Fig. 1a). He encountered re-bleeding from pseudoaneurysms of SMA at days 52, 63, 103 post surgery (Fig. 1b). For each re-bleeding, coil-embolization was performed for the pseudoaneurysms using interlocking detachable coils. The patency of SMA was preserved in order to prevent ischemia of the small intestine. He died of multiple organ failure 110 days after the initial surgery.

Table 2 Results of 15 patients encountering intra-abdominal arterial bleeding after pancreato-biliary surgery

Pt. no.	Sentinel bleeding	Bleeding site	Postoperative day of bleeding	Cause of intra-abdominal infection	Intervention for bleeding	Complication or result of coil embolization	Outcome
SMA group							
1	Yes	SMA	27	PJ leakage	Replacement of artificial vessel, choledocostomy, and pancreatostomy	Not performed	Died of hemorrhage
2	No	SMA	20	HJ leakage	Surgical hemostasis	Not performed	Died of hemorrhage
3	No	SMA	28	HJ leakage	Surgical hemostasis, choledocostomy, and pancreatostomy	Not performed	Died of hepatic failure
4	Yes	CHA	34	PJ leakage	Coil embolization of pseudoaneurysm	Re-bleeding	Died of hemorrhage
		SMA	46		Coil embolization of pseudoaneurysm 4 times		
HA group							
5	Yes	CHA	9	Pancreatic fistula	TAE of CHA	Unsuccessful	Died of hemorrhage
6	No	GDA	26	PJ leakage	TAE of CHA	Hepatic failure	Died of hepatic failure
7	Yes	PHA	23	HJ leakage	TAE of PHA	Hepatic failure	Died of hepatic failure
8	No	PHA	6	HJ leakage	TAE of PHA	Hepatic failure	Died of hepatic failure
9	Yes	GDA	26	HJ leakage	TAE of CHA	Hepatic infarction	Died of DIC
10	Yes	GDA	31	PJ leakage	TAE of CHA(LHA)	Success	Recovered
11	No	RHA	9	PJ and HJ leakage	TAE of PHA	Hepatic infarction	Recovered
12	Yes	RHA	25	GJ leakage	TAE of RHA	Success	Recovered
13	No	GDA	21	PJ leakage	TAE of CHA	Hepatic infarction	Recovered
14	No	GDA	27	Jejunal limb perforation	TAE of CHA	Hepatic infarction	Recovered
15	No	GDA	27	PJ leakage	TAE of CHA(LHA)	Success	Recovered

SMA Superior mesenteric artery; *PJ* pancreatojejunostomy; *HJ* hepaticojejunostomy; *CHA* common hepatic artery; *TAE* transcatheter arterial embolization; *PHA* proper hepatic artery; *GDA* gastroduodenal artery; *RHA* right hepatic artery

HA group

The HA group consisted of 11 patients in whom bleeding sites were major branches of the celiac artery: stump of GDA in six patients, PHA in two, RHA in two, and CHA in one. TAE was performed in all 11 patients. The sites of embolization were CHA in seven patients, PHA in three, and RHA in one. Portal vein patency was confirmed by superior mesenteric arteriography before embolization in all patients. In 10 of 11 patients, complete hemostasis was obtained and rebleeding was not noted in any of the patients.

All four patients undergoing hepatic lobectomy for biliary hilar malignancy (patients 5–8) died, three of hepatic failure, and one of persistent bleeding within 24 h. On angiography, there were collateral pathways for the arterial supply to hepatic parenchyma in two patients: accessory left hepatic artery arising from left gastric artery (patient 6) and right inferior phrenic artery (patient 7). In two of the four patients, serum total bilirubin concentrations before bleeding were more than 10 mg/dl, and in the other two patients they were less than 3.0 mg/dl.

In seven patients undergoing pancreatectomy (patients 9–15), one patient (patient 9) died of disseminated intravascular coagulation (DIC) 10 days after TAE, and six patients recovered. Hepatic infarction was encountered in four patients. The sites of infarction on CT were lateral segment and S5 in three patients and lateral segment in one. Three of the four patients encountering hepatic infarction improved with lateral segmentectomy performed 3 days after TAE in one patient (patient 11) (Fig. 2), plasma exchange in one (patient 13), and medical treatment in one (patient 14).

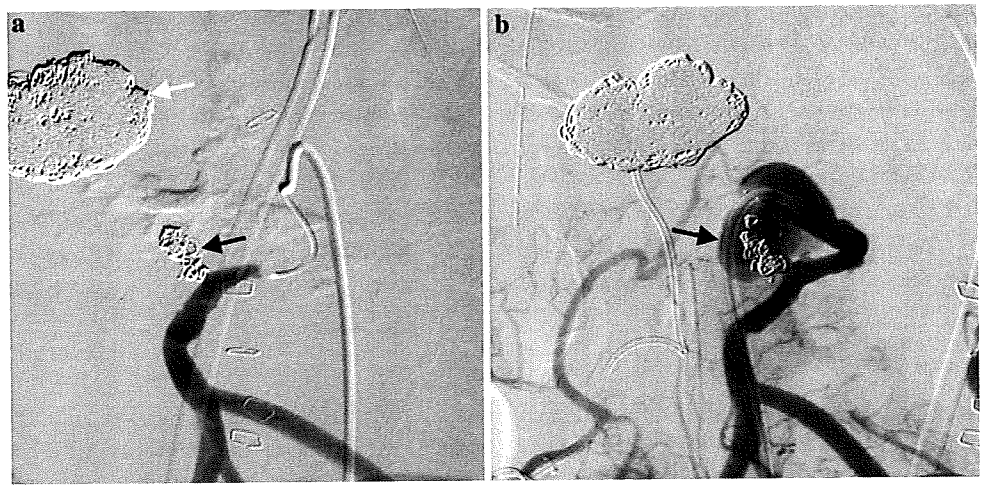

Fig. 1 Superior mesenteric arteriography of patient 4. **a** Coil embolization for pseudoaneurysm of tributary of the superior mesenteric artery was performed. Temporary hemostasis was obtained using interlocking detachable coils. The *white arrow* indicates interlocking detachable coils in the pseudoaneurysm of the stump of the GDA. The *black arrow* indicates interlocking detachable coils in the aneurysm of the tributary of the superior mesenteric artery. **b** Angiography revealed enlargement of pseudoaneurysm (*arrow*) after 6 days of coil embolization for a pseudoaneurysm of a tributary of the superior mesenteric artery. Re-bleeding from pseudoaneurysms of SMA occurred 52, 63, and 103 days after the initial surgery. For each re-bleeding, coil embolizations were performed for the pseudoaneurysms with interlocking detachable coils

In three patients without hepatic infarction, either right (RHA) or left hepatic artery (LHA) was preserved, as the sites of embolization were RHA (patient 8) and CHA (LHA) accompanied by replaced RHA arising from SMA (patients 10 and 15).

Discussion

Sentinel bleeding was first described by Brodsky and Turnbull [6] in 1991 as hemorrhage of drainage fluid or in the gastrointestinal tract. Sato et al. [11] emphasized the significance of early angiography promptly after the recognition of sentinel bleeding, since all 10 patients with massive bleeding after pancreatectomy had sentinel bleeding. However, the efficacy of early angiography for sentinel bleeding was not evaluated. In this study, on the occurrence of sentinel bleeding, CT and angiography failed to demonstrate the site of bleeding in five and four patients, respectively. Sentinel bleeding is less likely to be visualized by angiography, possibly due to the intermittent character of hemorrhage [10, 14]. Okuno et al. [15] reported that 6 patients with episodes of sentinel bleeding after hepato-biliary-pancreatic surgery obtained hemostasis through medical treatment. Although sentinel bleeding does not always accompany subsequent massive bleeding, surgeons should prepare for possible massive bleeding when sentinel bleeding is identified.

There have been a few reports describing postoperative hemorrhage from SMA in detail. Blockage of blood flow from SMA reduces ischemic necrosis of the small intestine. Therefore, we did not perform TAE for SMA in any of the patients. Unfortunately, none of the patients with bleeding from SMA was successfully treated in this study. Recently, a few case reports described the successful treatment of

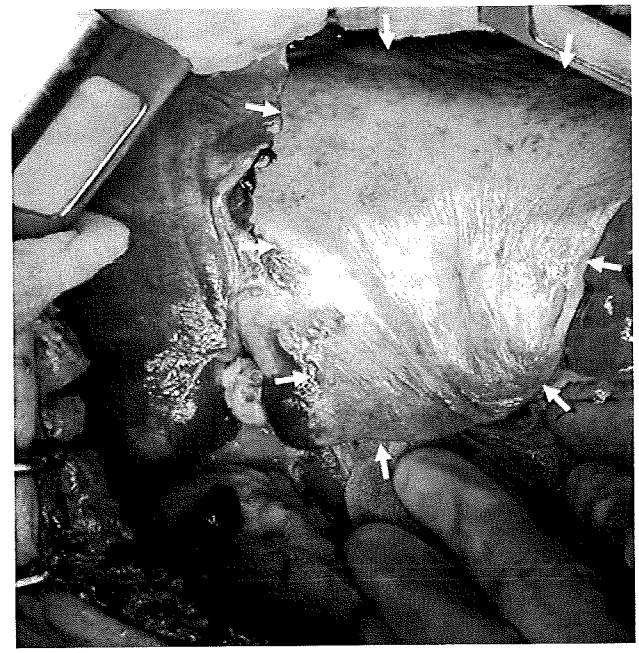

Fig. 2 Lateral segmentectomy was performed for hepatic infarction 3 days after coil embolization for CHA in patient 11. At laparotomy the entire portion of the lateral segment of the liver seemed to be necrotic (*arrows*)

patients with chronic pancreatitis encountering bleeding from SMA by TAE of pseudoaneurysm using interlocking detachable coils [16, 17]. Because the design of the interlocking detachable coil allows controlled release of the device and its accurate placement, it may also be an effective means of treating pseudoaneurysms [17]. In one patient (patient 4), we performed coil embolization for pseudoaneurysm of SMA by using interlocking detachable coils, but repeated bleeding occurred due to sepsis from pancreatojejunal anastomotic insufficiency. The long-term success of embolization of the pseudoaneurysm with interlocking detachable coils is unknown. One case report has described coil compaction after embolization of SMA pseudoaneurysm [18]. In cerebral aneurysm, coil compaction following successful embolization often occurs [16]. Even if an aneurysm is completely embolized, the "water-hammer effect" of blood pulsation transmitted into the coil-thrombus complex may induce eventful coil compaction or aneurysm regrowth with potential for late bleeding [19]. In aneurysms of SMA, abdominal movements and pancreatic enzymes may also increase the risk of coil compaction [18]. There is a report that continuous intra-arterial infusion of vasopressin for bleeding from a branch of SMA after PD obtained complete hemostasis [15]. However, whether this procedure using vasopressin would be effective for massive bleeding from SMA remains unclear.

The first line of treatment for massive bleeding from hepatic artery after pancreatectomy is TAE. However, the long-term result of this treatment is unknown, and liver infarction or ischemia has been reported as a mortal complication of TAE for pseudoaneurysms arising from hepatic arteries [11, 13, 20]. It is commonly believed that the dual blood supply and extensive collateral pathways of the liver serve to protect this organ from ischemic insult. Occlusion of the hepatic artery is usually well-tolerated and has been performed safely in some cases following PD. However, recently Yoon et al. [21] revealed a 30% incidence of liver abscess after hepatic artery occlusion for bleeding pseudoaneurysms after PD. Kim et al. [22] found that six out of seven patients had hepatic abscesses following hepatic artery embolization after having undergone a prior Whipple procedure. In our series, liver infarction occurred in five patients. Ligation of the PHA results in the development of small collateral vessels in the hepatic ligaments and around the common bile duct [23]. These collateral vessels may be noted immediately after temporary occlusion of hepatic arterial flow [23]. However, collateral arteries around the common bile duct were disrupted after bile duct resection. The fact that infarction of the entire liver did not occur in any patients must be attributable to recruitment of other collateral pathways including inferior phrenic arteries, intercostal arteries, and gastric arteries that were presumably not ligated at the initial surgery.

There have been a few detailed reports in the literature concerning postoperative hemorrhage after major hepatectomy for biliary hilar malignancy [15, 24]. Nagino et al. [24] treated nine patients who encountered postoperative hemorrhage after hepatectomy for hilar cholangiocarcinoma. Re-laparotomy was performed in eight patients, and seven survived. In only one patient was TAE used for rupture of the RHA after extended left hepatic lobectomy. The patient suffered from liver failure following embolization, but he recovered. In our series, all four patients after major hepatectomy with extrahepatic bile duct resection died of hepatic failure or persistent bleeding within 24 h after TAE of hepatic artery. These results imply that TAE for postoperative hemorrhage after major hepatectomy with extrahepatic bile duct resection is nearly contraindicative. Experimental and clinical studies elucidated that partial arterialization of the portal vein for hepatic artery occlusion after hepatic resection could preclude hepatic necrosis or failure [25, 26]. Iseki et al. [27] reported three successful cases of concomitant hepatic artery resection with arterioportal shunt for pancreato-biliary malignancy, advocating that mesenteric arterioportal shunt has the advantages of choice of arterial size and a site removed from the primary operative field.

The recent refinement of IVR has been solving the most serious complications of TAE, namely, mesenteric ischemia and hepatic infarction or failure after TAE for SMA and hepatic artery, respectively. In recent years, covered stent-grafts have been used in the endovascular repair of abdominal aneurysm. Obliteration of the aneurismal neck results in effective obliteration of the aneurysm without block of arterial flow [28]. In 1998, McGraw et al. [29] first reported the successful use of covered stent for management of a SMA pseudoaneurysm occurring after PD. In 2000, Burger et al. [30] first described the successful stent-graft repair of a ruptured hepatic artery aneurysm after PD. Some authors actually consider this therapeutic option as the first-line treatment for a bleeding pseudoaneurysm in critical situations where the consequences of superior mesenteric or hepatic artery occlusion may be disastrous [20, 30–32]. The procedure remains risky due to the possibility of rupturing the artery because of its fragile vascular wall and infection involving the covered stent itself, since the vessel is affected by septic condition [33]. It has to be mentioned, however, that this angiographic intervention in branches of the celiac trunk is technically demanding, even in experienced centers, and is not always possible for anatomical reasons [20]. In addition, long-term results of endovascular covered stent placement have yet to be evaluated; therefore further investigation will be necessary to determine the feasibility of this treatment. In

Japan, no covered stent for visceral arteries is commercially available; thus we do not perform this procedure.

Re-laparotomy is sometimes required for control of bleeding that was technically impossible to stop by angiography, or for recurrent bleeding after coil embolization [20]. Some authors claimed that surgical intervention was the first-line treatment for postoperative massive hemorrhage following pancreato-biliary surgery [34–36]. There are two major advantages of surgical intervention over TAE. Hemostasis by suturing could stop arterial bleeding without blocking blood flow. Control of infection could be achieved by completion pancreatectomy, tube pancreatostomy, or appropriate drainage tube placement [34, 35]. However, a surgical approach to bleeding pseudoaneurysm is sometimes unsuccessful because of dense adhesion and a poor operative view filled with blood. In such situations the aortic occlusion balloon catheter method is helpful for controlling oligemic shock and obtaining a good view of the operative field [34].

Randomized controlled trials (RCTs) are needed to elucidate the appropriate management for massive bleeding after pancreato-biliary surgery. However, it is difficult to plan RCTs in an ethically acceptable manner, and besides that, the number of patients encountering postoperative hemorrhage is fortunately quite limited. A drawback of the present study, of course, is this very fact, the limited number of patients.

In any event, we think that postoperative arterial hemorrhage after pancreato-biliary surgery should be managed according to the site of bleeding and the initial operative procedure. The first-line treatment for bleeding from major branches of the celiac artery after pancreatectomy is TAE, in spite of the fact that the possibility of hepatic infarction does exist. Careful consideration is required for the indication of IVR for bleeding from SMA after pancreatectomy and hepatic artery after major hepatectomy with bilioenteric anastomosis. Treatment options including partial arterialization of the portal vein and re-laparotomy should be considered for the prevention of hepatic failure and re-bleeding. Angiographic stenting with preservation of superior mesenteric and hepatic arterial flow, if technically possible, might represent the best treatment option.

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CASE REPORT

Resected case of eosinophilic cholangiopathy presenting with secondary sclerosing cholangitis

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Abstract

Eosinophilic cholangiopathy is a rare condition characterized by eosinophilic infiltration of the biliary tract and causes sclerosing cholangitis. We report a patient with secondary sclerosing cholangitis with eosinophilic cholecystitis. A 46-year-old Japanese man was admitted to our hospital with jaundice. Computed tomography revealed dilatation of both the intrahepatic and extrahepatic bile ducts, diffuse thickening of the wall of the extrahepatic bile duct, and thickening of the gallbladder wall. Under the diagnosis of lower bile duct carcinoma, he underwent pylorus-preserving pancreatoduodenectomy and liver biopsy. On histopathological examination, conspicuous fibrosis was seen in the lower bile duct wall. In the gallbladder wall, marked eosinophilic infiltration was seen. Liver biopsy revealed mild portal fibrosis. He was diagnosed as definite eosinophilic cholecystitis with sclerosing cholangitis with unknown etiology. The possible etiology of sclerosing cholangitis was consequent fibrosis from previous eosinophilic infiltration in the bile duct. The clinicopathological findings of our case and a literature review indicated that eosinophilic cholangiopathy could cause a condition mimicking primary sclerosing cholangitis (PSC). Bile duct wall thickening in patients with eosinophilic cholangitis might be due to fibrosis of the bile duct wall. Eosinophilic cholangiopathy might be confused as PSC with eosinophilia.

Key words: Eosinophilc cholangiopathy; Primary sclerosing cholangitis; Secondary sclerosing cholangitis; Obstructive jaundice

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INTRODUCTION

Benign disorders such as primary sclerosing cholangitis (PSC), IgG4-related autoimmune pancreatitis, eosinophilic cholangitis, intraductal stone disease, surgical or blunt abdominal trauma, intra-arterial chemotherapy, and portal biliopathy can present with obstructive jaundice. Eosinophilic cholangiopathy is also one of the causes of sclerosing cholangitis[1-5]. The literature contains only about 40 case reports on eosinophilic cholangiopathy[4-6], and therefore, little attention has been paid to this condition. The disease is characterized by a dense transmural eosinophilic infiltration of the biliary tract. It is highly responsive to oral steroid therapy[4-6]. Surgery, including bile duct resection, is unnecessary if a diagnosis of eosinophilic cholangiopathy is made; therefore, a detailed histopathological examination of this disease has not yet been performed.

Sclerosing cholangitis from the above-mentioned etiology is sometimes difficult to differentiate from PSC. PSC patients in Japan have a higher incidence of eosinophilia, a less frequent association with