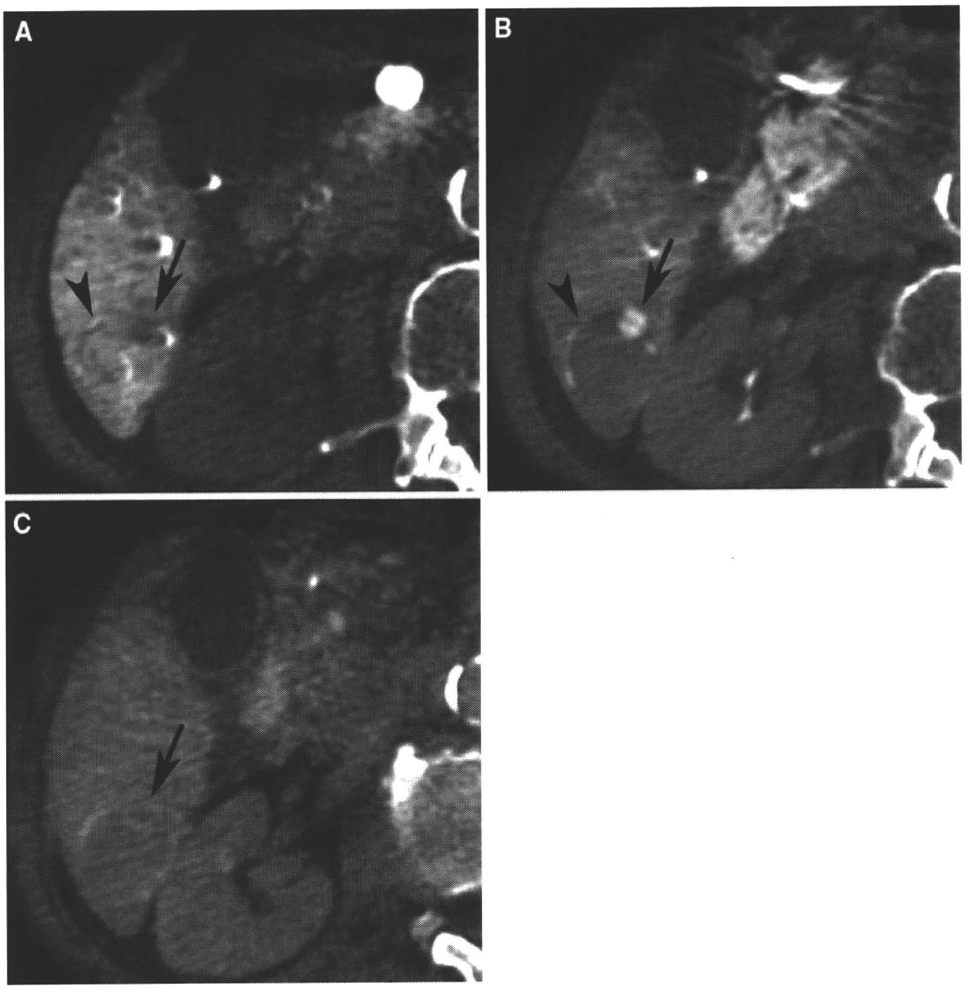

Fig. 3 Early-stage HCC, including a hypervascular focus (8-mm diameter), in the right lobe of the liver. **A** CBCTAP showed a small hypoattenuating lesion (*arrow*) in the isoattenuating nodule (*arrowhead*). **B** First-phase CBCTHA showed 3.3 cm-diameter hypoattenuating nodule (*arrowhead*), including the hypervascular focus (*arrow*). The nodule was diagnosed as early-stage HCC (nodule-in-nodule appearance). **C** Second-phase CBCTHA showed thin corona enhancement around the hypervascular focus within the hypovascular mass (*arrow*)

Inoue et al. [3] reported that corona enhancement was seen in 84% of HCC lesions and 73% of metastatic liver tumors and cholangiocarcinomas on late-phase conventional CTHA images. Our findings suggest that dual-phase CBCTHA has an ability to detect corona enhancement of HCC lesions that is almost equal to conventional CTHA; however, the spatial and contrast resolution of CBCT is inferior to that of conventional multidetector CT. We injected a larger amount of contrast material for dual-phase CBCTHA than that for single-phase CBCTHA [9] to facilitate the depiction of corona enhancement. This additional contrast material might have improved the detectability of corona enhancement on CBCTHA.

There are some limitations to the present study. First, corona enhancement was only evaluated on CBCTHA images, and conventional CTHA images were not obtained in any tumor. Therefore, absence of corona enhancement may not directly indicate that the tumor does not have obvious tumor drainage. Because all hypervascular HCC lesions may have tumor drainage around the surrounding liver parenchyma, we speculate that the absence of corona enhancement on second-phase CBCTHA images, especially in small tumors, may be influenced mainly by the degree of tumor vascularity and image quality of CBCT. Second, histological confirmation was not obtained in any of the tumors. However, we consider that advances in imaging modalities have facilitated the establishment of HCC diagnosis without biopsy. Third, we could not confirm whether tumors showing thick corona enhancement had a tumor capsule because all tumors were treated by TACE alone. Fourth, all arterioportal shunts in the present study were large and presented typical imaging findings; therefore, we could not conclude that small arterioportal shunts could be adequately differentiated from small HCC lesions by dual-phase CBCTHA technique. However, we consider dual-phase CBCTHA technique useful to distinguish small HCCs from hypervascular pseudolesions.

In conclusion, dual-phase CBCTHA can depict corona enhancement around almost all hypervascular HCC lesions. This technique may improve the diagnostic accuracy of HCC by CBCTHA.

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CLINICAL INVESTIGATION

Main Bile Duct Stricture Occurring After Transcatheter Arterial Chemoembolization for Hepatocellular Carcinoma

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Abstract The purpose of this study was to evaluate the clinical course of main bile duct stricture at the hepatic hilum after transcatheter arterial chemoembolization (TACE) for hepatocellular carcinoma (HCC). Among 446 consecutive patients with HCC treated by TACE, main bile duct stricture developed in 18 (4.0%). All imaging and laboratory data, treatment course, and outcomes were retrospectively analyzed. All patients had 1 to 2 tumors measuring 10 to 100 mm in diameter (mean ± SD 24.5 ± 5.4 mm) near the hepatic hilum fed by the caudate arterial branch (A1) and/or medial segmental artery (A4) of the liver. During the TACE procedure that caused bile duct injury, A1 was embolized in 8, A4 was embolized in 5, and both were embolized in 5 patients. Nine patients (50.0%) had a history of TACE in either A1 or A4. Iodized oil accumulation in the bile duct wall was seen in all patients on computed tomography obtained 1 week later. Bile duct dilatation caused by main bile duct stricture developed in both lobes ($n = 9$), in the right lobe ($n = 3$), in the left lobe ($n = 4$), in segment (S) 2 ($n = 1$), and in S3 ($n = 1$). Serum levels of alkaline phosphatase and γ-glutamyltranspeptidase increased in 13 patients. Biloma requiring drainage developed in 2 patients; jaundice developed in 4 patients; and metallic stents were placed in 3 patients. Complications after additional TACE sessions, including biloma ($n = 3$) and/or jaundice ($n = 5$), occurred in 7 patients and were treated by additional intervention, including metallic stent placement in 2 patients. After initial TACE of A1 and/or A4, 8 patients (44.4%), including 5 with uncontrollable jaundice or cholangitis, died at 37.9 ± 34.9 months after TACE, and 10 (55.6%) have survived for 38.4 ± 37.9 months. Selective TACE of A1 and/or A4 carries a risk of main bile duct stricture at the hepatic hilum. Biloma and jaundice are serious complications associated with bile duct strictures.

Keywords Hepatocellular carcinoma · Transcatheter arterial chemoembolization · Bile duct stricture · Jaundice · Biloma · Metallic stent placement

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Introduction

Transcatheter arterial chemoembolization (TACE) is a widely accepted treatment option for inoperable hepatocellular carcinoma (HCC) [1, 2]. A recent randomized trial showed a distinct survival advantage for TACE compared with the best supportive care [3, 4].

With the advancement of TACE technologies, therapeutic effects have improved, whereas adverse effects have decreased [5–8]. However, TACE may still cause several complications, such as pulmonary oil embolism, abscess formation, tumor lysis syndrome, and intrahepatic bile duct necrosis (biloma), in addition to postembolization syndrome [9–15].

Since the first report by Maciuchi et al. [16], several investigators have reported main bile duct stricture at the hepatic hilum after TACE [17, 18]. However, the causes and clinical futures of bile duct strictures are still not well known. In this report, we describe the clinical course and risk factors for main bile duct stricture at the hepatic hilum after TACE.

Materials and Methods

Patients

Between January 2004 and June 2009, we encountered 18 patients with main bile duct stricture at the hepatic hilum with intrahepatic bile duct dilatation developing after TACE for HCC among 446 consecutive patients treated by TACE. We excluded bile duct invasion of HCC by imaging findings. There were 8 men and 10 women, and the mean patient age was 71.9 ± 6.6 years (range 58 to 83). Patient profiles are listed in Table 1. All patients had liver cirrhosis. This was related to hepatitis C in 13 patients and to hepatitis B in 1 patient. The etiology was unknown in 4 patients. The diagnosis of HCC was established (1) by imaging findings of computed tomography (CT) and/or magnetic resonance imaging (MRI) (i.e., characteristic nodular enhancement on the arterial-phase images and washout on the delayed-phase images) in addition to (2) nodular stain on angiography and/or CT during hepatic arteriography (CTHA) and (3) nodular perfusion defect on CT during arterial portography (CTAP). Since May 2006, CTHA and CTAP images were obtained using a cone-beam CT (CBCT) technique (XperCT; Philips Medical Systems, Best, The Netherlands). The treatment records up to the initial treatment for HCC were retrospectively analyzed. Written informed consent was obtained from each patient before TACE. Institutional Review Board approval for this type of retrospective study is not required at our institution.

TACE Procedure

A 1.8F tip (Carnelian Pixie; Tokai Medical Products, Kasugai, Japan), 2F tip (Progreat α; Terumo, Tokyo, Japan) or 2.4F tip (Microferret; Cook, Bloomington, IN) microcatheter, passed through a 4F catheter, was used for all TACE procedures. To navigate the microcatheter, a 0.016-inch guidewire (GT-Wire; Terumo) was used. The microcatheter was advanced into the tumor-feeding branch as selectively as possible to minimize the embolized area in each patient.

After the microcatheter was inserted into the target branch, 0.5 mL 2% lidocaine (Xylocaine; Fujisawa, Osaka, Japan) was injected intra-arterially to prevent pain and vasospasm. First, the following was injected a mixture of (1) 2 to 10 mL iodized oil (Lipiodol; Andre Guerbet, Aulnaysous-Bois, France), (2) contrast material, i.e., 370 mg I/mL iopamidol (Iopamiron 370; Bayer, Osaka, Japan) or 350 mg I/mL iomeprol (Iomeron 350; Ezai, Tokyo, Japan) equal to one third the quantity of iodized oil, (3) anticancer drugs, i.e., 10 to 30 mg epirubicin (Farmorbicin; Kyowa Hakko, Tokyo, Japan), and (4) 2 to 6 mg mitomycin C (Mitomycin; Kyowa Hakko)—followed by injection of gelatin sponge particles. The total amount of iodized oil in a single procedure was determined based on tumor size (almost equal to the diameter of the tumor, e.g., a 3-cm tumor received 3 mL iodized oil) but did not exceed 10 mL in a single TACE session. Up until December 2006, we had used gelatin sponge (Gelfoam; Upjohn, Kalamazoo, MI) particles cut into approximately 1-mm cubes. Since January 2007, we have used commercially available 1 mm–diameter gelatin sponge particles (Gelpart; Nippon Kayaku, Tokyo, Japan). For all patients but 2, the particles were crushed into approximately 0.5-mm particles by pumping 20 times using a 3-way stopcock and 2 2.5-mL syringes, and then the gelatin sponge slurry was injected to obstruct the tumor-feeding branch. In the remaining 2 patients, who had tumors measuring 9.3 and 10 cm in diameter, respectively, 1-mm diameter gelatin sponge particles were used. Gelatin sponge particles were injected until the tumor-feeding branch was blocked and the targeted tumor stain disappeared on angiography. In addition, stepwise TACE sessions were performed at 3-to 10-week intervals to avoid severe complications, such as abscess formation or tumor lysis syndrome.

CBCT was performed in 7 patients during the TACE procedure. In 3 patients, CBCT images were obtained by injection of contrast material through A1 ($n = 2$) or immediately after TACE of both A1 and the medial segmental artery (A4) ($n = 1$) to confirm the embolized area.

Table 1 Patient profiles

Patient characteristics	
Male	8
Female	10
Mean age (years)	71.9 ± 6.6
Liver cirrhosis (%)	18 (100)
HCV related (%)	13 (72.2)
HB related (%)	1 (5.6)
Etiology unknown (%)	4 (22.2)
Tumor size near the hepatic hilum (mm)	24.5 ± 25.4
Intrahepatic multiplicity (%)	
Single	11 (61.1)
Two	4 (22.2)
Three	1 (5.6)
Four	2 (11.1)

Follow-Up

Unenhanced CT was obtained at 1 week after TACE in all patients to check for iodized oil distribution. All patients were followed-up, and dynamic CT was performed every 2 to 3 months after TACE to investigate any tumor recurrence. If possible, an additional TACE session was performed when local recurrence or newly developed lesions were demonstrated at other sites. MRI ($n = 11$), endoscopic retrograde cholangiopancreatography (ERCP) ($n = 8$), or percutaneous transhepatic biliary drainage (PTBD) ($n = 6$) was performed when main bile duct stricture with bile duct dilatation was demonstrated on follow-up CT. Laboratory data—including serum bilirubin (normal range 0.2 to 1.0 mg/dL), alkaline phoshatase (ALP; normal range 104 to 338 U/L), and γ-glutamyl-transpeptidase (γ-GTP; normal range 16 to 73 U/L)—were examined in all patients 1 day before TACE, 1 week after TACE, and every 1 to 3 months after TACE. Degrees of increased ALP level were divided into 3 grades: slight (<150 U/L), moderate (151 to 300 U/L), and marked (>301 U/L). Degrees of increased γ-GTP level were also divided into 3 grades: slight (<100 U/L), moderate (101 to 200 U/L), and marked (>201 U/L).

Data Analysis

All imaging results (arteriograms, CBCT, CT, MRI, cholangiograms), laboratory data, treatment courses, and outcomes were retrospectively evaluated in each patient.

Results

The results are summarized in Table 2. All patients were followed-up until death or to date.

Tumors

Eleven patients had a single tumor, and 7 patients had 1 to 3 tumors. All patients but 1 had a tumors in S1 and/or S4. The remaining patient had a tumor protruding from S2, which was partially supplied by A1. Five patients had a tumor located in S1, including 1 patient with 2 tumors in S1. Five patients had a tumor located in S4. Four patients each had a tumor located in the boundary between S4 and S5, between S1 and S4, between S1 and S8, and between S1 and S5. Two patients had a large tumor located in the right lobe extending to S1 and S4. One patient had 2 tumors located in both S1 and S4. In total, 20 tumors were located near the hepatic hilum. All were initially detected HCCs without any previous treatment ($n = 9$) or new HCCs in patients who had a history of previous treatment for HCC ($n = 11$). Mean tumor diameter was 24.5 ± 25.4 mm (range 10 to 100). Six patients had 1 to 3 other small viable tumors with a mean diameter of 1.7 ± 1.0 mm at the periphery of the liver.

Embolized Branches

All patients underwent TACE of A1 and/or A4 during the TACE procedure just before development of bile duct stricture. Three patients underwent TACE of A1 alone, and 5 patients underwent TACE of A1 and other hepatic branches except A4 (Figs. 1, 2), including 2 patients who underwent embolization of 2 A1 branches. Five patients underwent TACE of A4 and other hepatic branches except A1, including 1 patient who underwent embolization of 2 A4 branches (Fig. 3). Three patients underwent TACE of both A1 and A4. The remaining 2 patients underwent TACE of both A1 and A4, in addition to another hepatic arterial branch or extrahepatic collaterals. In total, A1 was embolized in 8 patients; A4 was embolized in 5 patients; and both A1 and A4 were embolized in 5 patients. During the TACE procedure, communications between the following were demonstrated: the right A1 and the left A1 ($n = 3$), the A1 and the A4 ($n = 2$), 2 A4 branches ($n = 2$), and the A1 and the 3 o'clock and 9 o'clock arteries ($n = 2$) (Figs. 1, 4). In 9 patients (50.0%), TACE of A1 and/or A4 had previously been performed. TACE of A1 had been performed once in 2 patients. TACE of A4 had been performed once in 2 patients and twice in 1 patient. TACE of both A1 and A4 had been performed in 4 patients, including 3 patients who had undergone TACE of 2 A1 branches and 2 A4 branches once to 3 times.

Among 446 consecutive patients treated by TACE during the same period, A1 was embolized in 54 patients; A4 was embolized in 152 patients; and both A1 and A4 were embolized in 86 patients. Thus, the incidence of bile duct stricture after TACE was 4.0% and that after TACE of A1 and/or A4 was 6.2%.

CBCT Findings Obtained During the Procedure

CBCT during arteriography of A1 showed wall enhancement of the common hepatic duct (CHD) in 1 patient (Fig. 1) and wall enhancement of the CHD and right hepatic duct (RHD) in another patient (Fig. 2). CBCT obtained immediately after TACE of both A1 and A4 showed iodized oil accumulation in the wall of the left hepatic duct (LHD).

CT Findings Obtained 1 Week After TACE

Iodized oil accumulation in the CHD wall was seen in 9 patients (50%), 4 with TACE of both A1 and A4, 4 with TACE of A1, and 1 with TACE of A4 (Figs. 1, 2). It

Table 2 Summary of 18 patients with main bile duct stricture occurring after TACE

Case no./age (years)/sex	Tumor diameter (mm)	Segment	Embolized branches	Anastomosed branches	Previous embolized branches (no. of times)	Iodized oil–accumulated portion	Site of bile duct dilatation	Shrinkage of gallbladder	Changes in bile duct dilatation	Complications	No. of additional TACE	Complications after additional TACE
1/80/F	19	S4	A4, etc.		A4 (1)	LHD	Both	+	Progressed	Biloma	1	Jaundice
2/76/F	16	S1	A1			CHD	Both	+	Progressed		1	
3/72/M	20	S2, etc.	A1 × 2, etc.	A1–A1		LHD	Left	+	Progressed		4[c]	
4/81/F	10	S1	A1			CHD	Right		Improved	Cholangitis	1	Biloma
5/61/F	10	S1	A1	A1–A4	A1 (1)	RHD	Right		Progressed			
6/74/M	20	S1/4, etc.	A1, A4	A4–A4		CHD	Both		Improved	Cholangitis, biloma,		
7/64/M	17	S1	A1, etc.		A1 (3), A4 (2)	CHD	Both		Progressed	Jaundice[a]	4	Biloma, jaundice[d]
8/58/M	100	S8-1-4	A4, etc.	A1–A1	A1 (2), A4 (1)	CHD	Both		Progressed	Biloma[b], jaundice[a],	2	Biloma[b]
9/83/F	93	S8-1-4	A1, A4, etc.	A1–3 and 9 o'clock As	A1 (3), A4 (1)	CHD	Both		Improved	Biloma	4	
10/73/F	16	S4	A1, A4, etc.	A1–A1	A4 (1)	CHD	Left		Progressed		3	Jaundice[a]
11/70/M	24, 12	S1, etc.	A1 × 2, etc.			CHD	Both			Jaundice		
12/71/F	12	S1/8, etc.	A1, etc.			LHD	S2		Progressed		1	
13/68/F	17	S4, etc.	A4 × 2, etc.			LHD	S3		Progressed		5	
14/70/M	36	S4/5	A4, etc.		A1 (1), A4 (2)	CHD, RHD, LHD	Both		Progressed	Biloma[b], jaundice[a]	1	Jaundice[d]
15/76/M	11	S4 × 2, etc.	A4, etc.	A4–A4		CHD, LHD	Left		Progressed		3	
16/77/F	17	S5/1	A1, etc.	A1–3 and 9 o'clock As	A1 (1)	RHD, CHD, CBD	Right	+	Progressed			
17/68/M	15	S4	A1, A4	A1–A4	A4 (2)	CHD	Both		Progressed		4	Jaundice[a]
18/72/F	10, 15	S1, S4	A1, A4			LHD	Left					

[a] Percutaneous transhepatic biliary drainage and stent placement was performed
[b] Percutaneous drainage was performed
[c] Metallic stent was placed to relief bile duct stricture before additional TACE
[d] Percutaneous transhepatic biliary drainage was continued until patients' death

Fig. 1 A 74-year-old man with HCC in S1/4 (patient no. 6). **A** Right hepatic arteriogram shows A1 arising from the proximate portion of the anterior branch of the right hepatic artery (*arrow*). **B** CBCT during arteriography of A1 shows wall enhancement of the CHD (*arrow*). This vessel was embolized. **C** Spot radiograph obtained during TACE of A1. **D** Left hepatic arteriogram shows 2 A4 branches (*arrows*). **E** One A4 branch was selected and embolized. During the TACE procedure, another A4 branch is demonstrated through anastomosis (*arrow*). The *arrowhead* indicates a tumor. **F** CT obtained at 1 week after TACE shows iodized oil accumulation and wall thickening of the CHD (*arrow*). **G** CT obtained 1 month after TACE shows wall thickening of the CHD and intrahepatic bile duct dilatation. **H** ERCP shows the stricture at the CHD (*arrow*) and occlusion of the LHD. Intraluminal defect suggesting coagula in the CHD is also seen (*arrowhead*). **I** CT obtained at 3 months after TACE shows improvement of bile duct wall thickening and intrahepatic bile duct dilatation

was seen in the LHD wall in 5 patients (27.8%), in 1 patient with TACE of both A1 and A4, in 2 patients with TACE of A1, and in 2 patients with TACE of A4 (Fig. 3). It was seen in the RHD wall in 1 patient (5.6%) with TACE of A1. It was seen in the LHD wall and CHD wall in 1 patient (5.6%) with TACE of A4 and in the bilateral hepatic ducts wall and CHD wall in 1 patient (5.6%) with TACE of A4. In 1 patient (5.6%) with demonstration of the 3 o'clock and 9 o'clock arteries during TACE of A1, iodized oil was accumulated in the RHD, the CHD, and the CBD (Fig. 4). Bile duct wall thickening at the site of iodized oil accumulation was also demonstrated in all patients (Figs. 2, 3, 4, 5). In all 3 patients who underwent selective CBCT, CT obtained 1 week after TACE showed iodized oil accumulation in the same portion of the bile duct where wall enhancement or iodized oil accumulation was observed on CBCT (Fig. 1).

Fig. 2 An 81-year-old woman with HCC in S1 (patient no. 4). **A** Arteriogram of A1 shows a faint tumor stain (*arrow*). **B** CBCT during arteriography of A1 shows a tumor stain (*arrow*). Wall enhancement of the CHD and RHD is also seen (*arrowheads*). **C** CT obtained at 2 months after TACE of A1 shows wall thickening of the CHD (*arrow*) and intrahepatic bile duct dilatation (not shown). **D** MR cholangiopancreatography shows short and smooth strictures at the CHD and RHD (*arrows*). Intraluminal defect suggesting coagula is also seen (*arrowhead*)

In patients who underwent repeated TACE sessions of A1 and/or A4, iodized oil accumulation was observed at the same portion of the bile duct on each CT obtained 1 week after the respective TACE procedures.

Serial CT Findings

Main bile duct stricture at the hepatic hilum and intrahepatic bile duct dilatation developed in all patients. Except for 1 patient, the site of bile duct stricture corresponded with the portion showing iodized oil accumulation on CT obtained at 1 week after TACE. In this 1 patient, CT obtained at 1 week after TACE showed iodized oil accumulation in the RHD, the CHD, and the CBD wall, but the stricture developed only at the RHD. Bile duct dilatation was seen in both lobes ($n = 9$ [50.0%]) (Fig. 1), in the right lobe ($n = 3$ [16.7%]) (Fig. 4), in the left lobe ($n = 4$ [22.2%]), in S2 ($n = 1$ [5.6%]), and in S3 ($n = 1$ [5.6%]) (Fig. 3). These findings were demonstrated after initial TACE of A1 and/or A4 in 9 patients (50.0%) and after 2 to 4 TACE sessions of A1 and/or A4 in 9 patients (50.0%). The findings were demonstrated 4 to 92 months (mean 32.6 ± 29.7) after the initial TACE of A1 and/or A4 and 2 to 4 months (mean 2.8 ± 0.8) after the last TACE of A1 and/or A4 before bile duct injury. In 5 patients (27.8%), hyperattenuating material suggesting coagula was seen in the CBD. Intrahepatic biloma developed simultaneously in 5 patients (27.8%).

During follow-up, bile duct dilatation improved in 3 patients (16.7%) (Fig. 1), whereas it gradually progressed in 13 patients (72.2%) (Fig. 4). In the remaining 2 patients (11.1%), follow-up CT was not obtained except for the CT that initially demonstrated the bile duct dilatation. In addition, shrinkage of the gallbladder and retention of concentrated bile developed in 4 patients (22.2%) (Fig. 4).

MRI Findings

MRI was obtained in 11 patients. Strictures of the CHD and bilateral hepatic ducts were seen in 7 patients (63.6%); strictures of the CHD and RHD were seen in 2 patients (18.2%) (Fig. 2); and stricture of the LHD was seen in 2 patients (18.2%). Intraluminal coagula was seen in 8 patients (72.7%) (Fig. 2).

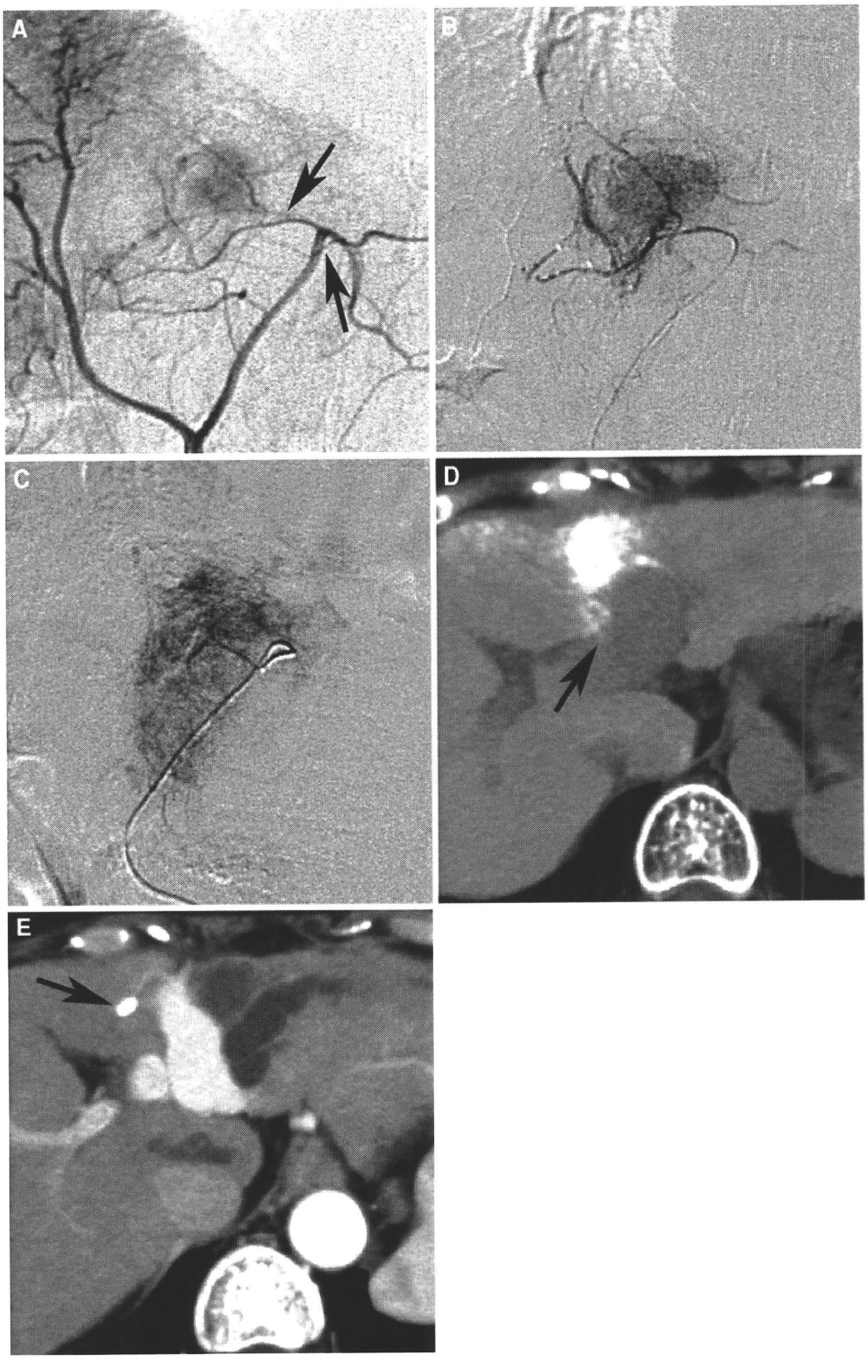

Fig. 3 A 68-year-old woman with HCC in S4 (patient no. 13). **A** Proper hepatic arteriogram shows a tumor supplied by 2 A4 branches (*arrows*). **B** One A4 branch was selected and embolized. **C** Another A4 branch was subsequently selected and embolized. **D** CT obtained at 1 week after TACE shows iodized oil accumulation in the tumor and LHD (*arrow*). **E** CT obtained at 72 months after TACE shows marked bile duct dilatation in S3 without any clinical symptoms. Iodized oil is also densely retained in the tumor (*arrow*)

ERCP and PTBD Findings

ERCP was performed in 8 patients, and PTBD was performed in 6 patients. Two patients underwent both examinations. ERCP showed occlusion of the CHD in 2 patients (25.0%): occlusion of the RHD in 1 patient (12.5%) and occlusion of the LHD in 1 patient (12.5%). In 3 patients (37.5%), strictures of the CHD and bilateral hepatic ducts were seen. In the remaining patient (12.5%), strictures of the CHD and RHD and occlusion of the LHD were seen (Fig 2). Intraluminal defects suggesting coagula were seen in 6 patients (75.0%) (Fig. 1).

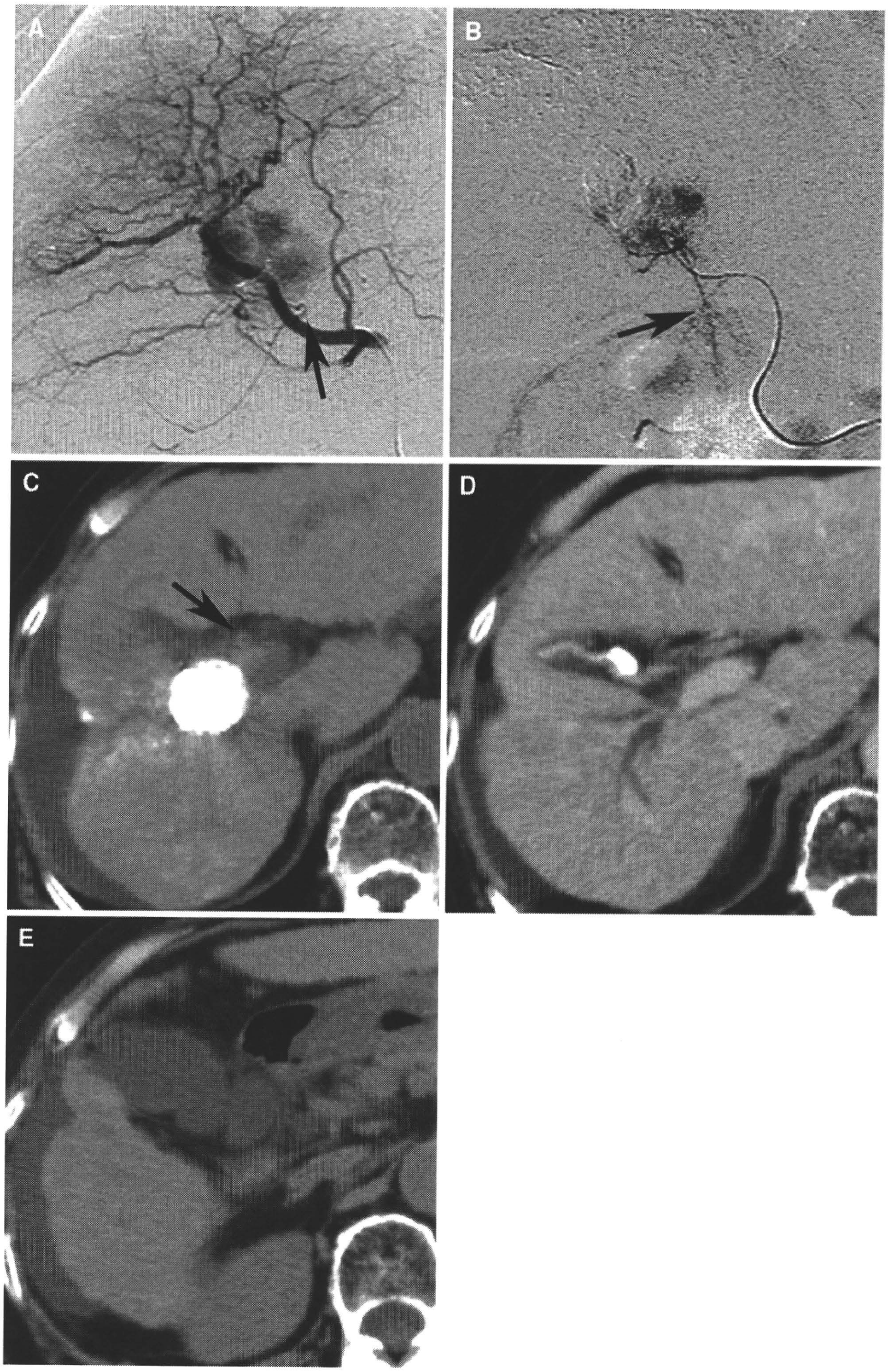

Fig. 4 A 77-year-old woman with HCC in S1/S5 (patient no. 16). **A** Right hepatic arteriogram shows a tumor stain supplied by the anterior branch of the right hepatic artery and A1 (*arrow*). Another A1 arising from the right hepatic artery was previously embolized (not shown). **B** Arteriogram of A1 shows a tumor stain. The 3 o'clock and 9 o'clock arteries that are not seen on (**A**) are also demonstrated (*arrow*). **C** CT obtained at 1 week after TACE shows iodized oil accumulation in the tumor and RHD (*arrow*). Iodized oil is also accumulated in the CHD and CBD (not shown). **D** CT obtained at 6 months after TACE shows intrahepatic bile duct dilatation in the right lobe of the liver. The gallbladder markedly shrinks and concentrated bile is retained. Gallstone is also seen. **E** CT obtained before TACE shows a normal appearance of the gallbladder without gallstone

PTBD was performed under sonographic guidance. The transhepatic approach was performed from the left in 4 patients and bilaterally in 2 patients. Four patients (66.7%) showed strictures of the bilateral hepatic ducts (Fig. 5); 1 patient (16.7%) showed stricture of the CHD; and 1 patient (16.7%) showed stricture of the LHD. Intraluminal defects suggesting coagula were seen in 4 patients (66.7%). In all patients, the occluded or stenotic segment was relatively short and smooth on cholangiography obtained by MRI, ERCP, and/or PTBD (Figs. 2, 5).

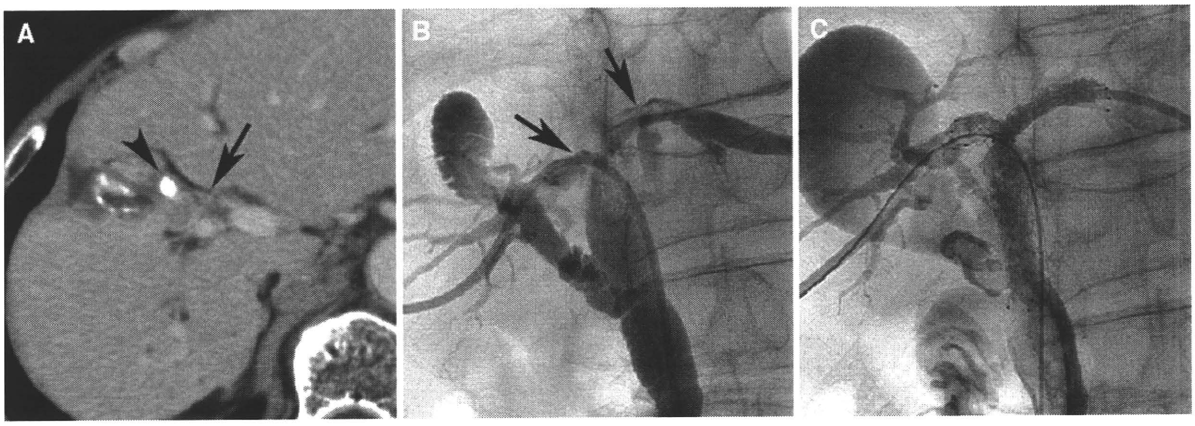

Fig. 5 A 68-year-old man with bile duct strictures at the hepatic hilum (patient no. 17). **A** CT obtained before the additional fourth TACE session (at 22 months after demonstration of bile duct dilatation) shows the stricture and wall thickening of the CHD (*arrow*) in addition to intrahepatic bile duct dilatation. The *arrowhead* indicates an iodized oil accumulated tumor. **B** This patient presented with jaundice 2 weeks after the additional fourth TACE and PTBD was performed. Cholangiogram obtained through the PTBD catheter shows strictures at the bilateral hepatic ducts and CHD (*arrows*). **C** Two metallic stents were placed by way of a bilateral approach

Changes in Laboratory Data After TACE

Before TACE, serum ALP level was high in 6 patients, and γ-GTP level was high in 5 patients. Serum ALP levels increased in 13 patients (72.2%) from 345.1 ± 89.6 U/L (range 244 to 591) to 582.1 ± 198.7 U/L (range 378 to 1083) at 1 week to 14 months (mean 3.7 ± 4.1) after the last TACE session before bile duct dilatation. Five patients were classed as showing a slight increase, 3 as having a moderate increase, and 5 as having a marked increase. In 1 patient with a slight increase and in all 5 patients with a marked increase, jaundice developed during the follow-up period. Cholangitis developed in 1 patient showing a moderate increase. In the same patients, serum γ-GTP levels increased from 71.8 ± 57.8 U/L (range 29 to 233) to 245.2 ± 169.3 U/L (range 107 to 604) at 1 week to 14 months (mean 4.2 ± 3.9) after the last TACE before bile duct dilatation. Five patients were classed as showing a slight increase, 4 as having a moderate increase, and 4 as having a marked increase. The grades of increase in ALP and γ-GTP were well correlated. Jaundice developed in 1 patient with a slight increase, in 2 patients with a moderate increase, and in 3 patients with a marked increase. Cholangitis developed in 1 patient showing a marked increase. In 9 of 13 patients, serum levels of these enzymes gradually increased. In 5 patients (27.8%), there were no remarkable changes despite bile duct dilatation in the right lobe ($n = 3$), in S2 ($n = 1$), and in S3 ($n = 1$).

Clinical Courses and Outcomes

All patients were followed-up for 3 to 129 months (mean 38.2 ± 35.4) after the initial TACE of A1 and/or A4 and for 3 to 72 months (mean 25.2 ± 17.6) after the last TACE before bile duct dilatation. Eleven patients (61.1%) presented with bile duct complications associated with main bile duct stricture. Cholangitis developed in 2 patients (11.1%) at 2 months after TACE and resolved after administration of antibiotics (Sulperazon; Pfizer, Tokyo, Japan). Bile duct dilatation was improved during follow-up in these patients (Fig. 1). Biloma developed in 4 patients (22.2%), and percutaneous drainage was performed in 2 of 4 patients at 2 and 4 months, respectively, after the last TACE before bile duct dilatation.

Jaundice (serum bilirubin level 9.3 to 14.0 mg/dL [mean 11.7 ± 2.3]) developed in 4 patients (22.2%), including 2 with a history of biloma drainage, at 5 to 11 months (mean 7.3 ± 2.9) after the last TACE before bile duct dilatation. Three patients underwent PTBD. In the remaining patient, bile duct drainage was not performed because of the patient's poor general condition. In another patient without jaundice, PTBD was performed to relieve bile duct dilatation before additional TACE with bile duct dilatation for HCC in the left lobe of the liver. Metallic stent placement (Zilver 635; Cook) was subsequently performed in 4 patients. In 2 patients, 2 metallic stents were placed from the bilateral hepatic ducts to the CBD using either a bilateral or left approach. In 2 patients, 1 metallic stent was placed from the LHD to CHD by way of a left approach. The PTBD catheter was removed after stent placement.

In 13 patients (72.2%), including 4 who underwent stent placement, 1 to 5 additional TACE sessions (mean 2.6 ± 1.5) were performed because of local recurrence and/or new lesions at other sites. In total, 24 TACE sessions to the area with bile duct dilatation or stented lobe were performed in 12 patients, and 10 TACE sessions to the area without bile duct dilatation were performed in 5 patients.

Complications associated with additional TACE sessions occurred in 7 of 13 patients (53.8%). Biloma developed in 3 patients. It developed in 2 patients at 2 months after the additional first TACE session to the area with bile duct dilatation or stented lobe and in 1 patient at 1 month after 4 additional TACE sessions to the stented lobe. Percutaneous drainage was required in 1 patient with previous stent placement. Jaundice (6.5 to 22.6 mg/dL [mean 12.6 ± 6.7]) developed in 5 patients at 3 to 26 months (mean 15.4 ± 19.7) after the first additional TACE session and at 2 weeks to 5 months (mean 2.8 ± 1.8) after the last additional TACE session, including 2 patients who had undergone stent placement. It developed after 1 ($n = 2$), 3 ($n = 1$), and 4 ($n = 2$) additional TACE sessions to the area with bile duct dilatation ($n = 3$) or to the stented lobe ($n = 2$). One of these patients also had a small biloma. All patients but 1 underwent PTBD. In 1 patient, 1 metallic stent was placed from the LHD to CBD using a left approach. In another patient, 2 metallic stents were placed from the bilateral hepatic ducts to CBD using a bilateral approach (Fig. 5). The bile duct was occluded in the stented segment in 2 patients who had previously undergone stent placement. Jaundice did not improve because of hepatic failure, and external drainage was continued until the patients' death. In the remaining patient, PTBD was not performed because of the patient's poor general condition. Cholangitis recurred in 1 patient at 1 month after stent placement without an additional TACE session, and it did not improve despite administration of antibiotics. One patient who had undergone stent placement to relieve bile duct stricture before additional TACE sessions did not develop any complications despite 4 additional TACE sessions, including 2 to the stented lobe.

Seven patients (38.9%) have not presented any symptoms for 3 to 68 months (mean 28.6 ± 24.3) since the development of bile duct stricture, and 3 patients (16.7%) including 2 who underwent metallic stent placement have not shown any symptoms associated with bile duct stricture for 3 to 17 months (mean 9.0 ± 7.2) since the additional intervention.

Eight patients (44.4%), including 3 who underwent stent placement ($n = 5$), died of hepatic failure ($n = 2$), uncontrollable jaundice, cholangitis, and/or tumor progression ($n = 1$) at 11 to 119 months (mean 37.9 ± 34.9) after the initial TACE of A1 and/or A4 and at 11 to 41 months (mean 21.3 ± 10.4) after the last TACE session before bile duct dilatation. Ten patients (55.6%) have survived 3 to 129 months (mean 38.4 ± 37.9) after the initial TACE of A1 and/or A4 and 3 to 72 months (mean 28.4 ± 21.9) after the last TACE session before bile duct dilatation. Four patients have not shown any viable HCCs, but 6 patients have viable HCCs despite repeated TACE sessions.

Discussion

TACE plays as an important role in the treatment of unresectable HCC [1–8]. However, several complications associated with TACE have been reported [9–18]. Main bile duct necrosis, followed by stricture, is one of the severe complications of TACE [16–18]. The liver is dually supplied by hepatic arterial and portal venous blood, whereas HCC is mainly supplied by arterial blood. When the tumor-feeding hepatic arterial branch is blocked, the tumor may develop ischemic necrosis, whereas the normal liver may only be slightly affected [1]. However, the biliary tree is supplied primarily by arterial blood alone [19]; therefore, it may be damaged by TACE. Chronic inflammation eventually leads to extensive focal fibrosis of large bile ducts [17].

The extrahepatic and intrahepatic bile ducts are surrounded by a vascular plexus, which is composed of branches arising directly from the right and left hepatic arteries and their segmental branches and indirectly from the gastroduodenal artery by way of the arteries supplying the CBD [20, 21]. The plexus around the RHD and LHD is continuous, with a similar plexus surrounding the CBD and CHD. This intrahepatic plexus is always closely associated with the arterial supply of S1, and A1 gives several branches to the hepatic duct plexus [20].

There is a communicating arcade between the right and left hepatic arteries. This arcade originates mainly from A4 on the left side and from the anterior branch of the right hepatic artery or the right hepatic artery on the right side and supplies S1 and the hilar bile duct [22]. Vellar [23] also reported that A4 is the most important blood supply to the LHD. We speculate that the right branch of the communicating artery, which was described in a report by Tohma et al. [22], might have been A1 in our previous report [24]. When embolic materials are injected into 1 of these vessels, they may flow into another vessel through this communication. In the present study, communications between A1 and A4, between A1 and the 3 o'clock and 9 o'clock arteries, and between two separate branches of A1 or A4 were also demonstrated during the TACE procedure. These communications may explain frequent iodized oil inflow in the posterior aspect of S4 seen on CT obtained after TACE of A1 [24].

Kim et al. [17] reported that focal strictures at the CHD level, or in hepatic ducts adjacent to the CHD level, were seen in their series of bile duct injury by TACE. In the present study, all strictures were also located at the CHD and/or the RHD or the LHD. This may reflect the vascular supply of the CHD and bilateral hepatic ducts from the hepatic arterial branches, including A1 and A4, whereas the CBD is mainly supplied by the branches of the gastroduodenal artery [20, 21]. The poor collateral supply to the CHD, compared with a denser arterial network and a relatively high proportion of supply to the lower CBD, could explain the vulnerability of the CHD to ischemic injury [21]. All strictures were focal and smooth, and intraluminal coagula was frequently detected on CT, MRI, and cholangiogram. We consider that these findings may be typical image findings of bile duct injury by TACE. Radiologists should not misdiagnose this condition as bile duct invasion by the tumor.

Kobayashi et al. [19] reported that TACE or hepatic arterial infusion chemotherapy might cause reduction of the inner layer vessels of the peribiliary capillary plexus. Kim et al. [17] and Bang et al. [18] also reported that repeated TACE sessions were a risk factor for ischemic bile duct injury. In the present study, all main bile duct strictures developed after TACE of A1 and/or A4. In addition, 50% of bile duct strictures developed even after a single TACE session of A1 and/or A4, suggesting that selective TACE of A1 and/or A4 is a main cause of bile duct necrosis. However, nonselective TACE of the right hepatic artery or left hepatic artery level may mainly damage "the peripheral branches," whereas "the central branches," such as A1 and A4, may be slightly damaged or occasionally preserved. Repeat TACE sessions may finally damage these central branches because the peripheral branches are severely attenuated by previous TACE procedures, and embolic materials flows mainly into these central branches.

Makuuchi et al. [16] reported that the intrahepatic and extrahepatic bile ducts would become necrotic if small particles <250 μm were used. In a report by Chen et al. [21], the artery that supplies the RHD and the LHD has an average diameter of 0.33 ± 0.15 mm. In the present study, gelatin sponge particles approximately 0.5 to 1 mm in diameter caused bile duct injury, although there was a possibility of contamination by small fragments <250 μm. We speculate that selective TACE of A1 and/or A4 presents a risk of developing bile duct stricture regardless of the size of gelatin sponge particles. The incidence of main bile duct necrosis by selective TACE of A1 and/or A4 was approximately 6% in the present study. This incidence may have been influenced by the magnitude of TACE, the position of the catheter tip, the patterns of arterial supply of the main bile duct, and the damage to the peribiliary plexus and collaterals by previous TACE sessions. In addition, the presence of multiple branches of A1 and A4 may salvage bile duct ischemia by acting as collateral circulation [25]. We speculate that the use of smaller particles may not significantly increase the incidence of main bile duct stricture, except when these are selectively injected into A1 and/or A4.

With advancements in catheter and guidewire technology, it has become possible to introduce a microcatheter into small branches of the hepatic artery [5–8]. Iodized oil and gelatin sponge particles injected with slight force through the microcatheter may distribute more distally rather than corresponding with natural arterial blood flow [8]. We reported that ultraselective TACE has strong therapeutic effects to achieve not only complete tumor necrosis but also massive peritumoral necrosis by blockage of both the hepatic arterial and portal blood flow [26]. During selective TACE of A1 and/or A4, injection of embolic materials with slight force may increase the risk of bile duct necrosis because embolic materials may flow into the vascular plexus around the main bile ducts directly or indirectly through anastomosis. Chen et al. [21] reported that the marginal artery of the CHD connects with the cystic artery. We speculate that embolic materials injected from A1 or A4 may also flow into the cystic artery through the anastomosis, and thus shrinkage of the gallbladder may occur; this was observed in 22% of patients in the present study.

It may be difficult to predict main bile duct injury by TACE. In 3 patients undergoing selective CBCT during the TACE procedure of A1 and/or A4, bile duct wall enhancement was clearly demonstrated. CBCT might be useful for predicting bile duct injury caused by TACE. We should always bear in mind that selective TACE of A1 and/or A4 carries the risk of causing bile duct necrosis. Therefore, complete blockage of these vessels should be avoided, especially when bile duct enhancement is seen on CT or CBCT during arteriography through A1 or A4. In addition, distal advancement of a thinner microcatheter with a tip <2F into A1 or A4 may be useful to avoid embolization of small vessels supplying the main bile ducts because these vessels may be estimated to arise from the proximate portion of A1 and A4. CT obtained at 1 week after TACE may also be useful to suggest main bile duct damage. Changes in laboratory data are not sufficiently sensitive, and abnormalities may become apparent long after demonstration of bile duct dilatation on CT in most patients, although marked changes suggest severe damage to the main bile ducts.

Optimal management of main bile duct stricture occurring after TACE has not yet been established. In previous reports, surgical resection was performed when jaundice developed [16–18]. In the present study, we treated 6 patients using metallic stents. Cholangitis and jaundice recurred in 3 patients after stent placement, including 2 who underwent repeated TACE sessions to the stented segment. In addition, a large biloma developed in 1 patient after an additional TACE session performed after stent placement. Metallic stent placement may be useful to manage main bile duct stricture caused by TACE because patients with inoperable HCC are not good candidates for surgical bile duct repair. However, bile duct complications may occur at high rates after additional TACE sessions. In addition, we should be well aware that intrahepatic bile duct dilatation is a significant risk factor in developing biloma after TACE [15].

In conclusion, selective TACE of A1 and/or A4 presents a risk of causing main bile duct stricture at the hepatic hilum regardless of the number of TACE sessions and the particle size of the embolic material. Focal and smooth bile duct strictures mainly at the CHD are characteristic findings, and intraluminal coagula are frequently detected on CT, MRI, and cholangiography. Radiologists should well aware this uncommon but severe complication of TACE.

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1. 肝外側副路経由のTACE

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はじめに

肝外側副路からの肝細胞癌への供血の主因は，肝動脈化学塞栓術(transcatheter arterial chemoembolization : TACE)などによる肝動脈損傷，手術や治療後の炎症性癒着であるが，腫瘍の大きさや位置によっては発見時からすでに肝外側副路が関与することもある。以前の我々の検討では[1]，肝外側副路からの供血は16.6%で認められ，発現頻度は右下横隔動脈，胆嚢動脈，大網動脈，左下横隔動脈，右内胸動脈，右肋間動脈の順であった。リピオドール抗癌剤混合液を使用したTACE (以下Lip-TACE) の成功率は，右肋間動脈で53%と最も低く，他は70～100%であったが[1]，近年のカテーテルや血管撮影装置の進歩により，内胸動脈や腰動脈の発現頻度が増加し，TACEの成功率も向上している。

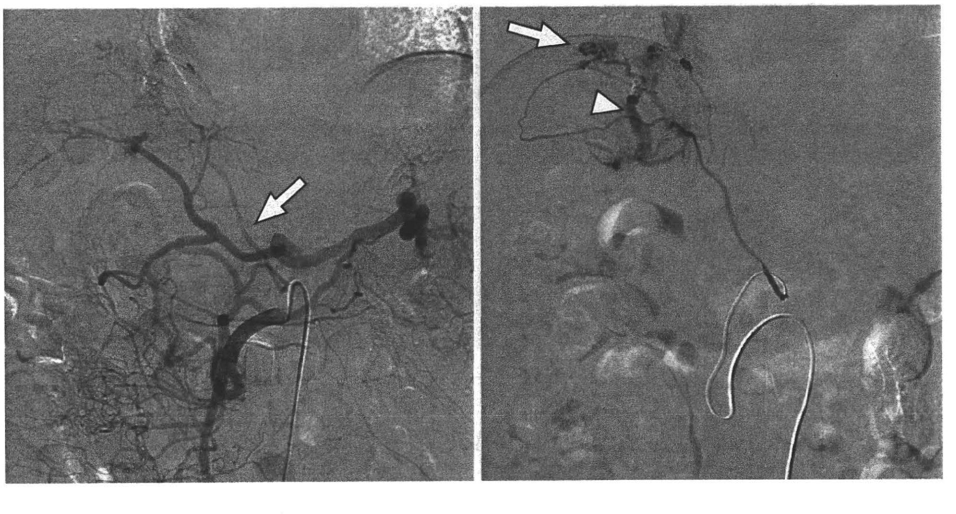
下横隔動脈

右下横隔動脈は無漿膜野を介して右葉背側や尾状葉，左下横隔動脈は左葉外側区域辺縁部の腫瘍を栄養するが[2]，まれに左下横隔動脈が左葉内側区域や右前区域の腫瘍を栄養する。下横隔動脈の40%は腹腔動脈，39%は大動脈，15%は腎動脈，4%は左胃動脈，2%は肝動脈から分岐する[2]。腹腔動脈から分岐する場合，起始部から左下横隔動脈，右下横隔動脈，左胃動脈の順に分岐する。また大動脈から分岐するものは，動脈硬化や弓状靭帯の圧排，以前のカテーテル操作などで起始部が閉塞し，種々の血管から再建される。描出頻度は背側膵動脈(**図1**)，副腎動脈，左胃動脈，対側の下横隔動脈の順で[3]，肋間動脈や腰動脈，内胸動脈，肝動脈からも描出される[4]。複数の血管から描出される場合，屈曲の少ない経路からアプローチすることでTACEの成功率は向上する[3]。

左下横隔動脈からは胃枝や食道枝が分岐し，ときに右下横隔動脈からも分岐する。肺の慢性炎症が存在する場合は，内胸動脈の枝である心横隔動脈や肺血管との吻合や短絡が認められる[2]。これらの枝にリピオドールが流入しないように塞栓するが，回避できない場合はゼラチンスポンジのみで塞栓する。合併症は胸水や肺梗塞に加えて，吻合を介した肋間動脈へのリピオドールの流入による皮膚壊死や脊髄梗塞の危険がある。

胆嚢動脈

胆嚢動脈は主に胆嚢床部の腫瘍を栄養するが，肝動脈損傷が強い場合は肝の深部まで栄養する[5]。胆嚢動脈深在枝は右肝動脈前下区域枝(A5)や左葉内側区域枝(A4)と吻合しており[4]，またしばしば胆嚢動脈からA5の小枝が分岐する。胆嚢壁の染まりが認められない位置までカテーテルが挿入できればLip-TACEを行うが(**図2**)，困難な場合はTACEを断念する[5]。上述の吻合を介してA4やA5の塞栓で胆嚢炎が生じたり，逆に胆嚢動脈が塞栓された際にも重篤な胆嚢炎の発症が回



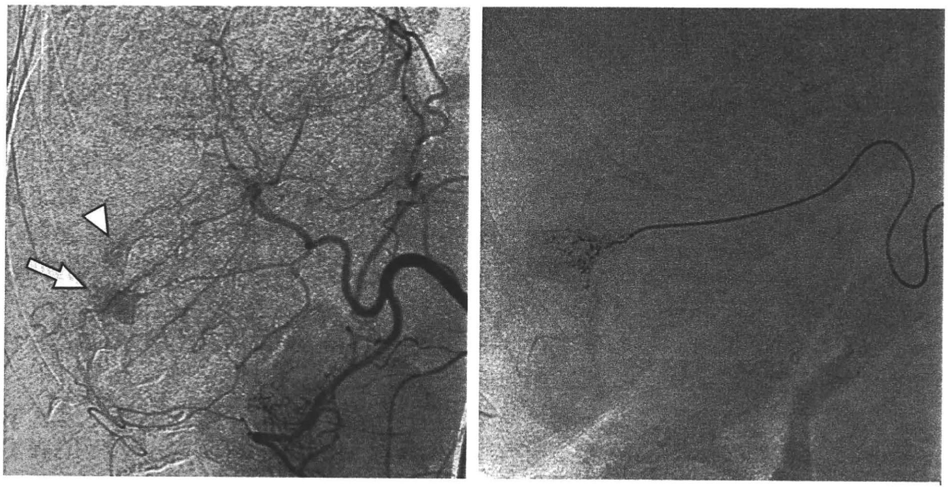
a | b

図1　背側膵動脈より描出される右下横隔動脈
a：上腸間膜動脈造影にて腹腔動脈は膵のアーケードを介して描出され，右下横隔動脈は背側膵動脈から描出されている(矢印)。
b：選択造影で腫瘍濃染（矢印）と動脈門脈短絡を認める(矢頭)。

避されている可能性がある。

右腎被膜動脈・副腎動脈

腎被膜動脈や副腎動脈は通常複数本存在し，互いに吻合しながら主に右腎窩周囲の腫瘍を栄養するが（図3, 4），尾状葉枝などと吻合して肝の深部まで栄養することがある[4]。腎動脈末梢から腎被膜を貫通する小枝も腫瘍の栄養血管となり，特に腎被膜動脈や副腎動脈のTACEを繰り返すうちに顕著化する[6]。選択できた場合にはLip-TACEを行うが，右肋間動脈や背側膵動脈

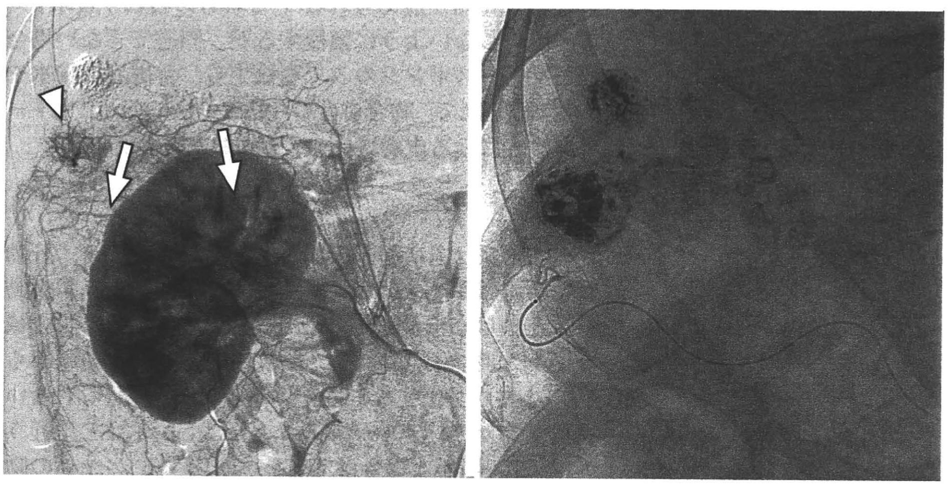


a | b

図2　胆嚢動脈より栄養される肝癌

a：腹腔動脈造影にて胆嚢動脈より栄養される腫瘍を認める（矢印）。他に肝動脈から栄養される小さな腫瘍を認める（矢頭）。

b：胆嚢壁が描出されない位置までカテーテルを進めTACEを施行した。



a | b

図3　右腎被膜動脈より栄養される肝癌

a：右腎動脈造影にて右腎被膜動脈（矢印）より栄養される腫瘍を認める（矢頭）。

b：カテーテルを末梢まで進めTACEを施行した。

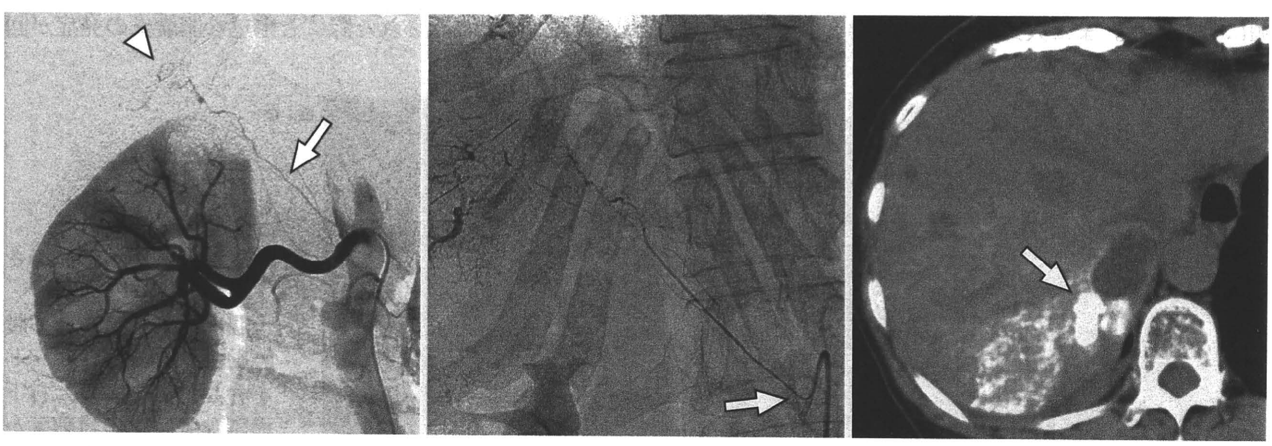


図4　右中副腎動脈より栄養される肝癌　a | b | c

a：右腎動脈造影にて右中副腎動脈（矢印）より栄養される腫瘍を認める（矢頭）。

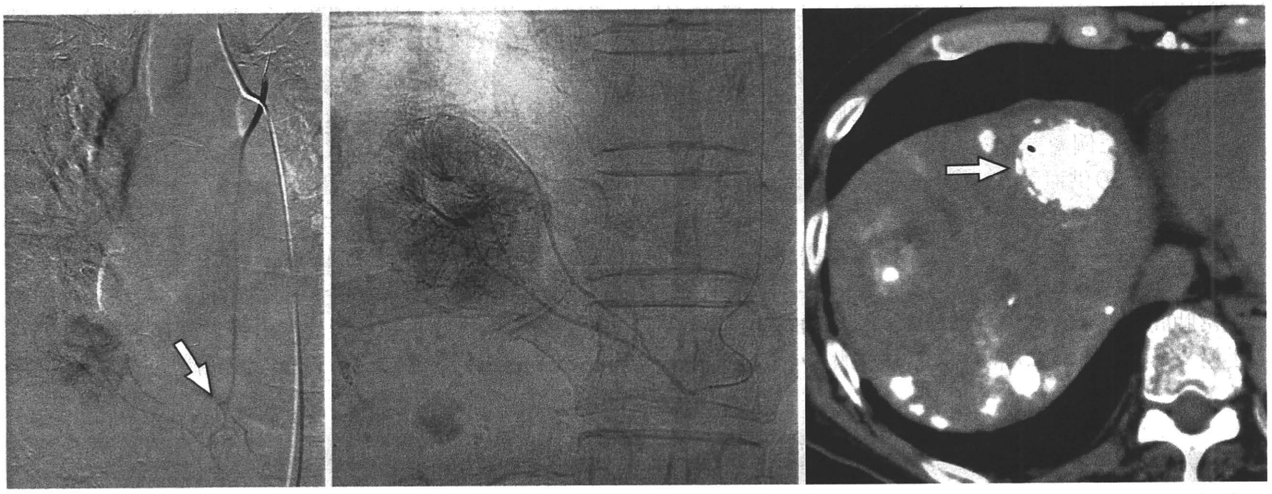
b：シェファードフックカテーテルに側孔（矢印）をあけて選択し，TACEを施行した。

c：TACE 1週間後のCTにて腫瘍には高濃度にリピオドールが集積している（矢印）。

などとも吻合するため[4]，腎梗塞の他に，皮膚壊死や脊髄梗塞，膵炎などの危険がある。

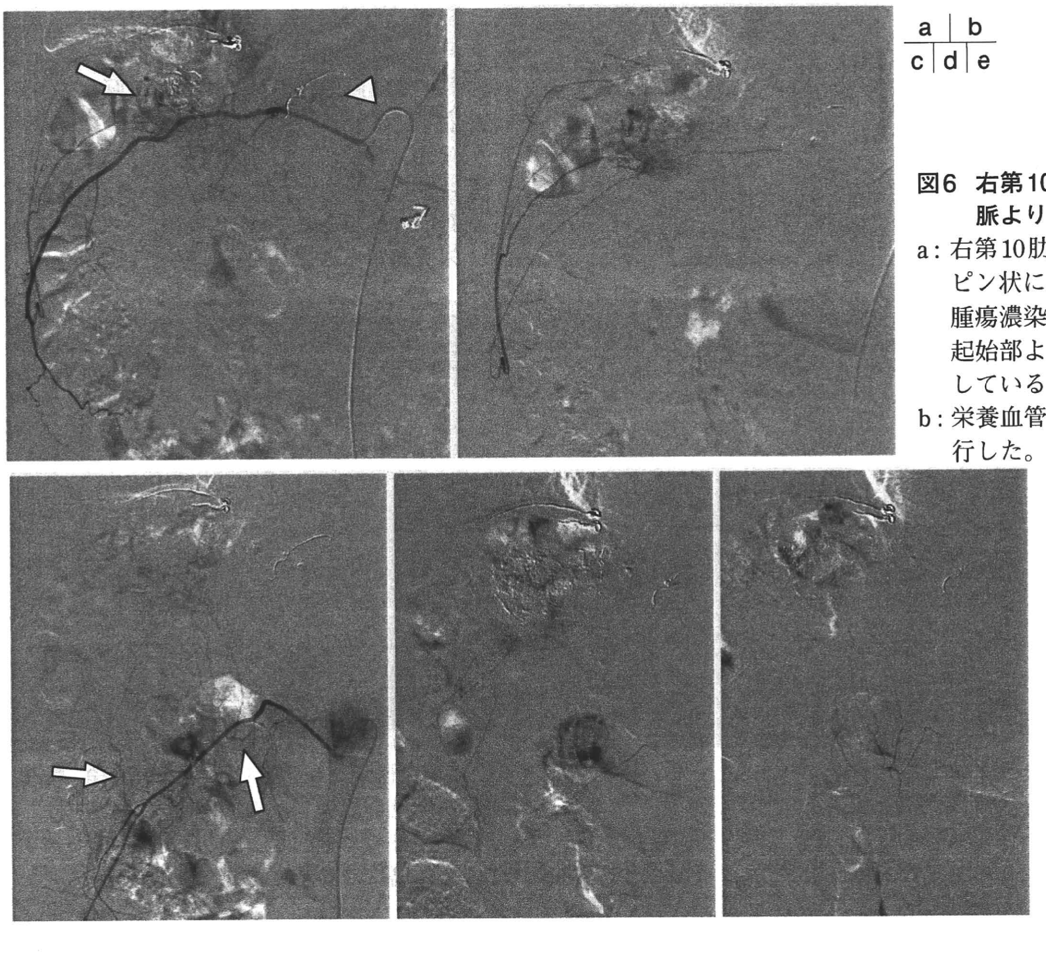
内胸動脈

内胸動脈は主に腹側の肝表に存在する腫瘍を栄養するが，まれに左内胸動脈が右側の腫瘍を栄養する(図5)。腫瘍への供血頻度は，横隔枝，筋横隔動脈，上腹壁動脈の順であり[7]，主に下横隔動脈からのTACE後の再発病変で関与が認められる[1,7]。内胸動脈が大動脈から単独分岐したり，横隔枝が高位から分岐する破格がある。



a|b|c

図5　左内胸動脈より栄養される肝癌
a：左内胸動脈造影にて横隔枝(矢印)より栄養される腫瘍を認める。
b：マイクロカテーテルを進めTACEを施行した。
c：TACE 1週間後のCTにて腫瘍には高濃度にリピオドールが集積している(矢印)。



a|b
c|d|e

図6　右第10肋間動脈と肋下動脈より栄養される肝癌
a：右第10肋間動脈造影にてヘアピン状に屈曲する栄養血管と腫瘍濃染を認める(矢印)。尚，起始部より前脊髄動脈が分岐している(矢頭)。
b：栄養血管を選択しTACEを施行した。
c：右肋下動脈造影にて筋枝から分岐する2本の栄養血管を認める(矢印)。
d, e：それぞれを選択しTACEを施行した。

内胸動脈でのカテーテル操作は，椎骨動脈の起始部が近傍にあるため，必ず全身へパリン化した後に行う。栄養血管が選択できればLip-TACEを行うが，前肋間動脈や上腹壁動脈に流入すると皮膚壊死を生じる危険があり，それらが避けられない場合にはゼラチンスポンジのみで塞栓を行う。

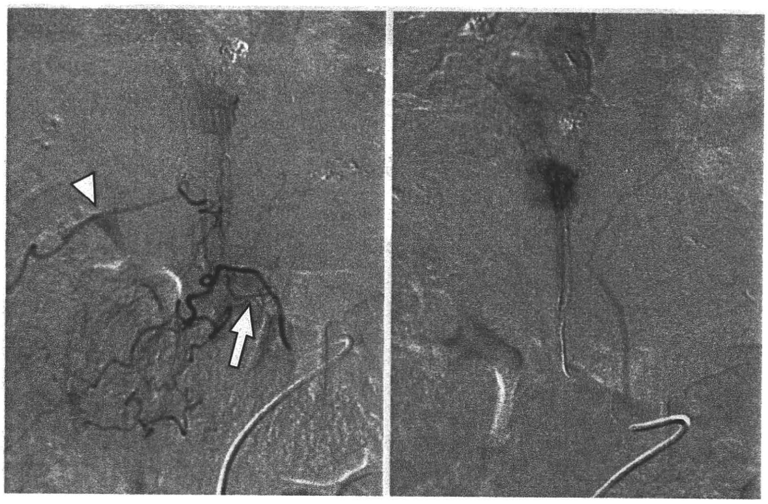
肋間動脈・肋下動脈・腰動脈

これらの血管は下横隔動脈や右腎被膜動脈とほぼ同じ領域を栄養するが，以前に下横隔動脈や右腎被膜動脈が塞栓された例や，腹壁に浸潤する腫瘍で主に関与する。腫瘍への供血頻度は第10，9，11肋間動脈，肋下動脈，第1腰動脈の順であるが[1]，これらの間には豊富な吻合が存在するため，最初に一通り撮影し，前脊髄動脈の分岐位置を確認する[8]。

通常肋間動脈からの肝を栄養する枝は肋骨軟骨接合部近傍でヘアピン状に屈曲し，肋下動脈からの栄養枝は筋枝から急峻に分岐する(図6)。腰動脈からの栄養血管は主に背側枝と筋枝の分岐部の少し手前から分岐する[8](図7)。栄養血管が選択できた場合にのみLip-TACEを行うが，塞栓物質のoverflowによる脊髄梗塞や皮膚壊死の危険がある。栄養血管が選択不能で前脊髄動脈が分岐していない場合は，大きめのゼラチンスポンジのみで塞栓する。前脊髄動脈が描出される血管から腫瘍血管が分岐する場合，栄養血管が選択できれば塞栓は可能であるが，適応は慎重に決定する(図6)。

消化管・膵・胆管動脈

大網動脈，左・右胃動脈(図8, 9)，中・右結腸動脈(図10)，背側膵動脈(図11)，胆管動脈(3時9時動脈)(図12)などは，肝表，尾状葉，肝門部の腫瘍を栄養する。大網動脈は主に右胃大網動脈から分岐するものが関与するが，まれに左胃大網動脈も関与する(図13)。また，腫瘍破裂例に関与したり腹膜播種病変を栄養す



a | b

図7 右第1腰動脈より栄養される肝癌
a：右第1腰動脈造影にて背側枝と筋枝の分岐部の手前から分岐する枝(矢印)に栄養される腫瘍を認める。また吻合を介して右肋下動脈が描出されている(矢頭)。
b：栄養血管を選択しTACEを施行した。

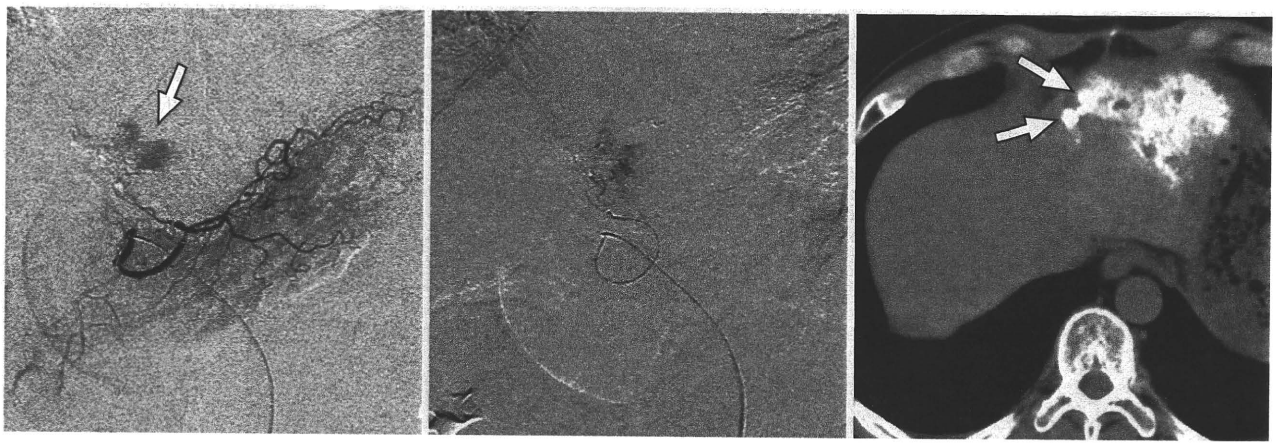
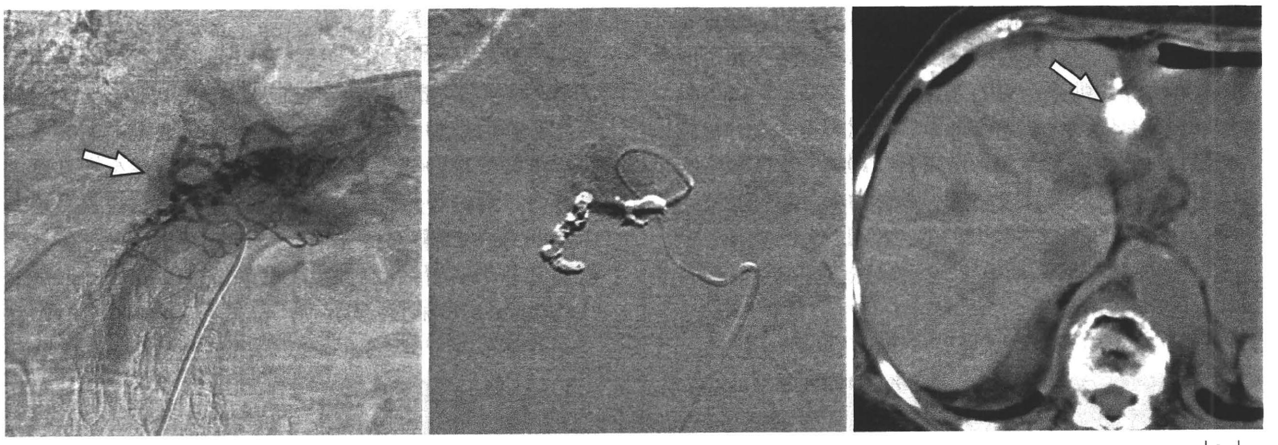


図8 右胃動脈より栄養される肝癌

a | b | c

a：右胃動脈造影にて腫瘍濃染を認める(矢印)。
b：栄養血管を選択しTACEを施行した。
c：左葉外側区域枝からもTACEが追加された。TACE 1週間後のCTにて腫瘍には高濃度にリピオドールが集積している(矢印)。



a | b | c

図9　左胃動脈より栄養される肝癌

a：左胃動脈造影にて腫瘍濃染を認める(矢印)。

b：栄養血管が選択できなかったため，3本の胃枝をコイルで塞栓した。その後の造影にて胃壁の染まりがないことを確認し，TACEを施行した。

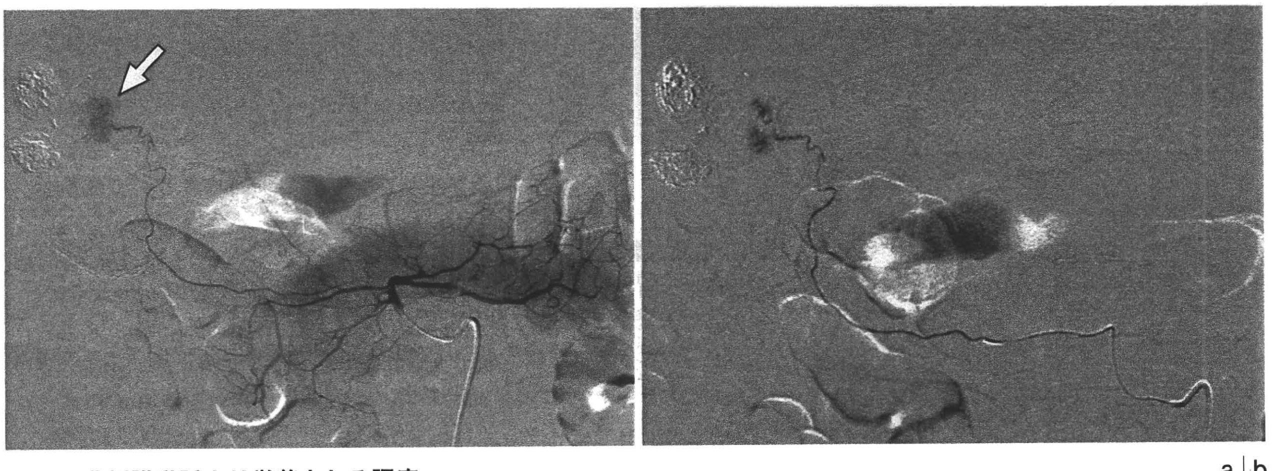
c：TACE 1週間後のCTにて腫瘍には高濃度にリピオドールが集積している(矢印)。

a | b

図10　右結腸動脈より栄養される肝癌

a：上腸間膜動脈造影にて右結腸動脈より栄養される腫瘍を認める(矢印)。

b：栄養血管を選択しTACEを施行した。



a | b

図11　背側膵動脈より栄養される肝癌

a：背側膵動脈造影にて腫瘍濃染を認める(矢印)。

b：膵実質の染まりのなくなる位置までカテーテルを進め，TACEを施行した。

ることもある[9]。胆管動脈は後上膵胃十二指腸動脈から急峻に分岐し，尾状葉枝や肝門部の小枝と吻合する。

消化管や膵の動脈からのTACEでは潰瘍や膵炎の危険があり，消化管壁や膵の染まりが認められない位置までカテーテルを挿入できた場合にのみLip-TACEを行うが，困難な場合はTACEを断念する。胆管動脈からのTACEでは胆管壊死の危険があり，大網動脈は消化管の動脈と吻合するため[4]，大網動脈の塞栓では腸管壊死の危険がある。

肝外側副路経由のTACEに対する考え方

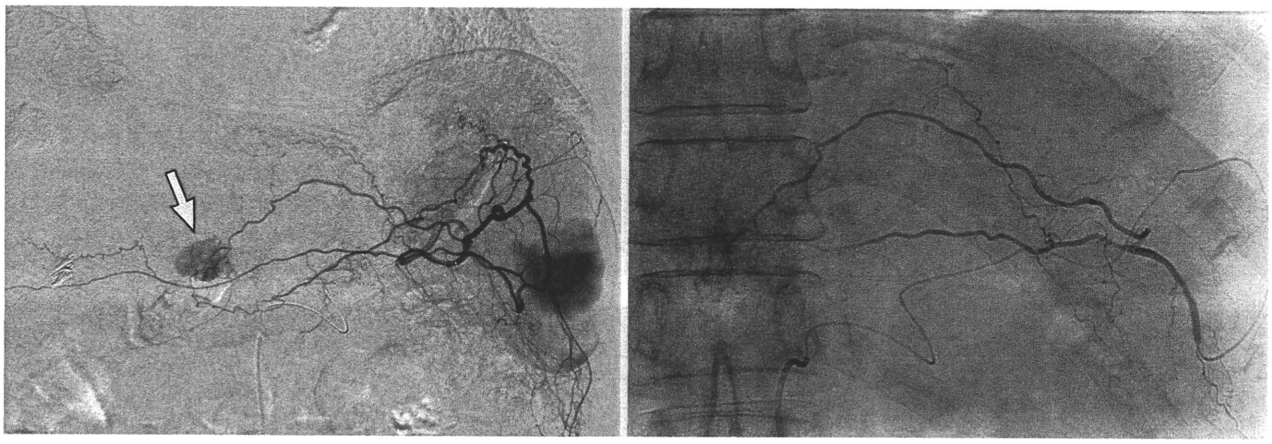
肝外側副路間には豊富な吻合があり，それに栄養される腫瘍は潜在的に複数の栄養血管を有している[1]。特に無漿膜野の腫瘍では，TACE後の再発を繰り返すたびに，別の肝外側副路の関与が認められることが多い[6]。同部位の腫瘍は，初回は肝動脈支配で一部は右下横隔動脈や右腎被膜動脈などのmajor diaphragmatic circulationから栄養されるが，再発時はmajor diaphrag-

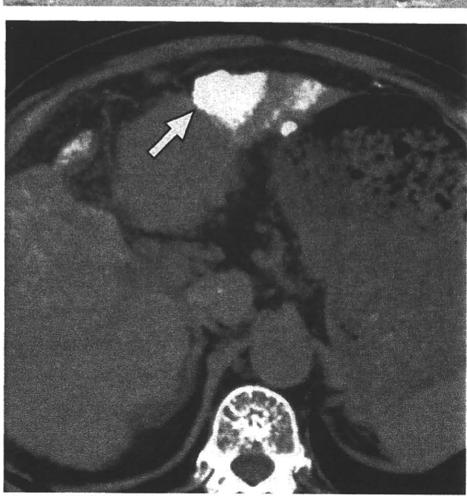
a | b

図12 胆管動脈（3時9時動脈）より栄養される肝癌

a：後上膵胃十二指腸動脈造影にて胆管動脈（矢印）より栄養される腫瘍を認める（矢頭）。

b：マイクロカテーテルを進めTACEを施行した。





a | b
c |

図13 左胃大網動脈より栄養される肝癌

a：左胃大網動脈造影にて腫瘍濃染を認める（矢印）。

b：栄養血管を選択しTACEを施行した。

c：TACE 1週間後のCTにて腫瘍には高濃度にリピオドールが集積している（矢印）。