

Original Article

Hepatic Infarction Following Abdominal Interventional Procedures

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To clarify the incidence, background, and progress of hepatic infarction following interventional procedures, cases of hepatic infarction following interventional procedures at our department during the last decade were identified by reviewing the clinical records of 1982 abdominal angiography and interventional procedures and records of abdominal CT. Nine episodes (0.5%) in 8 patients were identified as hepatic infarction following an interventional procedure. Five episodes were preceded by embolization of the hepatic or celiac artery at emergency angiography for postoperative bleeding with hemorrhagic shock. Three episodes followed the elected interventional procedure for hepatocellular carcinoma, and the remaining episode occurred after 12 months of chemoinfusion through an indwelling catheter in the hepatic artery and portal vein. Hepatic arterial occlusion in all episodes and portal venous flow abnormality in 5 episodes were observed on angiography. Four patients whose liver function was initially impaired died of hepatic infarction, although the extent of the disease on CT did not appear to be related to the mortality. Multiple risk factors, including arterial insufficiency, were observed in each patient. The incidence of hepatic infarction following interventional procedures in this series was low but sometimes fatal, and occurred most frequently in emergency embolization in hemorrhagic shock.

Key words: liver, infarction, interventional procedure, angiography, computed tomography

Hepatic infarction is believed to be relatively rare given the dual blood supply and abundant collateral pathways to the liver [1-3]. However, sporadic cases of hepatic infarction following interventional procedures have been reported [4], and some have been critical [5-7]. To reduce the incidence of hepatic infarction and to improve therapeutic effectiveness, the indications for interventional procedures of the liver have been determined according to an extensive evaluation of liver

hemodynamics [8, 9]. Nonetheless, although the possibility of hepatic infarction is always considered prior to intervention, and the incidence is quite low, the onset of hepatic infarction cannot be completely avoided [10]. To our knowledge, there have been no retrospective studies of hepatic infarction focusing on the period after interventional procedures in consecutive patients. In the present study, we reviewed cases of hepatic infarction following interventional procedures at our institute over the last decade in order to clarify the incidence, background, and progress of this phenomenon.

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Materials and Methods

During the last decade (January 1993 to December 2002), 1982 abdominal angiography and interventional procedures were performed in 1309 patients in our department. We initially performed a retrospective search of the abdominal angiography findings and interventional records of all patients, and then reviewed records of the related abdominal CT findings. Hepatic infarction was diagnosed entirely based on CT findings, laboratory data, and symptoms of hepatic infarction. These investigations were performed by two radiologists (H.F., S.K.), and the determination of patients with hepatic infarction following an interventional procedure was based on their consensual agreement.

After identification of suitable patients, we extensively reviewed patients' backgrounds and the interventional procedures undergone. Angiography, CT, and patient progress were also reviewed. Using that data, the existence of previously reported risk factors of hepatic infarction, including shock, liver transplantation, extensive abdominal surgery, general anesthesia, biliary disease, and cardiac disease [11-16] was determined in each patient.

When angiography was reviewed, prominent findings concerning flow changes in the hepatic artery and/or portal vein were evaluated. During the reviews, three radiologists (H.F., S.K., T.H.) analyzed the angiography findings in a combined review session with knowledge of patient history and the prospective study interpretation. Consensual agreement regarding the angiography findings was reached for each patient. CT scans were analyzed by the same radiologists using the same methodology as for the angiograms. Consensual agreement was reached in each case regarding the presence, shape, and distribution of perfusion defects, and the patency of the hepatic arteries and portal veins.

Levels of serum total bilirubin, aspartate aminotransferase, alanine aminotransferase, albumin, and PT% immediately before (day -1), immediately after (day +1 or 2), and 2 weeks after (day +14) the onset of hepatic infarction in each patient were derived from laboratory data and evaluated to determine the relationship between these data and prognosis. Peak values were observed 1 or 2 days after onset, and the larger value was adopted (Table 2). These data were not available for patient 2.

Results

Determination of patient cohort. Our retrospective analysis of medical records revealed that 8 patients (6 men, 2 women, age; 39-76 years, mean age; 60 years) developed hepatic infarction following an interventional procedure (Table 1). Of these 8, 1 (patient 7) had 2 episodes of infarction preceded by emergency interventional procedures. Thus, 9 episodes in 8 patients were determined as hepatic infarction following interventional procedures. Diagnosis was based on the coexistence of geographic, low-attenuation perfusion defects without mass effect on CT as well as the following findings: clinical symptoms of abdominal pain ($n=7$) and fever ($n=7$), acute increases in the serum alanine aminotransferase and C-reactive protein (CRP) levels with the hepatic injury (elevated in all 7 patients in whom these tests were performed), normal CT image of the liver before the hepatic injury (all patients), and follow-up CT scans demonstrating the expected patterns of infarct evolution (all patients).

Patients backgrounds and types of interventional procedures. Five of all 9 episodes (55.6%) were preceded by embolization during emergency arteriography (Table 1). Three patients who had undergone surgical procedures because of advanced abdominal carcinoma showed massive hemorrhage following the procedures (patients 4, 5, and 6) (Fig. 1). One patient who had undergone liver transplantation due to liver cirrhosis experienced 2 episodes of massive hemorrhage from the Penrose tube after transplantation (patient 7). Those 4 patients with massive hemorrhage underwent emergency angiography so that coil embolization of bleeding arteries could be performed. Angiography of all 4 patients with massive hemorrhage (patients 4, 5, 6, and 7) revealed pseudoaneurysm or contrast leakage at the bleeding artery. The celiac in 1, and the common to proper hepatic artery in the remaining 3 patients were completely embolized with coils. These 4 patients developed hepatic infarction immediately after coil embolization.

Three of the remaining 4 patients (1, 2, 3, and 8) had hepatocellular carcinoma (HCC) (patients 1, 3, and 8) (Table 1). Patient 1 presented with shock due to massive hemorrhage from the punctured femoral artery 12 h after completion of transcatheter arterial embolization of HCCs. In this patient, the left and middle hepatic arteries were embolized with gelatin sponge particles, and the infarct occurred in the left hepatic lobe. Patient 3 underwent coil

Table 1 Summary of patient data

Patient No.	Age	Sex	Disease	Surgical Procedure	Angiographic Findings	Interventional Procedure	CT Findings in the Liver (long axis × short axis)
1	70	F	Multiple hepatocellular carcinomas		Multiple hepatocellular carcinomas in the bilateral lobes	Chemoembolization of right and left hepatic arteries with gelatin sponge particles	Wedge-shaped infarct in a lateral segment of the left lobe (8.5 cm × 7 cm)
2	65	M	Multiple liver metastases of colon carcinoma		Narrowing of RHA and portal venous branches	Indwelling catheters in RHA and the PV	Crescent-shaped infarct in the right lobe (11 cm × 5 cm)
3	59	M	Multiple hepatocellular carcinomas		Tumor thrombus in LPV, multiple hepatocellular carcinomas	Embolization of replaced RHA with coils	Wedge-shaped infarct in the right lobe (12.5 cm × 7 cm)
4**	42	M	Gastric carcinoma	Partial gastrectomy, Bilroth-I anastomosis	Pseudoaneurysm at trunk of CA, arterioportal shunt	Embolization of CA with coils	Wedge-shaped infarct in a lateral segment of the left lobe (6 cm × 2.5 cm), crescent-shaped infarct in segment 6 (5 cm × 1 cm), splenic infarction
5**	76	M	Bile duct carcinoma	Partial hepatectomy, Bile duct resection, Roux-en-Y anastomosis	Contrast leakage at site of anastomosis of CHA, arterioportal shunt	Embolization of CHA to PHA and RHA with coils	Crescent-shaped infarct in the right lobe (8 cm × 3.5 cm)
6**	63	F	Bile duct carcinoma	Pancreaticoduodenectomy, Child's reconstruction	Pseudoaneurysm at origin of GDA, arterioportal shunt	Embolization of CHA to PHA with coils	Wedge-shaped infarct in a posterior segment of the right lobe (2 cm × 2 cm) and segment 3 (5 cm × 3 cm)
7**	39	M	Liver cirrhosis	Liver transplantation	Pseudoaneurysm at origin of GDA	Embolization of CHA to PHA with coils	Wedge-shaped infarct in segment 5 (8 cm × 5 cm)
7* **					Contrast extravasation from previously embolized site	Embolization of CHA with coils	Wedge-shaped infarct in segment 8 (9 cm × 4.5 cm)
8	65	M	Multiple hepatocellular carcinomas		Multiple hepatocellular carcinomas in the right lobe	Chemoembolization of RHA with gelatin sponge particles, PTPE (RPV was embolized 2 weeks after chemoembolization)	Crescent-shaped infarct containing gas in the right lobe (12.5 cm × 4 cm)

CA, celiac artery; CHA, common hepatic artery; LPV, left portal vein; NA, data not available; PTPE, percutaneous transhepatic portal vein embolization; PV, portal vein; RHA, right hepatic artery; RPV, right portal vein; *, a second latter infarction in same patient; **, emergency arteriography.

embolization of the replaced right hepatic artery from the superior mesenteric artery. This was performed out of need for conversion of dual hepatic arteries into a single vascular supply before the start of hepatic arterial infusion therapy. The patient showed occlusion of the left portal vein and the right superior anterior branch due to the tumor thrombus of HCC, while his infarct was observed in a large area of the right hepatic lobe where the replaced right hepatic artery was occluded. Patient 8, who had HCCs in the right hepatic lobe, underwent transcatheter embolization of the right hepatic artery 2 weeks before embolization of the right portal vein (Fig. 2). The purpose of this procedure was to increase the size of the uninvolved left hepatic lobe before right hepatic lobectomy [17]. Hepatic infarction was observed in the right hepatic lobe immediately after portal vein embolization.

The last patient, who had multiple hepatic metastases of colon carcinoma, had been administered an anticancer agent through indwelling catheters in the hepatic artery and the portal vein for 12 months (patient 2) (Table 1). A crescent-shaped infarction in the right hepatic lobe was detected after this infusion therapy. Blood-flow scintigraphy with 99m Technetium macroaggregated albumin through the respective catheters in both the hepatic artery and portal veins showed perfusion defects corresponding to the infarction area.

Incidence of hepatic infarction. The incidence of hepatic infarction was 0.5% (9/1982) for all angiography and interventional procedures, and 0.6% (8/1309) for all patients. Among all 1982 hepatic angiogra-

phy and interventional procedures in this period, 673 procedures were accompanied by arterial embolization, which included 12 emergency procedures. Therefore, the incidence of hepatic infarction in hepatic arterial embolization was 1.0% (7/673), and in the case of emergency hepatic arterial embolization, the incidence of hepatic infarction was 33.3% (4/12). In contrast, the incidence in elected hepatic arterial embolization was only 0.5% (3/661).

Angiography. In all 8 procedures in the 7 patients who underwent embolization of the hepatic or celiac artery, complete occlusion of the embolized artery was visualized by angiography immediately after embolization (Figs. 1, 2). In patient 2, who underwent infusion chemotherapy, narrowing and partial occlusion of the right hepatic artery was confirmed on angiography through the indwelling catheter. Patient 4, who showed a pseudoaneurysm at the celiac trunk and underwent a coil embolization of the celiac trunk at the initial angiography, had a second angiography performed 7 days after the first to evaluate the patency of the occluded artery. Occlusion of the celiac and left hepatic artery was depicted on the second angiography. The left hepatic artery was probably occluded by thrombus, and infarct in the left hepatic lobe was confirmed on CT. A small infarct in the right inferior posterior segment was also seen on CT. In short, occlusion of the hepatic artery in 7 patients, and narrowing in 1 were confirmed on angiography.

Occlusion of the portal vein was demonstrated in 3 patients (patients 2, 3, and 8) (Fig. 2). Portal vein

Table 2 Changes in liver function with each onset

Patient No.	Total Bilirubin (0.3–1.3 mg/dl)**			Aspartate Aminotransferase (11–32 IU/l)**			Alanine Aminotransferase (6–39 IU/l)**			Albumin (3.9–4.9 g/dl)**	PT% (75–120%)**	Outcome (days after onset)	Cause of death
	Day***	Day***	Day***	Day***	Day***	Day***	Day***	Day***	Day***	Day***	Day***	Day***	Day***
	–1	+1 or 2	14	–1	+1 or 2	14	–1	+1 or 2	14	–1	–1		
1	0.7	0.8	7.3	74	5190	41	58	2780	14	4.0	79	Death (40)	Liver failure
2	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA		
3	0.8	3.2	12.8	64	3600	67	23	1118	29	2.5	68	Death (20)	Liver failure
4	0.9	1.5	0.3	20	653	76	9	489	37	3.8	80		
5	2.9	4.6	17.9	24	2130	25	31	2710	37	3.1	75	Death (54)	Liver failure
6	2.1	5.9	1.1	33	805	27	33	423	48	3.0	87		
7	1.3	8.4	23.6	66	668	80	168	1051	83	4.3	65		
7*	6.7	7	13.9	54	815	27	34	461	32	3.6	62	Death (64)	Liver failure
8	0.8	1.5	0.6	37	1672	47	93	1402	68	3.7	93		

NA, data not available; *, second infarction occurred 2 months after first onset; **, values in parentheses are normal limits; ***, days before (–) or after (+) onset of hepatic infarction.

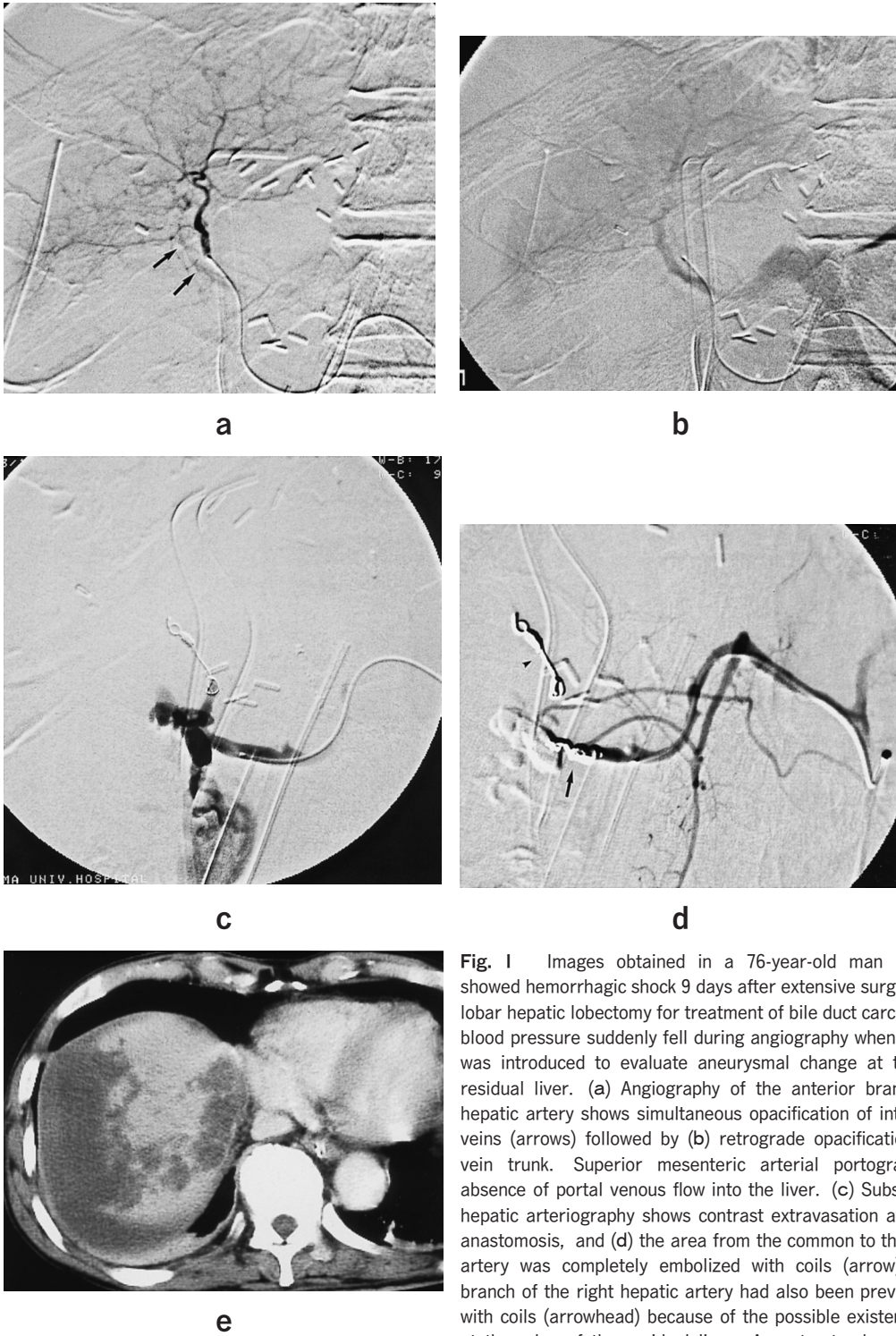


Fig. 1 Images obtained in a 76-year-old man (patient 5) who showed hemorrhagic shock 9 days after extensive surgery including left lobar hepatic lobectomy for treatment of bile duct carcinoma. Patient's blood pressure suddenly fell during angiography when a microcatheter was introduced to evaluate aneurysmal change at the edge of the residual liver. (a) Angiography of the anterior branch of the right hepatic artery shows simultaneous opacification of intrahepatic portal veins (arrows) followed by (b) retrograde opacification of the portal vein trunk. Superior mesenteric arterial portography shows an absence of portal venous flow into the liver. (c) Subsequent common hepatic arteriography shows contrast extravasation at site of arterial anastomosis, and (d) the area from the common to the proper hepatic artery was completely embolized with coils (arrow). The anterior branch of the right hepatic artery had also been previously embolized with coils (arrowhead) because of the possible existence of aneurysm at the edge of the residual liver. A contrast-enhanced (e) CT scan performed 13 days after embolization shows large peripheral low attenuation.

occlusion of patients 2 and 8 corresponded to the infarction areas. Patients 4, 5, and 6, who experienced massive hemorrhage after surgery, showed not portal vein occlusion but rather arteriportal shunt at the emergency angiography (Fig. 1). The superior mesenteric arteriography showed poor hepatopetal portal venous opacification. All of patients 4, 5, and 6 presented with hemorrhagic shock (systolic pressure less than 100 mmHg and a pulse rate of more than 100 bpm) at that time. The second angiography in patient 4, performed when under stable conditions, showed no arteriportal shunt.

CT scans. Both unenhanced and enhanced CT were performed in all patients when hepatic infarction was confirmed on CT. The initial CT scans were performed within 1–14 days (mean; 8.4 days) of the onset of infarction. Follow-up CT scans were obtained in all patients from 5–86 days (mean; 23 days) after hepatic infarction.

Wedge-shaped infarcts were observed in 6 onsets in 5 patients, and crescent-shaped infarcts in 4 onsets in 3 patients (Figs. 1, 2). Patient 4 showed one wedge-shaped lesion and one crescent-shaped lesion in separate areas of the liver. Lobar distribution of the hepatic infarct was seen in 4 onsets, and segmental or subsegmental

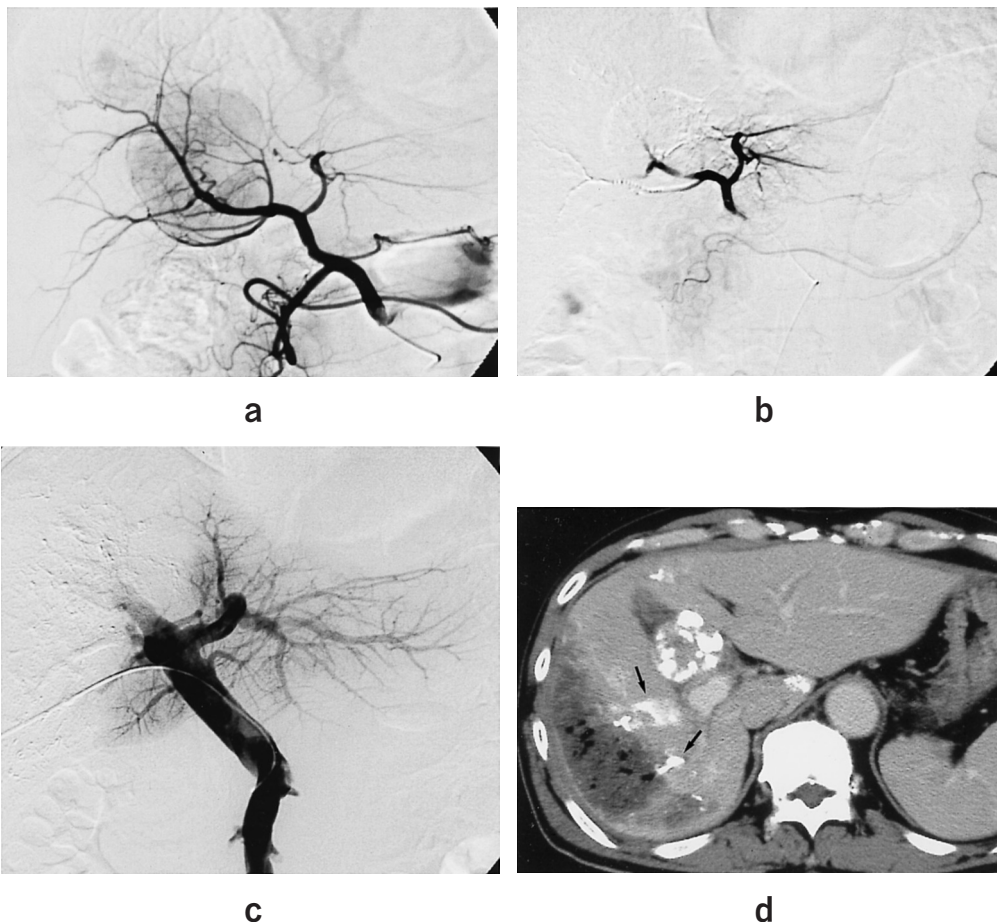


Fig. 2 Images obtained in a 65-year-old man (patient 8) who underwent transcatheter embolization of the right and middle hepatic arteries 2 weeks before embolization of the right portal vein. (a) Common hepatic arteriography shows HCCs in the right hepatic lobe and the medial segment of the left hepatic lobe. (b) Right and middle hepatic arteries were embolized with gelatin sponge particles immediately after arterial infusion of a mixture of lipiodol and an anticancer agent to HCC, and both tumor blushes disappeared. (c) Two weeks later, the right portal vein was percutaneously embolized with a mixture of lipiodol and gelatin sponge particles to increase the size of an uninvolved lateral segment of the left hepatic lobe. (d) A contrast-enhanced CT scan performed 5 days after portal vein embolization shows a crescent-shaped infarction containing gas in the right hepatic lobe. Occlusion of the right portal vein is shown by lipiodol retention (arrows).

distribution in 6. Three crescent-shaped infarcts appeared lobar in distribution. Two hepatic infarcts (in patients 5 and 8) contained gas (Fig. 2). An associated splenic infarct was identified in patient 4. Portal vein occlusion was confirmed by enhanced CT in patients 3 and 8 (Fig. 2).

Disease progress. The 8 patients for whom laboratory data were available (all except patient 2) showed a marked increase in the levels of total bilirubin (1.5–8.4 mg/dl, mean; 5.0 mg/dl), aspartate aminotransferase (668–5,190 IU/l, mean; 2,038 IU/l), and alanine aminotransferase (423–2,780 IU/l, mean; 1,160 IU/l) 1 or 2 days after the onset of infarction (Table 2). The levels of aspartate aminotransferase and alanine aminotransferase recovered in all 8 patients. However, 4 patients (patients 1, 3, 5, and 7) showed continuous high total bilirubin levels at 14 days after infarction onset (7.3–23.6 mg/dl, mean; 12.9 mg/dl) and subsequently died. In these nonrecovering patients, lobar distribution of the infarct was seen in patients 3 and 5, and segmental or subsegmental distribution was seen in patients 1 and 7. The liver function of patients 1 and 3 had already been compromised by liver cirrhosis, and the levels of total bilirubin in patients 5 and 7 were already high the day preceding onset.

Risk Factors. Occlusion or stenosis of the hepatic artery, which involves a decrease in hepatic arterial flow, was observed in 8 patients. Analysis of portal venous flow revealed portal vein obstruction upon infarction in 2 patients (patients 2 and 8). However, a decrease in portal venous flow was suspected in 5 patients (patients 1, 4, 5, 6, and 7) who presented with hemorrhagic shock. Three of the patients (patients 4, 5 and 6)

showed an arteriportal shunt at emergency angiography. We defined their blood-flow condition as a possible portal insufficiency, as superior mesenteric arteriography showed poor hepatopetal portal venous opacification. Only 1 patient was supposed to have no portal venous insufficiency (patient 3), with neither portal vein obstruction nor shock being observed. Shock was observed in 5 patients, and this was also the most frequently observed phenomenon after hepatic arterial occlusion (Table 3). Liver transplantation in 1 patient, extensive surgical procedures in 3, liver cirrhosis in 3, and biliary disease in 2 were noted, while no patient had cardiac disease, or had undergone general anesthesia immediately prior to onset.

Discussion

Interventional procedures such as arterial embolization are one of the causes of hepatic infarction [4, 8]. Many patients, however, never develop hepatic infarction after hepatic arterial embolization or ligation, probably because of the dual blood supply to the liver [4, 18–20]. During periods of hepatic ischemia, hepatocytes are able to survive by increasing the extraction of oxygen from portal venous blood [21, 22]. Thus, patients with hepatic infarction must have some other unusual hemodynamic changes to induce the onset of hepatic infarction. The present findings indicate that the incidence of hepatic infarction following elected hepatic arterial embolization was only 0.5% in this series. However, it was as high as 33.3% following emergency procedures.

In the 1970s–80s, sporadic cases of hepatic infarction following emergency arterial embolization for bleeding

Table 3 Risk factors in each patient

Patient No.	Arterial Insufficiency	Portal Venous Insufficiency	Shock	Abdominal Surgery	Liver Cirrhosis	Biliary Disease
1	Yes	Possible	Yes	No	Yes	No
2	Yes	Yes	No	No	No	No
3	Yes	No	No	No	Yes	No
4	Yes	Possible	Yes	Yes	No	No
5	Yes	Possible	Yes	Yes	No	Yes
6	Yes	Possible	Yes	Yes	No	Yes
7	Yes	Possible	Yes	Yes	No	No
7*	Yes	Possible	Yes	Yes	No	No
8	Yes	Yes	No	No	Yes	No

*The second infarction.

from the gastroduodenal or hepatic artery were reported [5-7]. Then in 1983, Miller *et al.* summarized the findings of 6 patients with hepatic infarction accompanying concurrent or recent hemorrhagic shock [4]. They surmised that hepatic infarction is likely to be secondary to decreased portal perfusion from recent or ongoing hemorrhagic shock coupled with transcatheter occlusion of several hepatic arteries by Gelfoam. We agree with this proposition.

In our review of the last decade, 5 of all 9 episodes (8 patients) were also observed immediately after emergency embolization for postoperative bleeding. Our angiography results revealed occlusion of the hepatic artery in all 8 patients. Furthermore, we suspected that 3 (patients 4, 5, and 6) suffered portal venous insufficiency. These 3 showed an arteriportal shunt on emergency hepatic arteriography in hemorrhagic shock, which has been, to our knowledge, rarely reported [23, 24]. When the arteriportal shunt appeared, superior mesenteric arterial portography showed poor portal venous inflow to the liver.

Miller *et al.* have suggested that embolization of the bleeding site not by Gelfoam but by coil or balloon can spare areas of normal liver from Gelfoam and prevent the onset of hepatic infarction [4]. However, our results indicate that coil embolization can cause hepatic infarction. The problem is not likely to be the type of embolizing material, but the hemodynamic status of the patient. In addition, hemorrhagic shock itself is a risk factor for hepatic infarction.

In livers experiencing shock, fibrin thrombi are frequently found, primarily in the sinusoid and portal veins of the infarcted area, suggesting the existence of disseminated intravascular coagulation (DIC) and/or microcirculatory disturbance in the liver [25, 26]. The second arteriography of patient 4 in the present study showed occlusion not only of the embolized celiac artery, but also the nonembolized left hepatic artery. The left hepatic artery might have been occluded by a thrombus that probably formed during shock. Patient 1 manifested shock due to massive hemorrhage from the punctured femoral artery 12 h after completion of transcatheter arterial embolization. In that case, in addition to arterial occlusion, shock itself and subsequent possible portal venous insufficiency may have caused the hepatic infarction.

Hepatic infarction following elected interventional procedures appears to be quite rare, accounting for only

0.5% of all elected procedures in the present study. According to Sakamoto *et al.*, only 4 cases of hepatic infarction were observed in a total of 2,300 procedures of transcatheter arterial embolization of malignant tumors [27]. Those 4 cases had a thrombus in the branch of the portal vein. Except for such cases, the background of hepatic infarction is usually very specific. For example, Sawhney *et al.* have recently reported a case of hepatic infarction complicated with a transjugular intrahepatic portosystemic shunt [28]. In patient 3 of our series, contrary to our expectation, intrahepatic collateral pathways from the left hepatic artery did not emerge after embolization of the replaced right hepatic artery, probably due to the presence of a large tumor in the liver. To our knowledge, this is the first reported case of hepatic infarction accompanied by the conversion of dual hepatic arteries into a single vascular supply. In patient 8, an hepatic artery embolized with gelatin sponge particles could not be recanalized 2 weeks after the time of portal vein embolization. Studies have revealed that the resorption time for Gelfoam is typically within 7-21 days of embolization [29, 30]. In patient 2, repeated chemofusion damaged the intrahepatic vascular structure, possibly leading to the hepatic infarction. In the same period, we had no other experience of the same interventional procedures as in cases 2, 3, and 8. These examples are very specific and suggest ways of avoiding the onset of hepatic infarction following certain elected interventional procedures.

Serum alanine aminotransferase levels have been shown to be a sensitive marker for hepatic dearterization injuries [10]. Our laboratory data indicate that the levels of not only alanine aminotransferase but also aspartate aminotransferase increase markedly immediately after the onset of infarction. Although these levels are usually recovered within 2 weeks, 4 patients showed continuous high total bilirubin levels at 14 days after the onset of infarction and subsequently died. Their basic liver functions had already been compromised by liver cirrhosis or biliary obstruction, whereas the size or distribution of infarct in these patients was not obviously different from that of the surviving patients. Thus, the prognosis of liver infarction may not depend wholly on lesion size, but also on the original liver function of the patient.

Our patients showed several risk factors that have been previously reported [11-15]. Apart from arterial insufficiency, shock and portal venous insufficiency were frequently observed, as described above. Liver transplan-

tation and extensive dissection, in which several potential collateral pathways may have been disrupted [31, 32], were also identified in our series. Especially in liver transplantation, hepatic infarction is known as a common complication, even if unexpected hepatic arterial embolization is not performed [33]. The likelihood of hepatic infarction is due to the complications related to vascular insufficiency being relatively easy to identify [34, 35]. Hepatic artery thrombosis is the most common vascular complication, occurring in 3–12% of the transplants in adults and up to 42% of the transplants in pediatric patients [36]. Careful postoperative imaging observation for hepatic infarction is therefore necessary. Although cardiovascular diseases [11] were not noted in the present study, biliary disease was observed in 2 patients. All patients showed other risk factors in addition to arterial insufficiency.

One limitation of our study is that our series was not large enough to determine the certain location with greater risk of hepatic infarction. The population included only one institute. However, our series includes many kinds of interventional procedures in consecutive patients. Therefore, the problems cannot be population-specific. The other limitation of our study is that CT scans were not always performed immediately before and/or after interventional procedure. We have possibly missed onsets of minor hepatic infarction that did not require CT scanning. However, this problem was not crucial problem to this clinical research.

In conclusion, the incidence of hepatic infarction following interventional procedures is low but appears to occur most frequently in emergency arterial embolization in hemorrhagic shock. Patients have multiple risk factors, and careful consideration should be paid to patients whose liver function is already compromised.

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