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Liver Parenchymal Changes After Transcatheter Arterial Embolization Therapy for Hepatoma: CT Evaluation

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Abstract. We retrospectively reviewed computed tomographic (CT) findings of 118 patients with hepatoma who received sequential follow-up CT after transcatheter arterial embolization (TAE). Thirty-five patients received TAE using Gelfoam particles with cisplatin, 37 patients using Gelfoam particles and iodized oil (Lipiodol) with cisplatin, and 46 patients using iodized oil with cisplatin. Liver atrophy was observed in 33 patients, lobarly or focally, depending on the embolized area. It was frequently associated with portal vein occlusion by the tumor, usage of iodized oil, and repeated embolization therapy. The lobar atrophy was seen in patients who had portal vein occlusion and/or received repeated embolization therapy. The focal atrophy was observed in patients who were administered iodized oil. Infarction developed in four patients who had a thrombus in the portal vein and received peripheral embolization therapy using iodized oil. We conclude that liver parenchymal changes occur frequently in patients who have portal vein occlusion and/or receive peripheral embolization using iodized oil.

Key words: Liver, neoplasm—Liver, CT—Liver, atrophy—Arterial blockade.

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Transcatheter arterial embolization therapy (TAE) of the hepatic artery is considered to be a safe technique if the portal vein is patent and liver function

Table 1. Frequency of liver parenchymal changes after TAE in relation to various factors

	Method of TAE					
	GS/CDDP/Lip−[a]	GS/CDDP/Lip+[b]	CDDP/Lip+[c]	Portal vein occlusion	Liver cirrhosis	No. of treatments (mean ± SD)
Atrophy						
Lobar (N = 15)	5/20	7/16	3/34	9[d]	14	2.8 ± 1.5
Segmental (N = 18)	0/15	12/21[d]	6/12	4	14	1.4 ± 0.7
Necrosis (N = 4)	0	2	1	3	1	1.0
None	29	14	36	9	71	1.3 ± 0.6

TAE, transcatheter arterial embolization therapy; GS, gelatin sponge particle; CDDP, cisplatin; Lip−, without Lipiodol (iodized oil); Lip+, with Lipiodol; SD, standard deviation.

[a] Lobar TAE, N = 20; segmental TAE, N = 15.

[b] Lobar TAE, N = 16; segmental TAE, N = 21.

[c] Lobar TAE, N = 34; segmental TAE, N = 12.

[d] The frequencies of the development of the liver parenchymal changes were statistically significant in chi-square analysis.

is not severely damaged. Recently, administration of iodized oil with Gelfoam particles has become popular in order to achieve peripheral embolization for the treatment of hepatoma [1, 2]. With this technique, liver parenchymal changes may occur more severely. However, changes of the liver parenchyma after TAE have not been well-documented. In this study, we reviewed computed tomography (CT) with special attention to liver parenchymal changes following TAE.

Materials and Methods

We retrospectively reviewed CT findings of 118 patients with hepatoma who received sequential follow-up CT after TAE. There were 91 men and 27 women with an age range of 41–80 years (average 62.3 years). Associated cirrhosis was observed in 107 patients (91.0%). TAE was performed according to a protocol reported previously [3]. Thirty-five patients received TAE using Gelfoam particles and 50–100 mg of cisplatin (20 patients were embolized lobarly and 15 patients segmentally), 37 patients with 3–6 ml of iodized oil (Lipiodol) with 50–100 mg of cisplatin followed by a small amount of Gelfoam particles (16 patients were embolized lobarly and 21 patients segmentally), and 46 patients only with 3–6 ml of iodized oil with 100 mg of cisplatin (34 patients were embolized lobarly and 12 patients segmentally).

CT was performed before, 1 week, 1 month, and every 2–3 months after TAE. Images were obtained using a CT 9800 HR (General Electric, Milwaukee, WI, USA) or Delta 2020 (Technicare, Cleveland, OH, USA). Precontrast, bolus dynamically enhanced, and postcontrast images were obtained for all images. CT was reviewed with regard to alteration of the segmental volume of the liver in the tumor-bearing area and nontumor-bearing area, alteration of the attenuation of the liver parenchyma, and time of appearance of these findings. The volume of the liver was assessed by measuring a previously reported method [4]. Atrophy was defined as decrease in volume of more than 20% of the embolized liver parenchyma after taking into consideration the decrease in the size of the tumor.

Results

Liver atrophy was observed in 33 patients (28.0%), seen lobarly (N = 15) or focally (N = 18), depending on the embolized area. It was associated with portal vein occlusion by the tumor, usage of iodized oil, and number of treatments. In all atrophy-developed patients, the main tumor responded to treatment. Cirrhosis did not significantly contribute to the progress of atrophy.

Lobar atrophy was usually seen in patients who had occlusion of a branch of the portal vein and/or received repeated embolization therapy, regardless of embolic materials (Fig. 1). Compensatory hypertrophy was observed in the untreated lobe. In patients with a portal vein occlusion, lobar atrophy developed rapidly after TAE, usually observed within a month. Focal atrophy was frequently observed in patients who were administered iodized oil

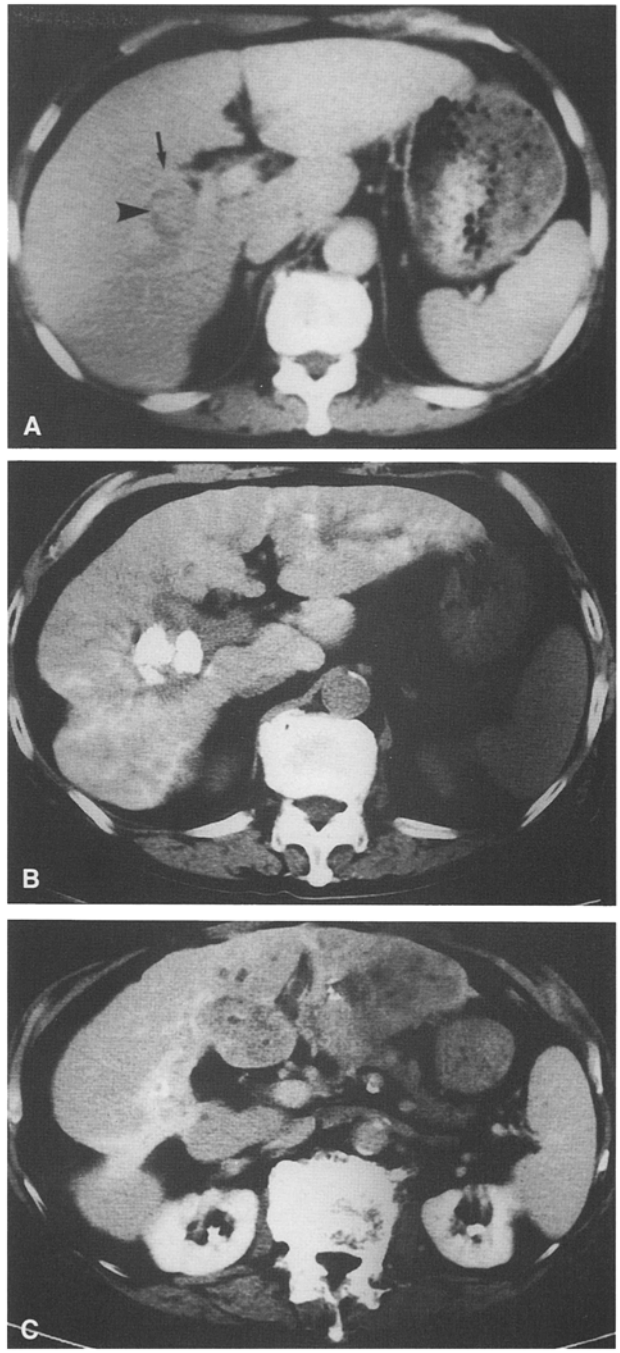

Fig. 1. A 61-year-old man with nodular hepatoma treated by iodized oil/cisplatin with Gelfoam particles. **A** Contrast-enhanced CT shows a nodular mass in the right hilum (*arrowhead*). The anterior branch of the portal vein was occluded (*arrow*). The right hepatic artery was embolized using iodized oil/cisplatin with Gelfoam particles. A small amount of iodized oil was given in the left hepatic artery for detection of small metastatic tumors. **B** Precontrast CT obtained 1 month after TAE shows atrophy in the anterior segment of the right lobe. A small amount of iodized oil is seen in the left lobe as well as the right lobe. The patient received TAE twice after this CT. **C** Contrast-enhanced CT obtained 26 months after initial TAE shows marked atrophy in the anterior segment of the right lobe. The posterior segment is also atrophic. A new lesion is seen in the left lobe.

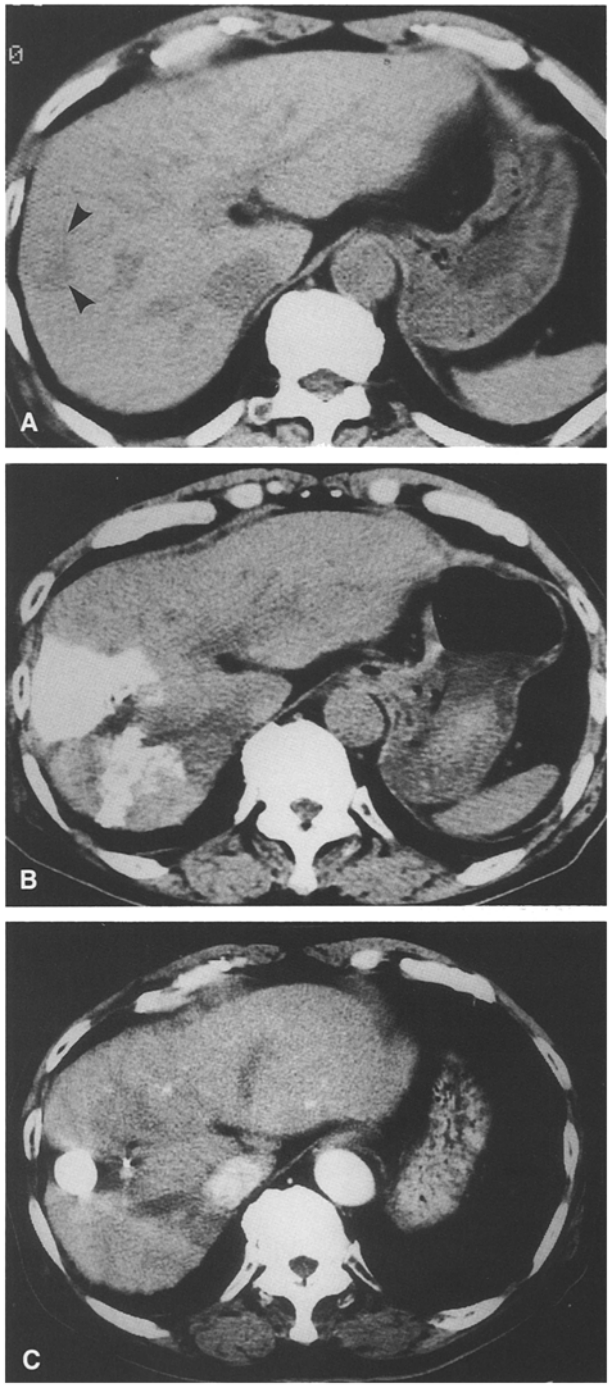

Fig. 2. A 72-year-old man with nodular hepatoma treated by embolization of the branch of a hepatic artery (S5) using iodized oil/cisplatin without Gelfoam particles. **A** Precontrast CT shows a slightly hypodense mass in the right lobe (*arrowheads*). A branch of the right hepatic artery was embolized using iodized oil/cisplatin (segmental TAE). **B** Precontrast CT obtained 1 week after TAE shows dense accumulation of iodized oil in the liver parenchyma of the tumor-bearing segment, as well as in the tumor. Iodized oil is also seen in the posterior segment (S6). **C** Contrast-enhanced CT obtained 20 weeks after TAE shows marked atrophy of the liver parenchyma surrounding the tumor, corresponding to the area of iodized oil accumulation.

(Fig. 2). In these patients, dense accumulation of iodized oil was observed in the surrounding liver on CT obtained 7–10 days after TAE (Fig. 2B). Atrophy became apparent usually at 2–3 months after TAE.

Infarctions were observed in four patients. Hypodense wedge-shaped lesions developed within 1 month after TAE (Figs. 3 and 4). These presented with severe abdominal pain and fever. All of these patients had a thrombus in the branch of the portal vein and were treated with iodized oil with cisplatin. Two of the lesions were proven histologically either with operation or autopsy. The other two lesions disappeared on follow-up CT.

Discussion

According to an extensive survey of Novak [5], the overall complication rate of TAE of the hepatic artery is 4.4%, including liver failure, necrosis of the gallbladder, splenic infarction, and hepatic infarction. Although three fourths of the total hepatic blood supply are provided by the portal vein, the hepatic artery provides 40–60% of the oxygen supply to the liver: the portal vein plays a secondary role as a source of oxygen supply [6, 7]. In cases with insufficient portal blood supply, the interruption of the arterial flow results in hypoxia of the hepatocytes, causing atrophy or necrosis depending on the degree of ischemia.

Atrophy of the liver is also seen as a complication of various hepatic and biliary diseases, including liver cirrhosis, surgical resection, hepatic vein obstruction, hydatid cyst, intrahepatic cholelithiasis, cholangiocarcinoma, and radiation therapy [4, 8–10]. The mechanism of liver atrophy in these diseases is not clear, but hepatocellular damage, vascular inflow obstruction, compression, and the combination of these are considered to be the etiology.

The immediate liver functional damage after TAE assessed by serum enzymes (GOT, GPT) is well-known, but chronic changes in the liver parenchyma have not been well-recognized. In our series, liver atrophy developed in 28.0% of patients after TAE. Atrophy can be divided into focal or lobar types, depending upon the area of the affected liver [9].

Lobar atrophy was observed regardless of the embolic materials. The changes developed rapidly after TAE. More than half the patients had obstruction of a branch of the portal vein. Repeated TAE was also a significant factor for lobar atrophy. Cirrhosis is the most common etiology of liver atrophy and 91.0% of our patients had cirrhosis as a background. However, liver cirrhosis usually does not progress so rapidly [11] and we assume it was not a significant factor for developing atrophy.

Focal atrophy was exclusively seen in patients

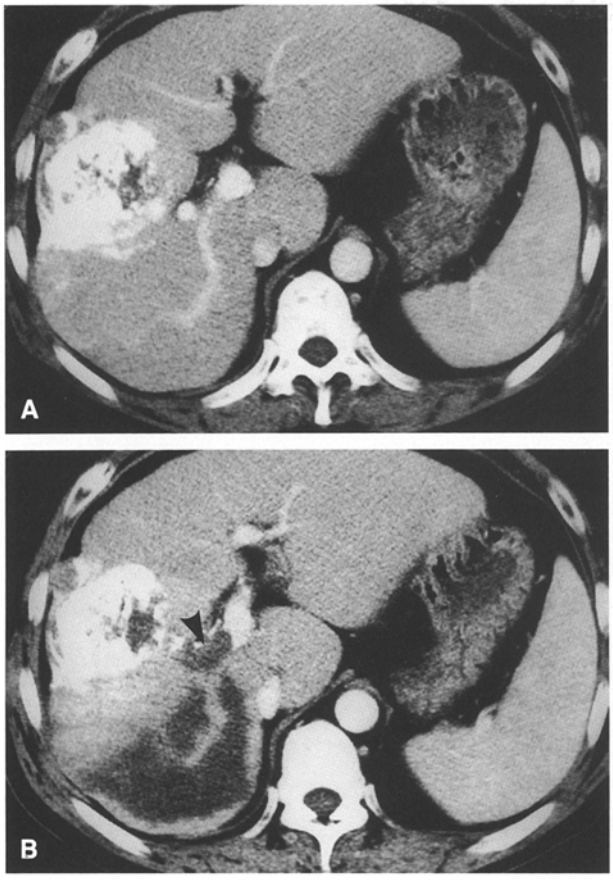

Fig. 3. A 42-year-old man with nodular hepatoma treated by iodized oil/cisplatin with Gelfoam particles. **A** Contrast-enhanced CT obtained 1 week after TAE shows accumulation of iodized oil in the tumor. The patient experienced sudden abdominal pain and fever 2 weeks after TAE, and abdominal CT was obtained. **B** Contrast-enhanced CT shows a hypodensity area in the posterior segment of the right lobe, which proved to be an infarction on operation. Note the tumor thrombi which developed in a branch of the portal vein after TAE (*arrowhead*). Peripheral branch of the portal vein is seen in the infarcted area.

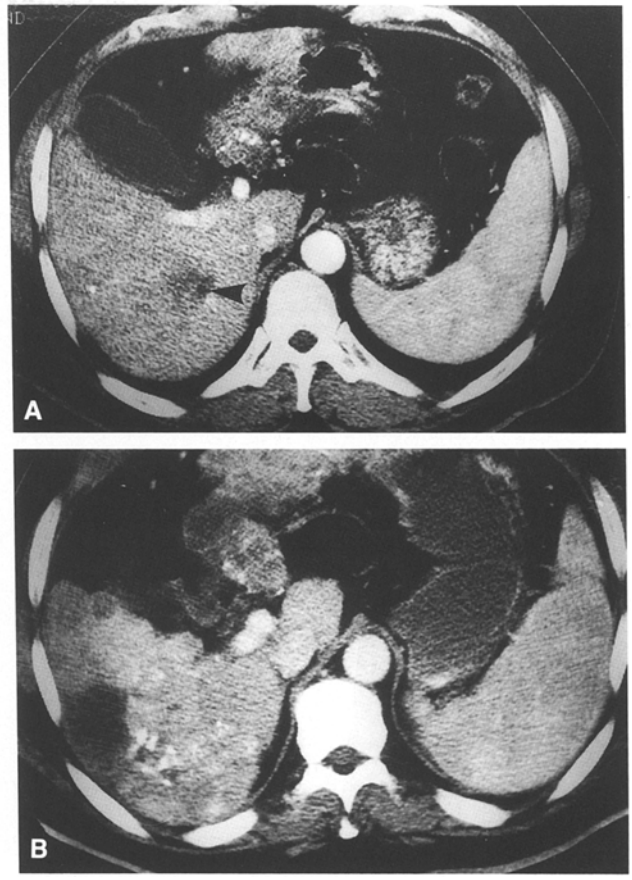

Fig. 4. A 41-year-old man with diffuse hypovascular hepatoma treated by iodized oil/cisplatin. **A** Contrast-enhanced CT before treatment shows an indiscreet mass with portal vein thrombus (*arrowhead*). The patient experienced sudden abdominal pain and fever, and abdominal CT was obtained. **B** Contrast-enhanced CT 1 week after TAE shows an irregularly shaped hypodensity area in the posterior segment of the right lobe. The patient was treated conservatively. The patient died 3 months after TAE and the liver infarction was demonstrated at autopsy.

receiving peripheral embolization using Lipiodol. The inflow of iodized oil into the portal vein was frequently observed after TAE, and atrophy developed gradually. In histologic examinations of the liver treated by TAE using iodized oil with Gelfoam particles, multiple small infarctions were reported in the nontumorous areas [2].

Liver infarction is a serious sequence rarely observed after an occlusion of the hepatic artery or portal vein, especially postoperatively [12]. Extent of the infarction depends on the site of the occlusion and the development of collateral blood supply. On the other hand, the frequency of liver infarction after hepatic artery embolization is very rare, being reported to be 0.61% [5]. In our series, most of the patients with portal vein obstruction showed only liver atrophy. In patients who developed infarction, they had a portal vein obstruction and received peripheral embolization using iodized oil. Probably, the obstruction of the portal vein and hepatic artery was so complete that severe hypoxia of the hepatocytes occurred.

In conclusion, liver parenchymal changes after TAE occur frequently as had been postulated. As a result, atrophy with compensatory hypertrophy is seen, but most of the patients are asymptomatic. However, in patients with portal vein obstruction or poor liver function, serious complications may occur. In the evaluation of treatment effect of TAE for hepatoma, there is need to consider not only the treated tumor, but also the changes in configuration of the surrounding liver parenchyma.

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