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Transient hepatic attenuation difference (THAD) following transcatheter arterial chemoembolization for hepatic malignancy: changes on serial CT examinations

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Abstract The purpose of this study was to investigate the natural history of transcatheter arterial chemoembolization (TACE)-induced, transient hepatic attenuation difference (THAD). Among the patients who underwent TACE for treatment of hepatocellular carcinomas during a 32-month period, 32 patients with 40 newly developed THADs defined as localized, transient, peripheral hepatic parenchymal enhancement during the hepatic arterial phase of dynamic CT (axial dimension, 1.9–8.8 cm; mean, 4.9 cm) in the vicinity of iodized-oil accumulation were subjected to a retrospective analysis of serial follow-up CT examinations. Among the 40 TACE-induced THADs, 18 (45%) and 9 (23%) were accompanied with arterial-portal venous fistula (APF) and portal venous stenosis (PVS), respectively, while 2 (5%) THADs showed both APF and PVS simultaneously. Thirty-eight (95%) THADs disappeared spontaneously during the fol-

low-up period (range, 4–26 months; mean, 9 months), and 11 (29%) of them showed progressive atrophy of the corresponding hepatic parenchyma. Regardless of the presence of APF, 10 (91%) of 11 THADs of following parenchymal atrophy had shown PVSs ($P < 0.001$). During the long-term follow-up period after TACE, almost all of the TACE-induced THADs spontaneously disappeared and either had or did not have corresponding parenchymal atrophy, which is strongly related to the presence of PVS. Understanding the nature of these CT features would be helpful for radiologists in determining the prognosis of the TACE-induced hepatic injuries as well as in distinguishing them from the recurrent tumour.

Keywords Hepatocellular carcinoma · CT · Chemotherapeutic embolization

Introduction

The liver has a dual blood supply with compensatory relationships between the two inflows, the hepatic artery and portal vein. The transient hepatic attenuation difference (THAD) is defined as an area of parenchymal enhancement visible during the hepatic arterial phase of dynamic CT examination [1]. THADs are related to a variety of conditions that induce an increase in arterial inflow from direct arterial-portal venous fistulas or portal/hepatic venous obliteration caused by venous thrombosis, sinusoidal or venous compression by mass lesions, hepatic abscess, direct trauma, long-standing biliary obstructions and other conditions [2–4].

Transcatheter arterial chemoembolization (TACE) has been widely used as an effective and palliative treatment of choice for hypervascular hepatocellular carcinoma [5]. Among the various complications of TACE for the non-tumorous hepatic parenchyma [6, 7], THADs appear not infrequently on the follow-up dynamic imaging studies. Aside from the diagnostic challenge for a differential diagnosis between the viable tumour tissue and THAD

Transcatheter arterial chemoembolization (TACE) has been widely used as an effective and palliative treatment of choice for hypervascular hepatocellular carcinoma [5]. Among the various complications of TACE for the non-tumorous hepatic parenchyma [6, 7], THADs appear not infrequently on the follow-up dynamic imaging studies. Aside from the diagnostic challenge for a differential diagnosis between the viable tumour tissue and THAD

itself, according to our experience over the past decades, we realized that the TACE-related THAD could be related to the subsequent non-tumorous parenchymal changes depending on the presence of biliary or vascular injuries. The purpose of this study was to investigate the fate and clinical implications of TACE-related THADs through a retrospective analysis of the serial dynamic CT examinations following TACE for hepatic malignancy in terms of portal tract injuries.

Materials and methods

Patients, TACE and CT

This retrospective study was approved by our Committee for Clinical Investigations and conducted according to the institutional review board rules for departmental review of records for research, and informed consent from subjects was waived. During a 3-year period (from May 2003 to December 2005), more than 300 patients underwent one or more TACE sessions for treatment of hepatocellular carcinomas at our institution. We employed our routine protocol for TACE, which includes the administration of an emulsion that consists of 1–20 ml of iodized-oil (Lipiodol; AndreGuerbet, Anulnay-sous-Bois, France) and 10–50 mg of doxorubicin hydrochloride (Adriamycin; Kyowa Hakko Kogyo, Tokyo, Japan), with the dose dependent upon the size, extent and vascularity of the tumour. If possible, the emulsion was injected exclusively into the segmental or subsegmental arterial branches feeding the tumour, and then gelatin sponge fragments (Gelfoam; Upjohn, Kalamazoo, MI) were administered. The first follow-up CT was performed 3 or 4 weeks after TACE, and in those patients without marginal tumour recurrence or newly developed tumours, further follow-up CT studies were performed 3 and 6 months after TACE.

For follow-up imaging, multiphase contrast-enhanced dynamic CT (unenhanced, arterial, portal and delay phases) was performed with a helical CT scanner (HiSpeed Advantage; General Electric Medical Systems, Milwaukee, WI) or a multichannel unit (Somatom Sensation 16; Siemens, Erlangen, Germany). After the intravenous administration of 150 ml of iodinated contrast agent (Ultravist 300; Schering AG, Berlin, Germany) by an automatic injector (EnVisionCT; Medrad, Pittsburgh, PA) at 3 ml/s, arterial, portal and delay phase imaging were started after a delay of 25–30 s, 65–70 s and 150 s, respectively, on the helical CT system. In the multichannel unit, a 15-s delay from the time of 100 Hounsfield units of aortic enhancement was set as the starting time of arterial phase imaging using the SmartPrep technique [8] followed by portal phase imaging conducted at 30 s from the start time of arterial phase imaging. Three-minute delayed equilibrium phase imaging was added for triple-phase imaging. The electronic data from the

CT examinations were sent to the picture archiving and communication system (PACS) for interpretation on the PACS workstations.

Among the 350 patients treated by TACE, 237 patients had serial CT examinations during a 2-year or longer follow-up period (total 927 follow-up CT examinations) with detailed reports of the TACE procedure available to retrospectively search computerized records. Forty patients with underlying liver cirrhosis had 60 transient, peripheral, wedge-shaped or irregular hepatic parenchymal enhancements visible during only the hepatic arterial phase and returned to normal or nearly normal attenuation on the portal venous phase in the vicinity of iodized-oil accumulation on follow-up CT examination. Digitally stored images of the follow-up CT and pre-TACE CT studies of the 40 patients were preliminarily reviewed by one radiologist with 10 years' experience in the field of hepatic imaging and intervention to determine whether the CT findings were directly related to the TACE procedure. Among these 60 THADs, ten showed recurrent tumour growth at the area of THAD during the follow-up period, and the possibility of residual or recurrent tumour-related THADs could be considered. Ten other THADs were already demonstrated before the TACE procedure, which might be related to preexisting portal vein thrombosis. After exclusion of the above 20 THADs, 40 THADs in 32 patients were enrolled for further analyses. The patient group comprised 26 men and 6 women with ages ranging from 35 to 78 years (mean age, 57.9 years).

Image analysis and clinical information

At the area of THADs in conjunction with the appearance of dense nodular or sparse iodized-oil accumulations in and around the focal lesions, other imaging features suggesting TACE-induced portal tract injury were investigated as follows. An arteriportal fistula (APF) was suggested for the early appearance of peripheral portal vein branches during the hepatic arterial phase of dynamic CT. Bile duct injury was suggested for the linear, branching, cystic or amorphous non-enhancing low-attenuation fluid-like densities along the portal tract. Portal venous stenosis (PVS) was suggested for the narrowing or obliteration of portal vein branches during the portal phase of dynamic CT. Localized atrophic change of the hepatic parenchyma was suggested for the area of reduced parenchymal volume with retraction of the liver surface. The presence of the above imaging features related to each THAD was determined by two radiologists with 5 and 12 years experience in the field of hepatic imaging.

The clinical results reviewed were the degree of underlying liver cirrhosis (Child-Pugh class as a parameter of the liver profile) and liver function, time interval between TACE and development of THAD and time span of THAD appearance during the follow-up period. This

current study included 22 Child-Pugh class A patients. Ten other patients had Child-Pugh B before TACE. The collated data, concerning the TACE method, included the location of the catheter tip during infusion of embolic materials, the use of gelatin sponge fragments and the total number of TACE procedures performed before the appearance of THAD. For patients who underwent more than one TACE session, the data obtained during the last session just before the appearance of THAD for the first time were used.

Statistical analysis

The chi-square test or Fisher's exact test was used to determine the significance of association between APF or PVS and corresponding hepatic parenchymal atrophic changes at the area of THAD during the follow-up period. The chi-square and multivariate linear logistic regression test were used to evaluate the relationship between the prevalence of APF or PVS and the various predisposing factors including the Child-Pugh classification before the TACE (A or B), total number of TACE (1, 2–4, or 5 or more), the level of TACE (subsegmental or segmental/lobar) and use of gelatin sponge fragments (used or not-used). A P value of less than 0.05 was considered statistically significant.

Results

The clinical data and CT findings of the 40 TACE-induced THADs in 32 patients are summarized in Table 1. The mean size of all THADs was 4.9 cm (range, 1.9 cm to 8.8 cm) in the longest dimension. Thirty-four (85%) THADs appeared at the first follow-up CT examination up to 1 month after TACE. Six others were noted at the second follow-up CT examination after TACE. The mean time interval from TACE to the follow-up CT examination showing THAD for the first time was estimated at 1.3 months (ranging from 0.6 to 3 months).

At the serial follow-up CT examinations (range of length of last follow-up, 24–48 months; mean, 32 months) after TACE, 18 (45%) THADs were accompanied by early opacification of the peripheral portal vein branches in the area of THADs, suggesting APFs (Figs. 1 and 2). Nine (23%) THADs demonstrated PVSs accompanied by bile duct injuries, which consisted of bilomas or dilated bile ducts alongside the portal tract. There was no THAD showing bile duct injury not accompanied by PVS (Figs. 2 and 3). Two (5%) THADs showed APFs and PVSs simultaneously (Fig. 2). Fifteen THADs (38%) had no evidence of APF, intrahepatic bile duct injury or PVS on any follow-up CT examinations (Fig. 4). Thirty-eight (95%) THADs spontaneously regressed and disappeared during the follow-up period; the mean time interval between the initial appearance and disappearance of THADs on the follow-up CT examinations was 9 months (range of interval time, 2–26 months). Two other THADs decreased in size, but were still present on 24-month follow-up images.

Eleven (28%) THADs demonstrated progressive atrophy of the hepatic parenchyma corresponding to the area of THAD, and the maintenance duration of the THADs was variable from 2 to 26 months (the mean interval time; 6 months) after TACE (Figs. 2 and 3). Among the 11 parenchymal atrophies corresponding to the area of THADs, 10 THADs were accompanied by PVSs ($P < 0.001$), whereas only one THAD without gross appearance of PVS had disappeared with parenchymal atrophy. Among the 18 THADs showing APFs, only two (11%) THADs simultaneously accompanied by PVSs showed resultant parenchymal atrophy. Regardless of the combination with APFs, the presence of PVSs ($n = 10$) always resulted in corresponding parenchymal atrophy (100%) (Table 1).

For the various assumptive predisposing factors for portal tract injuries after TACE, the multivariate analysis showed that there was no statistically significant factor ($P > 0.3$ for all factors) dependant on the appearance of APFs or PVSs among the 40 TACE-induced THADs (Table 2).

Table 1 THAD after TACE and hepatic parenchymal changes during follow-up period

CT findings	Atrophy	No atrophy	Size of THADs* (cm)	Total
APF and PVS	2 (18)	0	6.3	2
Only APF	1 (9)	15 (52)	4.5	16
Only PVS	7 (64)	0	4.9	7
No APF or PVS	1 (9)	14 (48)	4.8	15
Total	11	29	4.9	40

Data in parentheses are percentages

*Size of THAD is the mean size of the transient hepatic attenuation differences in the longest dimension on CT scan
THAD=transient hepatic attenuation difference, APF=arterial-portal venous fistula, PVS=portal venous stenosis, atrophy=hepatic parenchymal atrophy corresponding to the area of THAD

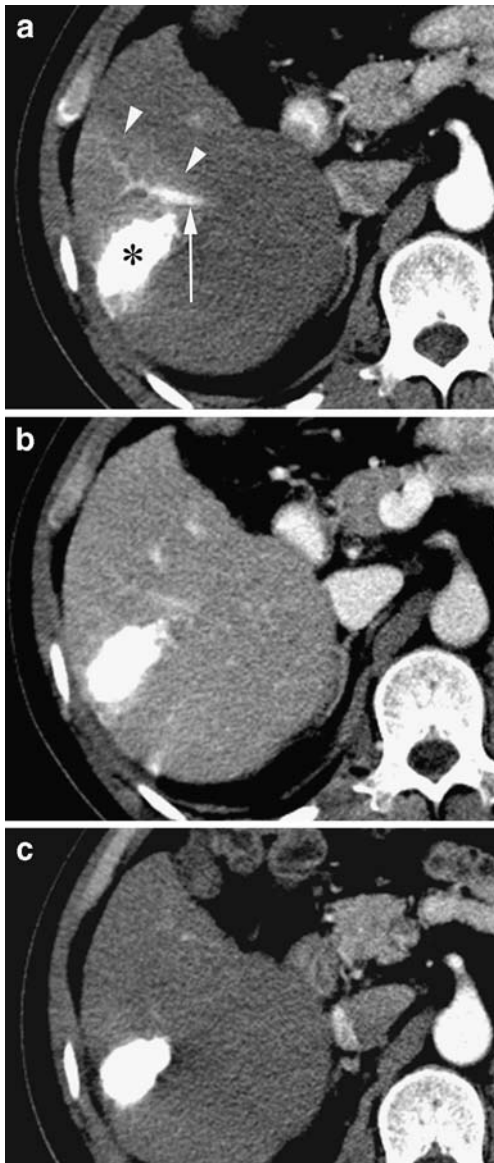


Fig. 1 A 66-year-old man who had undertaken selective TACE for a single hepatocellular carcinoma showing a THAD related to arterioportal fistula formation on follow-up CT. **a** Transverse contrast-enhanced hepatic arterial phase dynamic CT images obtained 4 weeks after TACE shows an early filling of contrast material in the peripheral portal vein branch (arrow) and surrounding subcapsular, wedge-shaped increased attenuation (arrowheads) adjacent to the area of iodized-oil accumulation (asterisk). **b** Portal phase dynamic CT shows no abnormal attenuation density at the corresponding area of arterial hypervascularity. **c** Three-month follow-up arterial phase dynamic CT shows disappearance of arterioportal fistula and THAD without gross parenchymal change

Discussion

In addition to the detection and characterization of focal hepatic lesions, dynamic CT is also an effective technique for evaluating the haemodynamic status as it relates to the unique dual blood supply of the liver. THAD reflects a

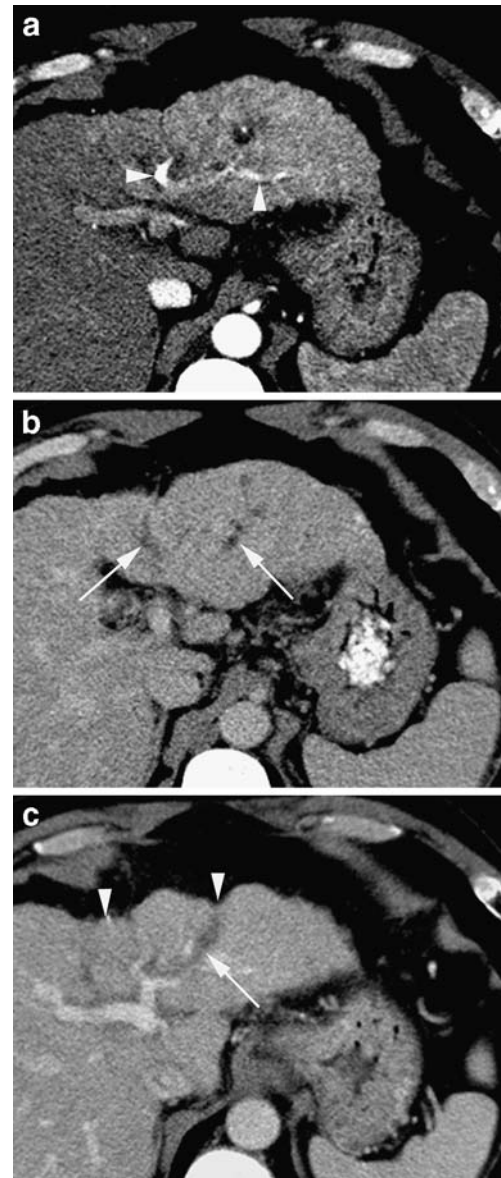


Fig. 2 A 45-year-old man underwent two separate sessions of selective TACEs for treatment of multifocal hepatocellular carcinoma showing portal tract injuries consisted of arterioportal fistula formation and portal venous narrowing in the left lobe of the liver. **a** Ill-defined THAD (arrowheads) in lateral segment of left lobe of the liver is associated with early appearance of subsegmental portal vein enhancements (arrowheads) on the arterial phase dynamic CT. **b** Equilibrium phase of contrast-enhanced CT shows normalized attenuation density of the previously enhancing segment. Linear low-attenuation densities suggests portal tract injuries (arrows) with alongside venous compromise. **c** Portal phase dynamic CT taken 5 months after TACE reveals that the considerable parenchymal atrophy (arrowheads). Subsegmental portal vein obliteration (arrow) is also demonstrated

change of the hepatic arterial or portal venous perfusion, which appears as an area of high attenuation during the hepatic arterial dominant phases and returns to normal or nearly normal attenuation on the portal phase images

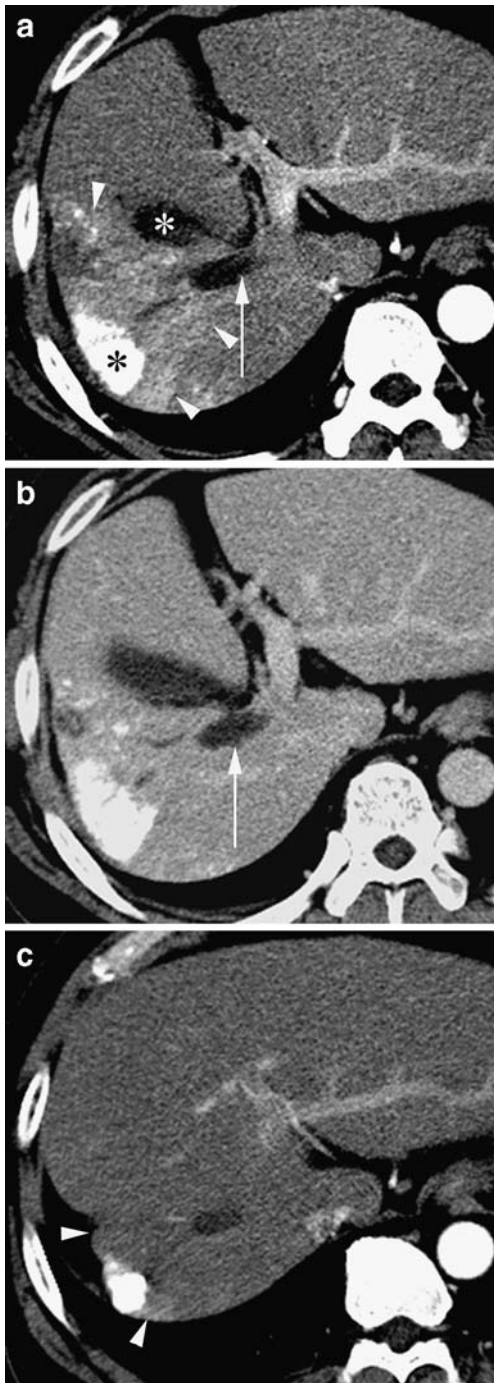


Fig. 3 A 50-year-old man with a single hepatocellular carcinoma treated by a single session TACE showing iatrogenic portal tract injury consisting of biloma and portal vein obliteration in right lobe of the liver. **a** Arterial phase dynamic CT obtained 3 weeks after TACE shows a wedge-shaped increased attenuation area (arrowheads) surrounding the tumor with iodized-oil accumulation (black asterisk) associated with the linear non-enhancing low density of portal tract injury (arrow). White asterisk=gallbladder. **b** Portal phase dynamic CT of the same level as **a** shows obliteration of right portal vein related to the biloma along the portal tract (arrow). **c** Five-month follow-up arterial phase dynamic CT shows that the THAD disappeared with marked parenchymal atrophy of right lobe of the liver (arrowheads)

during the multiphase dynamic CT [9]. The involved sites are usually subcapsular wedge-shaped with a well-defined straight border; however, they may have rather variable appearances related to a large variety of liver disorders [10, 11].

THAD is often caused by portal vein obstruction, and the resulting well-defined area of hyperattenuation during the hepatic arterial phase on dynamic CT is caused by increased arterial flow to compensate for the diminished portal vein flow and by the decreased dilution of the contrast material by the nonopacified portal vein flow [3, 4]. Patients with cirrhosis are at high risk for developing portal vein thrombosis due to portal hypertension and venous stasis. Other inflammatory disease processes (e.g., abscess, cholecystitis, cholangitis and pancreatitis), neoplasm, or hypercoagulable states can also induce portal venous thrombosis [2, 4, 12]. To avoid any bias induced by the non-TACE-induced condition, gross portal vein thromboses on the pre-TACE CT were excluded from the subjects of our study through the preliminary review of the

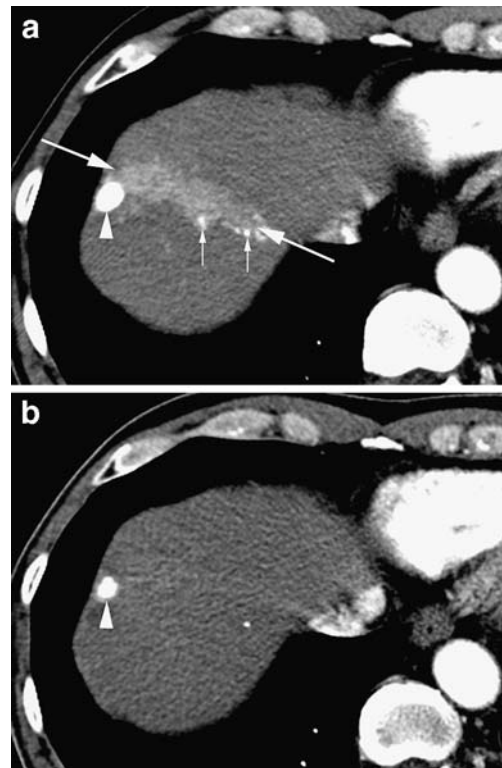


Fig. 4 A 49-year-old man who had undertaken two separated sessions of TACEs for a single hepatocellular carcinoma showing THAD without gross appearance of portal portal tract injury. **a** Arterial phase dynamic CT obtained 4 weeks after the second chemoembolization shows a localized increased attenuation density (large arrows) at the subsegment including the subcapsular iodized-oil accumulation (arrowhead). Iodized-oil densities in the peripheral vascular structures are also demonstrated (small arrows). **b** Three-month follow-up arterial phase dynamic CT shows disappearance of THAD without gross parenchymal atrophy except the localized contraction of iodized-oil accumulation lesion (arrowhead)

Table 2 Number of THADs according to the various assumptive predisposing factors of arterioportal fistula or portal venous stenosis

Characteristic	THADs with APF* (n=18)	THADs with PVS* (n=9)	THADs without APF and PVS (n=15)
Child-Pugh classification			
A	15 (83)	5 (56)	11 (73)
B	3 (17)	4 (44)	4 (27)
	<i>P</i> =0.48	<i>P</i> =0.54	
Total number of TACE			
1	6 (33)	5 (56)	7 (47)
2–4	10 (56)	4 (44)	6 (40)
5 or more	2 (11)	0	2 (13)
	<i>P</i> =0.71	<i>P</i> =0.77	
Level of TACE			
Subsegmental	6 (33)	2 (22)	3 (20)
Segmental/ lobar	12 (67)	7 (78)	12 (80)
	<i>P</i> =0.53	<i>P</i> =0.34	
Use of gelatin sponge fragments			
Used	16 (89)	9 (100)	14 (93)
Not used	2 (11)	0	1 (7)
	<i>P</i> =0.42	<i>P</i> = 0.51	

Data in parentheses are percentages in each factor
 THAD=transient hepatic attenuation difference, APF=arterial-portal venous fistula, PVS=portal venous stenosis; *each group included two THADs simultaneously showing APF and PVS. All of the *P* values were calculated comparing the number of THADs showing APFs or PVSs with the number of other THADs showing no such features depending on each factor

THADs. After TACE, localized intrahepatic bile duct injury consisted of acute biloma formation or chronic stricture and dilatation from the chemical and ischaemic insults easily led to narrowing or obliteration of the alongside portal vein branches by the compression effect combined with phlebitis [13]. In our study, ten (25%) TACE-induced THADs were related to portal vein narrowing accompanied by adjacent bile duct injuries.

Aside from the compensatory increase in arterial perfusion at the area of decreased portal venous perfusion, regardless of mechanism, arterioportal shunts are another cause of THADs on dynamic CT. Cirrhosis itself or previous history of direct parenchymal injury that is associated with fibrotic, ischaemic or inflammatory alteration of vascular integrity has been proposed as explanation for these shunts [14, 15]. Besides blunt or penetrating trauma including iatrogenic injuries related to the percutaneous transhepatic procedures or surgery, arterioportal shunts used to develop after TACE for treatment of hepatic malignancy [7, 14]. Ischaemic or chemical injury of small arterioles in the portal track could result in a direct communication between the hepatic artery and adjacent

portal vein branches. Even though depending on the size of vascular communications, direction of the vessels in the axial plane, temporary perfusion pressure of hepatic artery or proper timing of the arterial-phase imaging during the dynamic studies, early contrast filling in the peripheral subsegmental portal vein branches before the central portal vein enhancement is a representative finding of APF that is frequently demonstrated with the subsequent THAD after TACE. TACE-related arterioportal shunt tends to be large enough to show APF more frequently than the arterioportal shunts from cirrhosis itself [3, 4], and in our study, there were 18 TACE-induced THADs showing direct finding of APF as early enhancement of portal vein branches through THAD area, whereas 15 THADs were not accompanied with gross APF or PVS on the follow-up CT examinations. For the two THADs with APFs, bile duct injuries and PVSs were also present in the same area, suggesting the same mechanism of TACE-induced portal tract insults. As in our study, most of the iatrogenic localized APFs are reported to close spontaneously [15], and persistent high-flow APF causing hyperkinetic portal hypertension is considered relatively rare [3].

Hepatic parenchymal atrophy, a well-known complication of TACE, is related to ischaemic injury, especially in patients with decreased portal venous perfusion caused by thrombosis or in those who have undergone repeated embolization [6]. The results of numerous investigations have demonstrated that TACE-induced bile duct injury compromises the alongside portal venous inflow, which is related to the portal tract inflammation from chemical and ischaemic insult by embolic materials or extravasated bile, leading to phlebitis and thrombus formation in addition to biloma or dilated intrahepatic bile duct radicles compressing the lower-pressure portal venous radicles [13, 16]. Depending on the chronicity of portal venous depletion, hepatocyte dysfunction and fibrotic changes with parenchymal shrinkage may follow [9]. In our study, all TACE-induced THADs showing PVS were spontaneously regressed with hepatic parenchymal atrophy during follow-up period. For APFs, in the meanwhile, the overall sinusoidal perfusion pressure for the affected area is not decreased, and the ratio of portal venous and hepatic arterial perfusion is always changing, depending on the cardiac cycle (increased arterial flow on systole) [17] and food ingestion (increased portal flow) [18]. This is quite different from PVS in which incomplete compensation by the arterial flow can dominate the physiology and used to result in a functional infarct leading to parenchymal atrophy [19–20]. In the present study, 15 (94%) of 16 THADs with APF without PVS were spontaneously regressed without gross parenchymal atrophy.

We evaluated possible predisposing factors to make TACE-induced portal tract injuries through the comparative analysis between the number of APF or PTS appearance and the number of THADs without gross appearance of such features. According to the previous studies, non-

cirrhotic liver or cirrhotic liver with good liver profile (Child-Pugh class A) or more selective peripheral embolization are more vulnerable to the bile duct injuries reflecting the portal tract injury after TACE [21]. In this study, however, we could not find any statistically significant differences of various predisposing factors for the gross appearance of APF or PVS among the limited number of 40 THADs. The deficient statistical value in this study might be related to the small number of control groups; however, this study was not designed to evaluate the overall prevalence of APFs or PVSs in all of the patients treated by TACE, and further discussion is beyond the scope of this article.

A limitation of our study requires comment. Due to the nature of the study dealing with the gross imaging features, there was no pathologic proof for the presence of fistula between the hepatic artery and portal vein branches at the area of THADs. In the same way, we had no proof about whether the portal vein narrowing originated from the phlebitis or simply from narrowing due to a simple compression effect related to adjacent bile duct insults. For the THAD cases (n=15) that did not show direct evidence of APF or PVS, speculation about the mechanism of THAD was not possible. There might be several possibilities, including suboptimal timing of the arterial phase imaging, inadequate angle in the fixed plane of axial direction for visualization of fistulous vasculature or

suboptimal spatial or temporal resolution for depiction of obliterated portal vein branches. However, our study was just designed to investigate the fate and clinical implication of the TACE-induced THADs according to the gross dynamic CT features rather than a direct imaging-pathologic correlation. According to the results of our study, we could reliably classify the different imaging features, which showed different rates of substantial parenchymal changes, and in addition clarify the temporal nature of the THADs.

Conclusion

Almost all of the TACE-induced THADs in the cirrhotic liver spontaneously regressed during the long-term follow-up periods in conjunction with or without hepatic parenchymal atrophic changes. Portal venous compromise closely related to the alongside bile duct injury is a prerequisite for the gradual parenchymal atrophy at the area of THADs, whereas APF, which is also spontaneously closed during the variable follow-up period, does not induce any gross parenchymal changes. Understanding the nature and gradual changes of the CT features of TACE-induced THADs would be helpful for radiologists in determining the prognosis of the TACE-induced hepatic injuries as well as distinguishing them from the recurrent tumour.

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