Intersegmental Attenuation Difference on CT Caused by Partial Budd–Chiari Syndrome Secondary to Hepatocellular Carcinoma: Report of Two Cases

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For two patients with partial Budd–Chiari syndrome secondary to hepatocellular carcinoma, dynamic CT was evaluated. The obstructed hepatic veins were both the middle and left hepatic veins in Case 1 and the right hepatic vein in Case 2. The area affected by obstructed hepatic vein(s) was seen as low density on both unenhanced and contrast enhanced CT in Case 1 and as high density on enhanced CT in Case 2. The border of attenuation differences caused by the obstruction of the middle (Case 1) or right (Case 2) hepatic vein was intersegmental planes of the anterior segment of the right lobe, and that caused by the obstruction of the left hepatic vein was the intersegmental plane of the medial third of the left lateral segment. Once intersegmental attenuation difference is recognized on CT, partial Budd–Chiari syndrome should be considered.

Key words: Budd–Chiari syndrome (partial) – CT – hepatocellular carcinoma

INTRODUCTION

With the advance of such CT technology as table incremental CT and helical CT, several types of attenuation differences on CT due to the obstruction of the portal vein (1–3) or hepatic veins (Budd–Chiari syndrome) (4,5), or due to arterio-portal shunt have been encountered in routine study. However, to our knowledge, CT imaging features of partial Budd–Chiari syndrome (obstruction of one or two of the three major hepatic veins) have only received a few reports (4,6,7).

CASE REPORTS

CASE 1

A 66-year-old man presented with fever (39°C) and jaundice, and a hepatic tumor was detected by ultrasonography (US) at another hospital. Subsequently, hepatic angiography was carried out and a hypervascular tumor strongly suggestive of hepatocellular carcinoma (HCC) was demonstrated in the caudate lobe. Transcatheter arterial embolization therapy with emulsion of Lipiodol and 50 mg of doxorubicin followed by gelatin sponge particles (Lip-TAE) was performed in the right hepatic artery and he was referred to our hospital for surgery.

On admission, slight elevation of GOT (39 IU/l; normally <26) and GPT (58 IU/l; normally <26) were recognized. Hepatitis B virus surface antigen (HBsAg) was negative and hepatitis C virus antibody (anti-HCV) was positive. Alpha-fetoprotein (AFP), normally <20 ng/ml, was high at 498 ng/ml. Unenhanced CT showed a slightly low density area with tumor containing Lipiodol whose main location was segment 1. Table incremental CT in the arterial dominant phase, which started 40 sec after the beginning of the injection of 90 ml of 65% methylglucamine diatrizoate, at a speed of 2 ml/sec, demonstrated tumor thrombi in both the middle and left hepatic veins (Fig. 1a). At 1 cm caudal (Fig. 1b), a fan shaped low density area was bounded by two lines; one is the ventral half of the anterior segment and the other the medial third of the left lateral segment. Late enhanced CT performed five minutes later showed the whole liver as isodense. Hepatic angiography demonstrated thread and streak signs in the middle and left hepatic veins which coincided with tumor thrombi seen in arterial dominant enhanced CT. Lipiodol-CT (Fig. 1c) performed immediately after the injection of 5 ml of Lipiodol into the right hepatic artery for diagnosis of daughter tumors showed the retention of Lipiodol only in the right lobe of the liver, which was clearly bounded from the left lobe by the Cantlie line.

CT arterial portography (CTAP) performed one year later still showed filling defects in the middle and left hepatic veins due to the tumor thrombi (Fig. 1d). The patency of the right and superficial left hepatic veins was confirmed.
CT of partial Budd–Chiari syndrome

Figure 1. Case 1. Hepatocellular carcinoma arising from the caudate lobe with tumor thrombi in both the middle and left hepatic veins. (a) Table incremental CT in the early phase showing enhancement of the tumor thrombi of the middle (arrow) and left (curved arrow) hepatic veins. Main tumor abutting on tumor thrombi contains Lipiodol injected for treatment. (b) At 1 cm caudal to (a), a fan shaped low density area corresponding to the low density one in unenhanced CT (not shown) is seen. The border of different attenuation is shown by the two different intersegmental planes; one is midplane of the anterior segment (arrows) and the other of the medial third portion of the left lateral one (curved arrows). An arrowhead indicates the ventral branch of the anterior portal veins. (c) Lipiodol-CT demonstrating the retention of Lipiodol only in the right lobe after the injection of Lipiodol in the replaced right hepatic artery. Note the discrepancy of the border between the interlobar line (Cantlie line) and the intersegmental one observed in (b). (d) CT arterial portography performed one year later shows filling defects of the middle and left hepatic veins due to tumor thrombi. Instead, the right (arrow) and thick dilated superficial left (curved arrow) hepatic veins are observed.

Case 2

A 66-year-old man had undergone esophagostomy for esophageal cancer two years before. During follow-up, a tumor was asymptptomatically detected in segment 7 of the liver by US. Angiography disclosed a hypervascular tumor and Lip-TAE was performed in the right hepatic artery. The patient was referred to our hospital for surgery.

On admission, slightly abnormal liver function was recognized; GOT of 59 IU/l and GPT of 57 IU/l. HBsAg was negative and anti-HCV was positive. AFP was 378 ng/ml and PIVKA-II was 0.104 AU/ml (normally <0.063). Unenhanced CT (Fig. 2a) showed a 7 × 4 cm low density mass with slight retention of Lipiodol in the central area and a fan shaped slightly low density area lateral to the mass. Helical CT was performed 40 sec (arterial dominant phase), 70 sec (portal dominant phase) and five min (late phase) after the injection of 100 ml of iopamidol (300 mg I/ml) at a speed of 3 ml/sec with a 7 mm slice thickness (1:1 pitch).

The fan shaped area was well enhanced in the arterial dominant phase (Fig. 2b), was changed to low density in the portal dominant phase (Fig. 2c) and isodensity in the late phase. The right hepatic vein was not demonstrated in any phases. The border of different attenuation (Figs. 2a, 2b and 2c) was seen in the midplane of the anterior segment of the right lobe (intersegmental attenuation difference). The tumor was depicted as a low density one with slight enhancement within it in the arterial dominant phase (Fig. 2b).

Discussion

As an attenuation difference of the liver seen on CT, lobar or segmental attenuation difference secondary to the obstruction of
Figure 2. Case 2. Hepatocellular carcinoma located in segment 7 associated with the obstructed right hepatic vein. (a) Unenhanced CT demonstrating a 6 × 4 cm hepatocellular carcinoma with central retention of Lipiodol injected previously. A fan shaped area with slightly low density (arrowheads) spreads right lateral to the tumor. An arrow shows retention of Lipiodol in the intrahepatic metastasis. (b) In the arterial dominant phase of helical CT, the fan shaped area is well enhanced by contrast medium. Only the hepatic artery and portal vein are delineated in this phase. (c). In the portal dominant phase, the fan shaped area becomes low density. The right hepatic vein is not visualized, in spite of the opacification of the middle (arrow) and left (curved arrow) hepatic veins.

the portal vein is well known (1–3). However, the attenuation difference due to the obstruction of one or two of the three main hepatic veins (partial Budd–Chiari syndrome) is still rarely recognized (4,6,7), even though that of Budd–Chiari syndrome (total obstruction of all main hepatic veins and/or the inferior vena cava) is well known (4,5).

To date, the following findings are given as characteristics of Budd–Chiari syndrome: for angiography, thin and stretched hepatic arteries, prolonged sinusoidogram and reversed portal blood flow (7,8) and for CT, low density area in the peripheral (subcapsular) zone with obstructed drainage veins and high density area in the central zone (perihepatic area and caudate lobe) on both unenhanced and postcontrast CTs (5). The peripheral zone is more enhanced than the central one by CT arteriography (CTA) and vice versa by CTAP (4,5). The following hemodynamic changes are postulated: in the peripheral zone of the liver, the portal vein plays a draining role to compensate for the obstructed hepatic veins (7,8) and consequently dense opacification similar to the arterio-portal shunt is seen. However, in the central zone, normal hepatic circulation is kept via the compensated enlarged caudate vein and/or inferior right hepatic one.

Recently, hemodynamic change in the liver due to the occlusion of one hepatic vein by an inflated balloon catheter has been reported: Kanazawa et al. (9) showed in pigs, by both angiography and radioisotope studies, increased hepatic arterial blood flow in the area where the hepatic vein is occluded. A similar study was performed using CTA and/or CTAP in human liver by Murata et al. (10); the area whose draining vein is blocked by the inflated balloon is clearly demonstrated as a fan shaped perfusion defect on CTAP and as higher density one on CTA corresponding to the perfusion defect area on CTAP. The mechanism of decrease of portal blood flow and increase of arterial blood flow could be explained as follows: the arterial blood flow with much higher pressure than the portal blood flow regurgitates into the portal vein via the transsinusoidal route and replaces the portal blood flow.

Case 1 did not theoretically show early enhancement of the affected area by the obstructed hepatic vein(s) in the arterial dominant phase of dynamic CT (Fig. 1b), whereas Case 2 showed early enhancement (Fig. 2b) which was consistent with the results of animal experiments (9). This could be because of the influence of Lip-TAE performed in Case 1 prior to the CT, and/or obstruction by the tumor thrombi caused hypervascularity in Case 1 rather than compression by the tumor as in Case 2.

The border of attenuation differences on CT passed through the intersegmental plane of the anterior segment of the right lobe, i.e., in the midplane of the ventral and dorsal area of the anterior segment, when the obstruction occurred in the middle hepatic vein in Case 1 (Fig. 1b) or in the right hepatic vein in Case 2 (Fig. 2b). This border is definitely different from the interlobar line (Cantlie line) which is frequently seen in the obstruction of one of the main (first order) portal veins (1–3) and was delineated by Lipiodol CT in Case 1 (Fig. 1c). The intersegmental line is possibly due to the difference of drainage of hepatic blood flow; the dorsal half of the anterior segment in Case 1 and the ventral half of it in Case 2 drain into the hepatic vein via the neighboring branch of the right and middle hepatic veins.
The border of attenuation difference caused by the obstructed left hepatic vein in Case 1 was seen in the medial third portion of the left lateral segment (Fig. 1b). The left lateral area with normal hepatic blood circulation was thought to drain to the compensated enlarged superficial left hepatic vein; this was confirmed by CTAP (Fig. 1d). Even though the relationship between the segment of the liver and the draining veins is evaluated by imaging, especially in the right lobe (11, 12), further study seems necessary in the left lobe.

References