Transient hepatic attenuation differences at biphasic spiral CT examinations

Kutsi Köseoğlu, Füsun Taşkın, Yelda Özsunar, Burak Çildağ, Can Karaman

ABSTRACT
Transient hepatic attenuation difference (THAD) is a perfusion disorder that can sometimes be observed in hepatic arterial phase of a biphasic spiral CT examination. The involved site of liver by THAD appears as an area of high attenuation on the hepatic arterial phase image and returns to normal attenuation on portal venous phase image. The knowledge of the shape, distribution and causes of THAD is essential to differentiate the THAD from other pathologies such as neoplasm.

Key words: • tumor • liver • tomography, spiral computed

Transient increase in the hepatic attenuation that can be confused with tumoral lesions can be observed in the arterial phase of biphasic hepatic spiral CT examinations (1-4). While transient hepatic attenuation differences (THADs) appear as areas of high attenuation in arterial phase, they are observed to be isodense with normal parenchyma on portal venous phase images. Transient hepatic attenuation differences are wedge shaped areas with regular contours. They do not cause mass effect on the vascular structures and normal hepatic vascular structures are seen passing through them (2, 3). Transient hepatic attenuation differences can be encountered in an entire lobe (lobar), in a segment (segmentary), in a subsegment (subsegmentary) or in a subcapsular area of the liver.

Transient hepatic attenuation differences may be noted to accompany tumors; however, they may occur due to non-tumor vascular reasons as well. Among the reasons of THAD are arterioportal shunts, vascular compressions affecting the hepatic artery, portal vein or hepatic vein, inflammatory changes, vascular variations, hepatic parenchymal compressions and steal syndrome in hypervascular tumors.

Arterioportal shunts
Arterioportal (AP) shunts may develop as a result of trauma, tumors, interventional processes and liver cirrhosis. They may develop due to macroscopic fistulas secondary to trauma, transsinusoidal microscopic shunts (between the interlobular artery and venules), transvasal routes (due to tumor thrombus), transtumoral routes (in hypervascular tumors) and transplexal routes (peribiliary) (3). In the event of occurrence of obstruction in the portal vein, these shunts play an important role in the achievement of flow.

Arterioportal shunts are among the frequent reasons of THADs and it may be difficult to differentiate the shunts from tumors when they accompany vascular tumors. Hepatocellular carcinoma (HCC) is one of the most frequent tumoral reasons of the AP shunts in the liver. Hemangiomas and cholangiocellular carcinoma are also among the tumoral causes of AP shunts. Liver biopsies, abscess drainages and biliary drainages are among the iatrogenic causes of AP shunts (5).

In spiral CT examinations, the AP shunts are seen in the arterial phase as enhancing branches of the portal vein before the enhancement of the main portal vein where AP shunts may be accompanied by wedge shaped THADs (2, 3) (Figures 1 and 2). If THAD areas are accompanied by tumors, they are generally observed as areas of high attenuation with regular contours located in the periphery of the tumor and in the portal phase areas of THAD return to normal attenuation.
Vascular compressions

Hepatic artery invasion

As a result of the invasion of the hepatic artery due to tumoral lesions, the hemodynamic balance between the portal vein and the hepatic artery in the affected lobe or segment may change. Areas of THAD may be seen due to the decrease in the blood flow of the affected hepatic artery (2).

Obstruction of the portal vein

Transient hepatic attenuation differences due to obstruction of the portal vein generally develop due to compression or invasion of the portal vein branches by tumors. Hepatic arterial blood flow increases due to obstruction of the portal vein. In the arterial phase of the spiral CT, increased arterial blood flow and the decreased flow in the portal vein, which is not yet enhancing, causes development of THAD in certain regions of the liver which are affected by the ob-

Figure 1. a-c. THAD due to arteriportal shunt is seen in a patient with HCC (post-contrast transverse spiral CT images). a. Wedge shaped increased attenuation anterior to the mass and pointing to the portal hilus is seen in the hepatic arterial phase (30 sec) (arrows). b. Above mentioned area (arrows) in the portal venous phase (70 sec) in the same slice is seen to be isodense with the normal parenchyma. c. A portal vein branch (arrow) due to AP shunt is enhancing earlier than main portal vein and splenic vein, in a more caudal image.

Figure 2 (below). a, b. In another HCC case, THAD due to microscopic arteriovenous shunt is seen accompanying the mass (post-contrast transverse spiral CT images). a. Enlarged hepatic artery and peripheral THAD (arrows). b. The mass lesion is seen in a more cranial slice (arrowheads).
struction (Figure 3). Areas of THAD that develop as a result of portal obstruction are seen as wedge shaped areas with regular contours pointing towards the portal hilus (3). Areas of THAD can be seen in a whole lobe, a segment, or a peripheral subcapsular region.

**Hepatic venous obstructions**

The flow in the portal vein may decrease or revert in hepatic venous obstructions due to the increase in the sinusoidal pressure. This may then cause increase in the arterial flow and development of AP shunts, as seen in cirrhosis.

As in the case of portal vein obstruction, areas of THAD can be observed in heart failure, mediastinal diseases and Budd-Chiari syndrome (BCS) (Figure 4). However, the edge of the wedge shaped THAD, which points towards the portal hilus in portal vein obstruction, points towards the vena cava in

**Figure 3.** a, b. Hypovascular metastatic lesion in the 7th segment of the liver and THAD that has developed due to the compression (stasis) of the portal vein branches by the mass is seen in the periphery of the mass lesion (post-contrast transverse spiral CT images). a. Mass lesion (*arrowhead*) and THAD (*arrow*) are seen in the arterial phase. b. In the portal venous phase, THAD becomes isodense with the liver and normal course of a hepatic vein branch through THAD is seen (*arrow*).

**Figure 4 (below).** a, b. Thrombus in the inferior vena cava at hepatic level and THAD in the caudate lobe are seen (post-contrast transverse spiral CT images). Since the venous drainage of the caudate lobe is different, the THAD due to the hepatic stasis is seen as an increased attenuation area (*arrow, a*). In the portal phase (*b*) THAD area becomes isodense with the liver.

**Figure 5.** a, b. Acute cholecystitis and the accompanying THAD (post-contrast transverse spiral CT images). a. In the arterial phase, THAD (*arrow*) adjacent to the gall bladder is seen. b. In the portal phase, THAD and liver parenchyma are isodense with each other.
hepatic vein occlusions. The THADs that are observed in the venous occlusions may continue in the venous phase as well (3).

**Inflammatory lesions**

The inflammatory changes that occur in the liver and the neighbouring organs may result in THAD which is due to hyperemia and edema as a result of increased arterial flow and stasis in the portal vein. Liver abscess, cholecystitis and cholangitis are classified in this group (6). In such cases, the THADs are observed as hyperattenuating in the arterial phase and isoattenuating with liver in the portal phase adjacent to inflamed tissues (Figures 5 and 6).

**Vascular variations**

Vascular variations such as capsular vein, accessory cystic vein and aberrant gastric vein may drain the systemic venous circulation into the liver (7). In the presence of such vascular variations, the systemic venous flow that drained nonopacified blood into liver may cause appearance of THAD.

**Figure 6.** a-c. Appearance of THAD in a patient with posttraumatic subcapsular hematoma who has developed cholecystitis during the follow-up period (post-contrast transverse spiral CT images). a. Subcapsular hematoma (arrow) and THAD areas are seen in the arterial phase. b. THAD areas (arrows) adjacent to cholecystitis in the left lobe and adjacent to the hematoma in the right lobe are seen at a more cranial level. c. Portal venous phase image shows THAD areas to be isodense with the liver.

**Figure 7.** a, b. Post-contrast transverse spiral CT images. a. Subcapsular THAD (arrow) due to vascular variation is seen in the arterial phase. b. The subcapsular THAD area becomes isodense to the liver parenchyma in the portal phase.

**Figure 8.** a, b. Post-contrast transverse spiral CT images. In another patient, in arterial phase (a) and in portal phase (b) the THAD (arrow, a), due to vascular variation, is seen in the subcapsular region adjacent to the gall bladder.
in the affected liver segment (3). Transient hepatic attenuation differences that occur due to vascular variations are often seen in the neighbourhood of the gall bladder and the falciform ligament, anterior border of the porta hepatis, and in the subcapsular region (Figures 7 and 8).

**Hepatic parenchymal compressions**

When there is compression in the parenchyma of the liver, the portal venous flow decreases due to the pressure and the hepatic arterial flow in the affected segment increases. Reasons like rib compressions, peritoneal implants, perihepatic collections, and masses (2) (Figures 9 and 10) may cause THAD. When the compression is no longer present, THAD is noted to disappear.

**Other causes**

The steal syndrome seen in hypervascular tumors can cause THAD in the parenchyma adjacent to the tumor. Portal hypertension and fibrosis...
due to cirrhosis are among the other reasons that cause THAD. Transient hepatic attenuation differences that appear in the medial segment of the left lobe due to the diversion of flow into the paraumbilical veins secondary to the occlusion of the superior vena cava are rarely seen (8).

In addition to the above mentioned factors, the causes for some THAD occurrences remain unknown (Figure 11).

Conclusion

Transient hepatic attenuation differences appear as a result of hepatic hemodynamic changes and are differentiated with their typical locations and configurations in the biphasic spiral CT examinations. Knowing the characteristics of THAD and differentiating them may prevent the tumor diameter from being measured larger than it actually is as in the case of hypervascular tumors such as HCC. In the arterial phase, it also prevents the misinterpretation of high attenuation areas, which are due to hemodynamic changes, as true lesions. Nevertheless, if these areas that are observed in the arterial phase are still suspicious, further examinations should be performed to rule out clinically significant lesions.

References