Transient Hepatic Attenuation Differences in Computed Tomography from Extrahepatic Portal Vein Compression

Humberto Wong, MD, Terry S. Desser, MD, and R. Brooke Jeffrey, MD

Objective: To describe the appearance of transient hepatic attenuation differences (THADs) of extrahepatic origin.

Materials and Methods: Five cases of THADs produced by compression of the extrahepatic portal vein at its confluence with the splenic vein were identified prospectively over a four-month period. Two additional cases of peripheral THADs resulting from main portal vein thrombosis were identified from retrospective review of a departmental database.

Results: Streamlining of portal venous flow resulted primarily in left lobar THADs when the portal vein is compressed at its confluence with the splenic vein. THADs were seen in the periphery of the liver in the two cases of main portal vein thrombosis.

Conclusion: Lobar and/or peripheral THADs can be produced by compromise of splenic and extrahepatic portal venous flow. Radiologists should be familiar with the “central pseudotumor” created by the peripheral THAD that can result from portal vein thrombosis.

Introduction

The liver has a dual blood supply, with approximately 70% of its perfusion coming from the portal vein and 30% from the hepatic artery. Relative to the duration of a CT scan through the liver performed with current generation scanners, there is significant temporal separation between the arrival time of the iodine bolus between the two vessels following intravenous contrast administration, with the hepatic artery enhancing earlier than the portal vein. If the proportion of hepatic arterial supply relative to portal supply is constant across the different regions of the liver, the hepatic arterial flow may appear earlier in the portal venous phase compared to the hepatic arterial phase.
Transient hepatic attenuation differences (THAD) are areas of enhancement on hepatic arterial phase imaging computed tomography that occur as a result of a localized variation in the proportion of hepatic arterial and portal venous supply. If there is regional diminution in portal flow because of venous thrombosis or elevated hepatic sinusoidal pressure, then hepatic arterial blood supplying that region will not be diluted by unopacified portal blood and a focal area of increased attenuation will be present during scanning in the arterial phase. The compensatory increase in hepatic arterial flow that occurs when portal flow diminishes accentuates this phenomenon.

In the current era of rapid multiphasic scanning, segmental and subsegmental THADs caused by intrahepatic abnormalities such as segmental portal branch occlusion, arterioportal shunting through tumors, biliary obstruction, and hepatic venous outflow obstructions have become quite familiar. However, we have observed that THADs may also be caused by extrahepatic alterations of portal venous flow, and in these cases the resultant perfusion patterns in the liver may be less predictable. We will illustrate a variety of perfusion abnormalities in the following series of cases of extrahepatic portal vein occlusion or compression.

We describe five patients with THADs secondary to compression of the main portal vein near or at the confluence of the splenic and superior mesenteric veins, which resulted in left lobar THADs. We also illustrate THADs occurring in the periphery of the liver in two cases as a consequence of main portal vein occlusion.

Case Reports

Over a four-month period, five cases with transient hepatic attenuation differences due to extrahepatic venous compression were identified prospectively during review of abdominal CT scans performed for a variety of indications. An additional two cases of peripheral THADs due to portal vein occlusion were identified from retrospective search of a department of radiology teaching file. Except where noted, these cases were acquired using a multiphasic liver protocol with either 8 and 16 multi-detector row CT. Using bolus tracking technique, non-ionic iopamidol 370 contrast was administered intravenously as a single bolus at 4 cc per second in order to acquire the hepatic arterial phase. This was followed by further imaging at approximately 70 seconds after the start of the injection in order to acquire a portal venous phase and, finally, a delayed hepatic phase at 3 minutes after the original injection of contrast. While coronal reconstructions are not routinely performed, the raw data is acquired with 1 mm collimation and therefore available to be reviewed on an independent 3-D workstation, which is at the discretion of the reviewing radiologist.

Cases With Extrahepatic Portal Venous Compression

Case 1

A 40-year-old man initially experienced an episode of maroon-colored and tarry stools, fatigue and tachycardia for which he was seen in the ER. An EGD performed at the time demonstrated bleeding gastric varices. He was stabilized and eventually discharged home. A month later he experienced a second episode of maroon-colored, tarry stools and jaundice. A CT scan at the time revealed a pancreatic head mass without involvement of the regional vessels. The patient underwent endoscopic ultrasound-guide fine-needle aspiration which yielded a presumptive diagnosis of pancreatic adenocarcinoma. He underwent his first round of chemotherapy and then was referred to our institution where a pancreatic protocol CT was performed and a 4.2 x 5.0 x 4.0 cm heterogeneous ill-defined mass involving the head and neck of the pancreas was found.
There was main pancreatic ductal dilation, arterial encasement, and encasement of both the superior mesenteric and splenic veins with abrupt termination of venous flow near confluence with the portal vein and reconstitution of the portal vein via collaterals (Figure 1A). THADs were seen in the periphery of the right and left liver lobes (Figure 1B).

**Case 2**

A 20-year-old man sustained pancreatic trauma in a motor vehicle accident. He was initially treated at an outside institution and then referred to our institution for definitive repair of his maturing pancreatic pseudocyst. On CT, a multiloculated, rim enhancing fluid collection measuring 14 x 11 x 23 cm was seen within the right abdomen, with venous compression at the confluence of the superior mesenteric and splenic veins (Figure 2A). A left lobar THAD was seen on the arterial phase (Figure 2B), and a replaced left hepatic artery from the left gastric artery was incidentally noted.
Case 3
A 60-year-old man initially presented with a cough and was initially thought to have allergies or pneumonia. A chest x-ray revealed extensive right lung consolidation that did not clear over the course of several weeks. A CT of the thorax revealed multiple pulmonary nodules and mediastinal adenopathy, bronchoscopy was performed and non small cell carcinoma favoring a lung primary was diagnosed. A staging abdominal CT demonstrated a 3.2 x 2.4 cm nodal metastasis compressing the portal venous confluence (Figure 3A) and a THAD involving the caudate and left hepatic lobe (Figure 3B).

Figure 3A. 60-year-old man with lung cancer. CT shows external compression of the portal confluence by nodal metastases (arrow).

Figure 3B. CT shows resultant THAD in left lobe of the liver.

Case 4
A 32-year-old woman initially underwent a laparoscopic appendectomy for acute appendicitis. Two months later, she presented with high fevers, and right upper quadrant tenderness, and a white blood cell count of 22 K. A CT scan was performed and a 5 x 4 cm extrahepatic abscess was identified in the portocaval space, with mass effect on local structures including caudate lobe of the liver, and the SMV, SMA and portal vein (Figure 4A). A left lobe THAD was seen (Figure 4B). Subsequent scan following resolution of the abscess showed disappearance of the THAD.

Figure 4A. 32-year-old woman with intra-abdominal abscess after laparoscopic appendectomy. CT shows compression of the portal confluence by the abscess.

Figure 4B. CT images cranial to Fig. 4A show THAD of the left lobe of the liver.
Case 5
A 20-year-old asymptomatic woman sustained blunt trauma to the epigastric area while playing soccer, after which she experienced abdominal pain, nausea and vomiting. CT scan performed at an outside institution showed a pancreatic mass, and biopsy suggested a solid pseudopapillary tumor of the pancreatic head. Pancreatic protocol CT performed at our hospital revealed a multiloculated cystic mass of the pancreatic head and neck invading the duodenum and posterior wall of the stomach. There was compression of the superior mesenteric vein and splenic vein with flow preserved in the portal vein just beyond the confluence (Figure 5A). A THAD was evident in portions of the left hepatic lobe (Figure 5B). She underwent surgical resection of the mass, which required total pancreatectomy, duodenectomy, partial gastrectomy, and splenectomy. The splenic vein was resected; the superior mesenteric vein was carefully dissected from the mass and normal flow through it was restored. Pathology confirmed the original biopsy diagnosis.

Cases With Portal Vein Thrombosis

Case 6
A 34-year-old previously healthy man presented to the emergency room with a 3 day history of increasing abdominal pain. He also complained of right upper quadrant stiffness and discomfort with coughing or laughing. Contrast-enhanced CT showed complete thrombosis of the portal vein. The hypervascular hepatic periphery representing THAD is evident (Figure 6). Decreased enhancement in central portion of liver is due to dilution with unopacified splanchic blood supplied via collaterals which are relatively abundant in central portion of liver compared to the periphery.

Figure 5A. 20-year-old with solid pseudopapillary neoplasm of pancreas. CT shows multilocular cystic mass compressing portal and splenic veins (arrow).

Figure 5B. 20-year-old with solid pseudopapillary neoplasm of pancreas. CT shows THAD is present in left lobe of liver.

Figure 6. 34-year-old man with acute portal venous system thrombosis. Contrast-enhanced CT shows expansile thrombus in main portal vein (arrow), with hypervascular hepatic periphery representing THAD. Decreased enhancement in central portion of liver is due to dilution with unopacified splanchic blood supplied via collaterals which are relatively abundant in central portion of liver compared to the periphery.
Transitient Hepatic Attenuation Differences in Computed Tomography from Extrahepatic Portal Vein Compression

Figure 7. 48-year-old woman with alcoholic cirrhosis and chronic portal vein thrombosis. Arterial phase CT shows crescentic THAD in periphery of liver, with decreased enhancement of hypertrophied central zone of liver where portal flow is relatively preserved.

Discussion

THADs in CT were first described in the setting of portal cavernoma by Itai et al. [1]. Multiple disorders can lead to transient hepatic attenuation differences including benign and malignant intrahepatic hepatic tumors, abscesses [2], and arteriovenous shunts, as well as inflammatory disorders such as acute pancreatitis [3] or acute cholecystitis [4]. Occasionally, the etiology of the THAD is not immediately identified, and depending on the clinical presentation, a follow up CT is recommended. Multiple morphologic patterns of THAD have been described including lobar multisegmental, sectorial, polymorphous and diffuse [5] or lobar, segmental, subsegmental and subcapsular [6]. Of these patterns, the lobar pattern has been described in relationship with intrahepatic masses that lead to a decrease in portal venous flow and compensatory hepatic arterial hyperperfusion. To our knowledge, few authors have focused specifically on cases that demonstrate THADs in the setting of extrahepatic disruption of portal venous blood flow.

Portal vein flow is streamlined, meaning that the superior mesenteric vein contributes a preferential amount of blood flow to the right hepatic lobe and that the splenic vein provides a larger amount of flow to the left hepatic lobe. An explicit demonstration of streamlining of portal vein flow was performed by Gallix et al. [7] using time-of-flight MR angiography with pre-saturation pulses to null the signal selectively from either the splenic or superior mesenteric vein. Using this technique they showed that there is little mixing within the portal vein. Streamlining is such that, when viewing the portal vein in cross-section, the anteromedial quadrant represents blood from the splenic vein and the posterolateral segment arises from the superior mesenteric vein [7]. Based on this, it is reasonable to expect that decreased splenic venous inflow would result in a left lobar THAD. Indeed, left lobar THADs resulting from splenic vein occlusion in pancreatitis were described by Arita et al. in three of nine patients [3]. In addition, streamlining within the portal vein itself suggests that portal compression could also result in THADs, with anteromedial compression producing left lobar THADs similar to those observed in splenic vein occlusion. In our series, we observed left lobar THADs in four cases with masses compressing the anteromedial aspect of the portal vein at the confluence.

Itai et al have pioneered the concept of the central and peripheral zones of the liver having differing potential for...
Transient Hepatic Attenuation Differences in Computed Tomography from Extrahepatic Portal Vein Compression

collateral blood supply [8]. The central or hilar region of the liver has abundant collaterals, and in the event of portal vein thrombosis continues to receive portal blood supply via collaterals. The periphery of the liver has relatively fewer areas of communication and thus when the main portal vein flow decreases, there is decreased portal flow in the periphery of the liver, increased hepatic arterial flow, and a resultant THAD. Our two cases of portal vein thrombosis illustrate this phenomenon. Radiologists should be careful not to mistake the central low attenuation region for a hepatic tumor.

In conclusion, obstruction of the main portal vein at the confluence of the superior mesenteric and the splenic veins by extrinsic masses can lead to decreased portal venous flow and relative increased hepatic arterial flow in regions of the liver in either a lobar distribution or within the liver periphery. The result will be a peripheral or lobar THAD.

References

1. Itai Y, Moss AA, Goldberg HI. Transient hepatic attenuation difference of lobar or segmental distribution detected by dynamic computed tomography. Radiology 1982; 144:835-839. [PubMed]

2. Lee KH, Han JK, Jeong JY, et al. Hepatic attenuation differences associated with obstruction of the portal or hepatic veins in patients with hepatic abscess. AJR Am J Roentgenol 2005; 185:1015-1023. [PubMed]

3. Arita T, Matsunaga N, Takano K, Hara A, Fujita T, Honjo K. Hepatic perfusion abnormalities in acute pancreatitis: CT appearance and clinical importance. Abdom Imaging 1999; 24:157-162. [PubMed]

4. Choi SH, Lee JM, Lee KH, et al. Relationship between various patterns of transient increased hepatic attenuation on CT and portal vein thrombosis related to acute cholecystitis. AJR Am J Roentgenol 2004; 183:437-442. [PubMed]

5. Colagrande S, Centi N, La Villa G, Villari N. Transient hepatic attenuation differences. AJR Am J Roentgenol 2004; 183:459-464. [PubMed]

6. Chen WP, Chen JH, Hwang JJ, et al. Spectrum of transient hepatic attenuation differences in biphasic helical CT. AJR Am J Roentgenol 1999; 172:419-424. [PubMed]