Imaging Diffuse Liver Disease – Part III

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Vascular Disorders

Vascular stasis, hypercoagulability states and endothelial disruption (Virchow's triad) are known to be main mechanisms that cause thrombosis. Portal venous stasis caused by cirrhosis, hepatocellular carcinoma or pancreatic carcinoma may lead to portal venous thrombosis. Sepsis, direct trauma, inflammatory bowel disease and phlebitis all lead to endothelial damage that may result in portal venous thrombosis. Portal venous thrombosis is usually a transient condition, however if it involves the smaller intrahepatic branches revascularisation will not occur. When it persists, cavernous transformation of the porta hepatis occurs with the formation of numerous collateral venous channels that replace the main portal vein. CT will show both thrombosis of the portal vein and the sponge-like conglomerate of veins replacing it in the porta hepatis (Figure 1). It may also demonstrate the cause of the portal vein thrombosis.

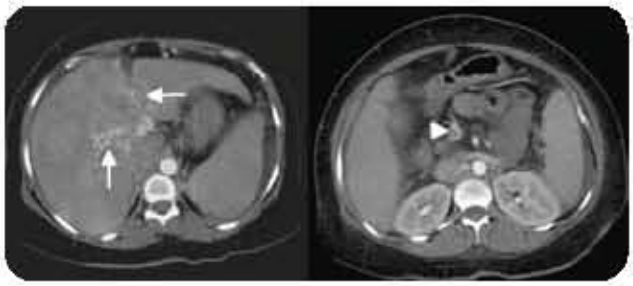

Figure 1
Cavernous transformation of the portal vein due to pancreatic cancer. Collateral veins (arrows) are seen filling the intrahepatic portion of the main portal veins. Thrombus in the extrahepatic portal vein (arrowhead).

Budd-Chiari syndrome results from occlusion of the hepatic veins and is classified into 3 types: Type 1 involves occlusion of the inferior vena cava, in type 2 there is occlusion of the major hepatic veins while in type 3 veno-occlusive disease of the liver or progressive thrombotic occlusion of small centrilobular veins is present. Stasis in the hepatic veins or IVC may result from external compression by a hepatic or retroperitoneal tumour or due to increased intraluminal pressure by congestive heart failure, constrictive pericarditis or a right atrial myxoma. CT can confirm hepatic venous or IVC thrombosis and can help identify the cause (Figure 2).

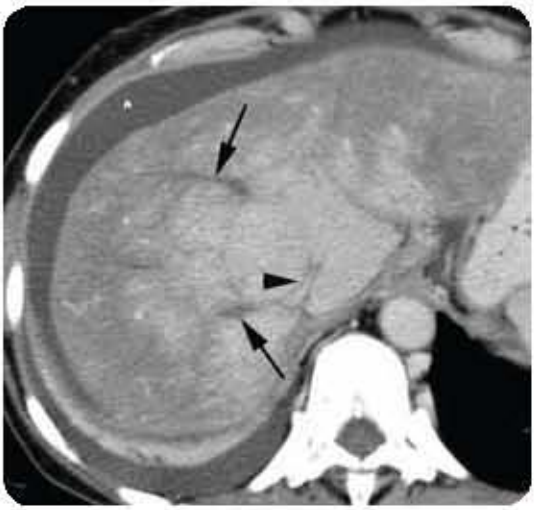

Figure 2
Budd-Chiari syndrome on CT: Portal phase CT showing diminished perfusion of the peripheral portions of the liver with increased central perfusion, thrombosed hepatic vein (arrows), a compressed IVC (arrowhead) and ascites (A).

Congestive heart failure results in stasis and increased pressure in the hepatic veins with hepatic congestion that if persistent for a long time will lead to hepatocyte necrosis, fibrosis and micronodular cirrhosis. In such cases, CT will demonstrate hepatic venous widening and evidence of cirrhosis.

Hepatic arterial occlusion is rare and is more frequently seen in transplant livers due to direct trauma. It is otherwise the result of embolic disease. The hepatic artery contributes only 25% of the liver's blood supply and if occluded does not grossly disrupt liver function. Metastases and primary liver tumours have been noted to receive most of their blood supply from the hepatic artery and chemo-embolisation of the hepatic artery is one of the treatment methods used to control tumour growth. Occlusion of the hepatic arteries, therapeutic or otherwise, is readily assessed by CT.

Significant liver infarction only occurs when both hepatic arterial and portal venous occlusion occur. Such situations include acute shock, trauma and hypercoagulability, as well as preeclampsia or HELLP (hemolytic anemia, elevated liver enzymes, low platelets) syndrome and as a vascular complication after liver

transplantation. CT will show the zone of infarction as a wedge shaped area of low density that follows the segmental vascular anatomy of the liver.

Inflammatory diseases of the liver

Viral hepatitis results in cellular alterations with varying degrees of periportal hepatocellular necrosis, Kupffer cell mobilization, and portal infiltration with plasma cells depending on the underlying infectious, toxic, or autoimmune cause. These inflammatory entities can be self-limiting, progress to segmental scarring, or end in an overall cirrhotic state. The acute variant of hepatitis lasts less than 6 months; chronic hepatitis represents any inflammatory condition of the hepatic parenchyma that does not show signs of regression for periods longer than 6 months.

CT of acute and fulminant courses of hepatitis shows generalized hepatomegaly combined with peripheral edema. Furthermore, nonenhanced CT can show heterogeneous attenuation patterns. The overall hepatic parenchymal attenuation is usually equal to or less than that of the spleen. Contrast-enhanced CT can demonstrate irregular perfusion with heterogeneous regions of diminished attenuation (Figure 3).

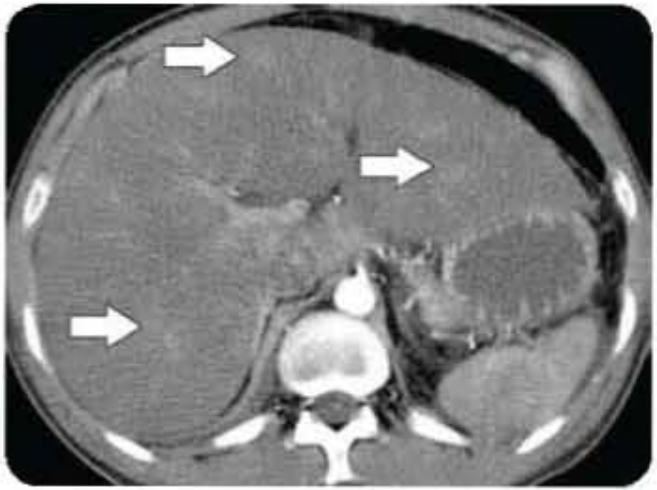

Figure 3
Acute viral hepatitis in a 39-year-old man: Arterial phase image shows heterogeneous enhancement (arrows) of the edematous enlarged liver.

Conclusion

The aim of this article was to demonstrate the prominent role of CT for the diagnosis of diffuse liver disease primarily due its excellent morphologic visualization capabilities.