Sonography of Diffuse Liver Disease

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Sonography is often the first imaging procedure performed in the evaluation of individuals with suspected liver disease. Evaluation for biliary dilatation is always performed, because bile duct obstruction can cause abnormal liver test results, raising the suspicion of liver disease. Ultrasound is a useful but imperfect tool in evaluating diffuse liver disease. We discuss the uses and limitations of sonography in evaluating parenchymal liver disease. Sonography can show hepatomegaly, fatty infiltration of the liver, and cirrhosis, all with good but imperfect sensitivity and specificity. Sonography is of limited usefulness in acute hepatitis. Increased parenchymal echogenicity is a reliable criterion for diagnosing fatty liver. Cirrhosis can be diagnosed in the correct clinical setting when the following are present: a nodular liver surface, decreased right lobe–caudate lobe ratio, and indirect evidence of portal hypertension (collateral vessels and splenomegaly). Ultrasound plays an important role in the imaging of conditions and procedures common in patients with diffuse liver disease. ***Key words:*** hepatocellular carcinoma; portal hypertension; transjugular intrahepatic portosystemic stent shunts; venous clot.

Abbreviations
CDS, color Doppler sonography; CT, computed tomography; HCC, hepatocellular carcinoma; NASH, nonalcoholic steatohepatitis; 3D, three-dimensional; TIPSS, transjugular intrahepatic portosystemic stent shunt

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Sonographic detection and evaluation of diffuse liver disease may be difficult, because diffuse liver disease does not always cause distortion of the liver parenchymal texture, internal liver architecture, or shape of the liver. This is especially true in patients with acute hepatitis. Liver surface nodularity, parenchymal nodularity, or atrophy of the right lobe, when present, can be useful signs of cirrhosis. Parenchymal echogenicity may be increased in diffuse disease, especially fatty infiltration, but may be difficult to evaluate, because no absolute echo amplitude standard exists; in sonography, there is nothing like the Hounsfield numbers (attenuation numbers) used in computed tomography (CT). Liver echogenicity is judged by comparison with adjacent organs, most often the kidney.

Sonographic Technique in Diffuse Liver Disease

It is difficult to image the entire liver, even in the best circumstances. Difficult areas include the superficial liver

above the costal margin, the left tip of the lateral segment of the left lobe, and the ventral subdiaphragmatic regions. The liver is best imaged with the patient in the supine and left lateral decubitus positions, starting with 3- to 7-MHz curved linear array transducers. A subcostal acoustic window should be used first, supplemented with intercostal scans. Small sector transducers should be used to image areas inaccessible to the larger curved linear transducers. The liver surface (usually the ventral left lobe) should be evaluated for nodularity with a near-field optimized 5- to 12-MHz linear array transducer or, in more obese patients, newer technology high-frequency curved linear transducers. It is easier to appreciate subtle nodularity during real-time examination or on video clips than on hard copy images. Surface characteristics are easier to appreciate when ascites is present. Angulation of the transducer from perpendicular to the liver surface may falsely simulate nodularity.

Routine color flow imaging is useful in patients with suspected liver disease. Optimal color flow and spectral Doppler sonography of the liver generally require relatively low-frequency (2- to 3-MHz) scanning and good acoustic access. Spectral Doppler sonography may be useful in specific clinical situations or to distinguish arterial from venous flow.

Bile duct obstruction must be excluded as a possible explanation for abnormal liver tests, because ductal obstruction can cause laboratory abnormalities, raising the suspicion of liver disease.

Hepatomegaly

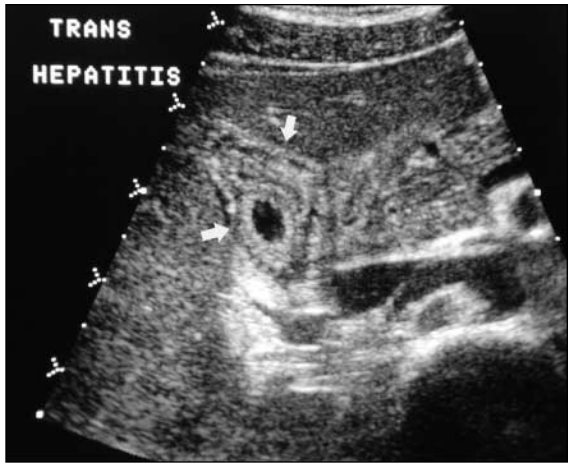
Hepatomegaly is difficult to diagnose objectively with sonography. The normal span of the adult liver is 15 to 17 cm. The most reliable measurement is probably the sagittal dimension from the dome to the tip of the right lobe, measured at the midclavicular line. If this exceeds 15.5 cm, the liver is probably enlarged. Hepatomegaly can be confidently diagnosed when the liver extends caudal to the right kidney and the left lobe is subjectively of normal size or larger. The use of this approach may be inaccurate in the occasional patient with a Riedel lobe. Three-dimensional (3D) techniques in CT, magnetic resonance imaging, and sonography ultimately promise to make accurate volumetric measurements feasible.

Hepatitis

There are no specific sonographic findings in acute hepatitis. The findings are quite variable because of many different etiologic factors that cause hepatic inflammation. The most common sonographic finding in hepatitis is probably hepatomegaly.[1] The so-called "starry night liver" pattern,[2,3] increased periportal echoes coupled with decreased parenchymal echogenicity, is not useful clinically. One series showed the starry night liver pattern in only 19 of 791 patients.[4] In the same study, there was no difference in sonographic findings between a control group without abnormalities and patients with acute viral hepatitis. Striking irregular gallbladder wall thickening is sometimes present in patients with acute hepatitis, especially hepatitis A (Fig. 1).[5] Direct inflammation and edema cause wall thickening, sometimes reaching 20 mm (normal, <3 mm). This finding alone, although nonspecific, can sometimes suggest the diagnosis of hepatitis A in the correct clinical situation.

Hepatomegaly and inhomogeneous patchy or diffuse increased echogenicity are common in chronic hepatitis and are related to the amount of fatty infiltration and fibrosis present. The liver surface is smooth, unless cirrhosis is also present. In chronic active hepatitis, often alcohol

Figure 1. Thick gallbladder wall in hepatitis A. This transverse sonogram of the gallbladder shows a markedly thickened gallbladder wall (arrows) in this patient with hepatitis A. The alternating of echogenic and hypoechoic layers is typical of an edematous wall in hepatitis A. This appearance alone is sufficient to suggest the diagnosis of hepatitis A in the correct clinical situation, although other conditions can cause edema of the wall of the gall bladder.



related, enlarged arteries are noted on color Doppler sonography (CDS) because of increased arterial flow. This may cause a "double-channel" sign on gray scale images that may be confused with biliary dilatation. Lymph nodes are sometimes detected in the hepatoduodenal ligament.[6]

Fatty Liver

Fatty infiltration of the liver is a manifestation of hepatocellular injury. The causes of injury are numerous (e.g., hepatitis, diabetes, and toxins), and often no cause is determined. Severe fatty infiltration often results in an enlarged liver with diffuse increased echogenicity (Fig. 2, A and B). Acoustic penetration may be decreased, resulting in indistinctness of blood vessels and the diaphragm. The liver surface is smooth. The normal echogenicity of the liver is determined by comparing the liver echogenicity with that of the cortex of the kidney. Hepatic echogenicity is usually equal to or greater than that of the renal cortex. Although subjective assessment of the renal cortex and liver parenchyma echogenicity is useful in severe fatty infiltration, this approach is error prone and insensitive. Although unproved, we prefer to use the relative echogenicity of the kidneys compared with the spleen and liver. The normal spleen is slightly more echogenic than the normal liver (Fig. 3). Therefore, if the difference in echogenicity between the liver and right kidney is greater than the difference between the spleen and left kidney, the liver parenchyma has abnormally increased echogenicity. This increased echogenicity is usually caused by fatty infiltration. This approach assumes, of course, that the echogenicity of the kidneys is equal bilaterally.

Fatty infiltration is often patchy or focal. Focal fat appears as an area of increased echogenicity. A less affected region ("spared" area) in a fatty liver appears as a conspicuous hypoechoic area (Fig. 4). Often spared areas are seen dorsal to the gallbladder. Other common locations include the region of the porta hepatis, near the falciform ligament, the dorsal left lobe, and the caudate lobe. Both focal fat and spared areas have a tendency to be pyramidal, with flame-shaped, tapered margins. Although both focal fatty infiltration and focal sparing can simulate a neoplasm,[7–9] an appreciation of the usual appearance and location generally suffices to avoid confusion. In fact, discovery of a typical spared area can suggest the diagnosis of fatty liver when no other findings are present. Geographic fatty infiltration typically has well-defined borders between areas of greater and lesser involvement; usually entire lobes or segments are affected in geographic fatty infiltra-

Figure 2. Fatty infiltration of the liver: comparison with normal spleen. **A**, Longitudinal sonogram of the right lobe of the liver and right kidney shows increased overall echogenicity of the liver. Note the slight attenuation of sound deep in the liver. **B**, The difference in echogenicity between the spleen and left renal cortex is less pronounced than that of the liver and right renal cortex (**A**). The normal spleen is slightly more echogenic than the normal liver (see Fig. 3). Therefore, when the difference in echogenicity between the liver and right kidney is greater than the difference between that of the spleen and left kidney, the liver parenchyma has abnormally increased echogenicity. This usually indicates fatty infiltration of the liver. LK indicates left kidney.

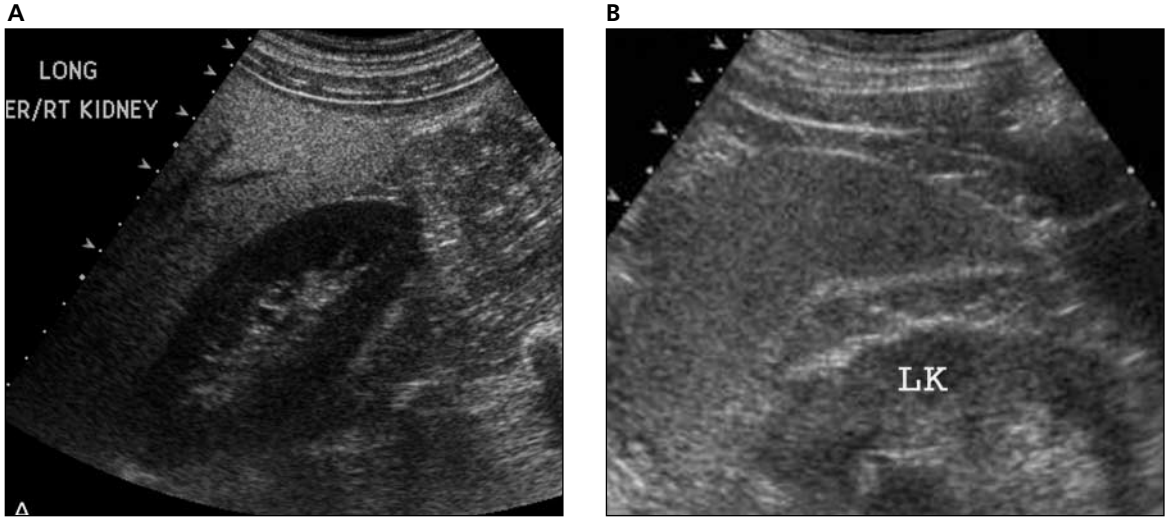
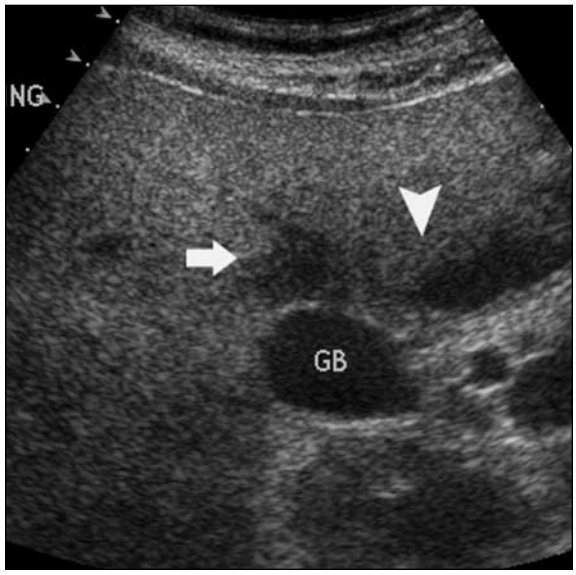


Figure 3. Normal liver and spleen echogenicity: long left lobe of the liver. A longitudinal sonogram through the left upper quadrant is shown. The unusual extension of the left lobe of the liver lateral to the spleen allows this direct comparison. The normal spleen is slightly more echogenic than the normal liver. Both are equal or greater in echogenicity compared with the normal renal cortex. The relative echogenicity of the liver and spleen can be determined by indirect comparison with the adjacent renal cortex (see Fig. 2). LK indicates left kidney.

Figure 4. Fatty liver: hypoechoic spared areas. This transverse sonogram of the liver shows 2 hypoechoic areas ventral to the gallbladder (arrow) and at the dorsal (posterior) portion of the lateral segment of the left lobe (arrowhead). Both of these areas are common locations for regions of relative sparing in a fatty liver. The lack of a mass effect and tapered margins are part of the usual appearance of sparing. GB indicates gallbladder.



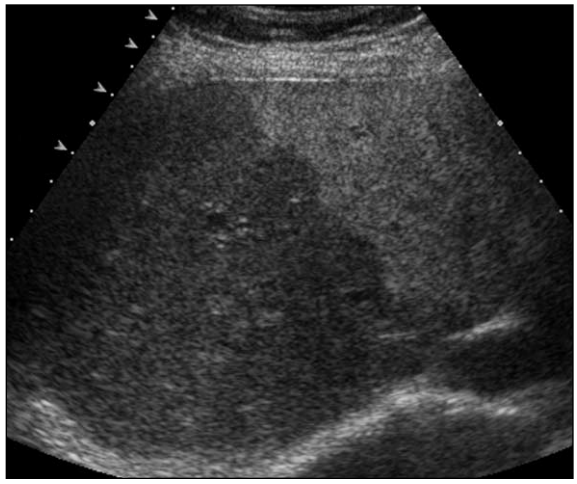
tion (Fig. 5). Occasionally, CT, magnetic resonance imaging, or a biopsy may be required to clarify the diagnosis.

Nonalcoholic steatohepatitis (NASH) is a condition in which severe fatty infiltration leads to hepatomegaly,[10,11] coupled with inflammation and fibrosis, characterized by elevated serum aspartate aminotransferase and alanine aminotransferase levels. Histologically, NASH is similar to alcoholic hepatitis but occurs in people who are not alcohol abusers. Most patients are obese women; there may be an association with diabetes, as well as hypercholesterolemia and hypertrigliceridemia. NASH may, on occasion, eventuate in cirrhosis. About 1% of patients undergoing liver transplants have a preoperative diagnosis of NASH. Sonographically, NASH is indistinguishable from other causes of severe fatty infiltration of the liver (Fig. 6).

Cirrhosis

Cirrhosis is the end stage of chronic hepatocyte injury, characterized by bridging fibrosis and regeneration. Sonographic findings of cirrhosis include changes in the shape of the liver, parenchymal inhomogeneity, and nodularity of the liver, notably at the surface. Intrahepatic vessels may be indistinct. Unfortunately, these signs are both insensitive and insufficiently specific for cirrhosis to be diagnosed reliably. Nevertheless, evaluation of the smoothness or nodularity of the

Figure 5. Geographic fatty infiltration: lobar distribution. This transverse image of the liver shows hyperechoic fatty infiltration of the left lobe of the liver, with a relatively hypoechoic spared right lobe. Note the well-demarcated geographic margin.



liver surface with a high-resolution linear array transducer is useful.[12–14] Surface nodularity may be the only sonographic sign of cirrhosis. Although some studies have not confirmed the usefulness of evaluating surface nodularity,[14] we think that the specificity for macronodular cirrhosis is good (Fig. 7), although micronodular cirrhosis is often missed. Three-dimensional imaging of the liver when it becomes more robust may allow better visualization of the liver surface (Fig. 8). The only other notable cause of surface nodularity is multiple subcapsular tumor nodules, usually from metastasis. Rarely, surface involution from treated metastasis or from liver necrosis with parenchymal collapse causes surface nodularity.

The caudate and left lobes tend to be relatively less affected by cirrhosis than the right lobe. This sometimes results in a small right lobe with left and caudate lobe hypertrophy, especially in hepatitis B. Ratios comparing the size or volume of the caudate lobe with that of the shrunken right lobe have been used to diagnose cirrhosis.[15,16] These ratios are not always useful, however; a large series revealed sensitivity of 43% and accuracy of 79% for the caudate-right lobe ratio.[17]

Color Doppler sonography may show portal vein flow reversal or portal collaterals, prompting the diagnosis of portal hypertension. Flow reversal or portal collaterals may be the only finding indicating severe liver disease in a patient with otherwise normal findings. Conspicuously enlarged and tortuous hepatic arteries are sometimes shown on CDS in cirrhotic livers (Fig. 9). This finding, similar to "corkscrew" arteries seen angiographically, probably occurs because of truncation of arteries from cirrhosis-related liver atrophy, coupled with the increased arterial flow[18] that occurs when portal venous flow decreases. These enlarged hepatic arteries usually have higher velocity (frequency shifts), usually with aliasing, compared with normal hepatic arteries. Similarly enlarged arteries may occur with portal thrombosis, portal vein flow reversal, and portosystemic shunts. Cirrhosis often causes narrowing of the hepatic veins with loss of the normal phasic waveform on duplex studies (Fig. 10).

Cirrhosis is the most common cause of portal hypertension. Finding portosystemic collaterals or portal vein flow reversal may prompt

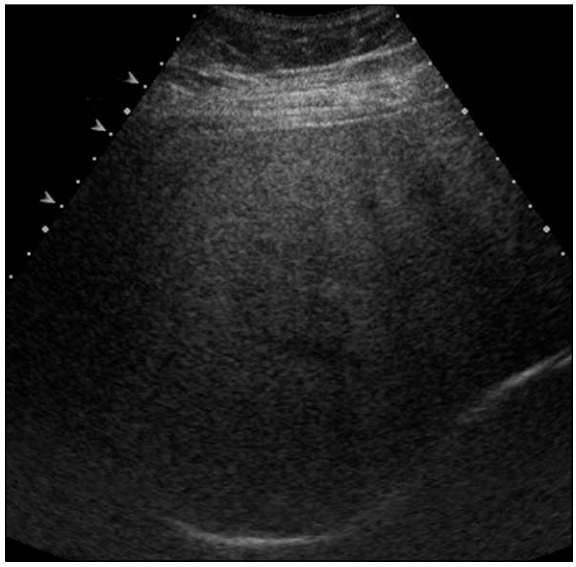
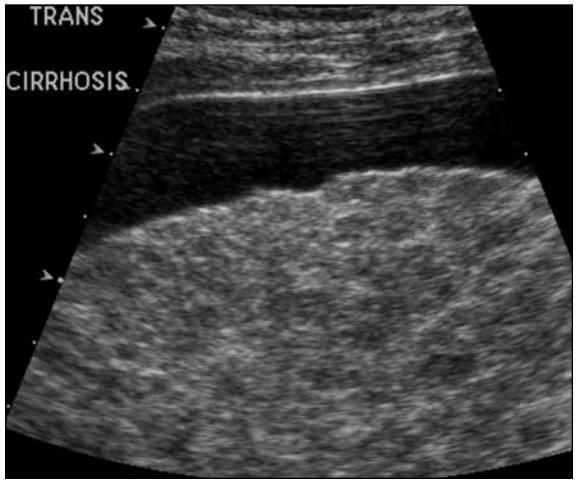


Figure 6. Fatty liver: NASH. This condition, which may result in cirrhosis, is indistinguishable from other causes of fatty liver. Note the attenuation of sound; hepatic vasculature is often poorly shown, as in this image.

Figure 7. Cirrhosis: nodular liver surface and liver nodules. The surface nodularity is shown clearly, facilitated by the presence of ascites. This image was obtained with a curved linear abdominal transducer operating at 6 MHz. The high-resolution longitudinal image of the surface of the liver shows evidence of an irregular surface. This is a common finding in liver cirrhosis. Also note the abnormal coarse, nodular texture of the liver.



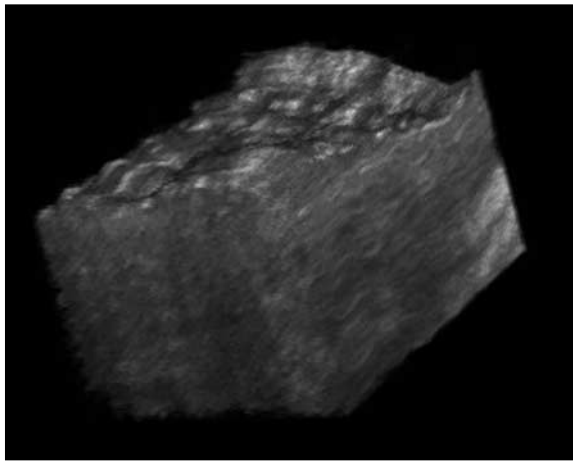
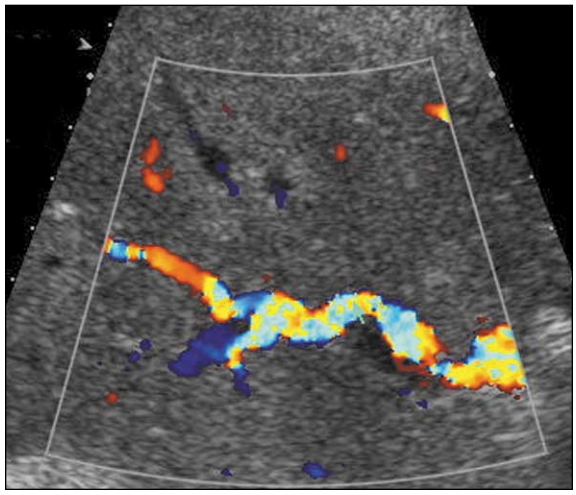


Figure 8. Three-dimensional sonogram of the liver surface. The nodular changes caused by cirrhosis are clearly shown in this 3D sonographic image. The liver surface is shown well because of the high acoustic difference between ascites and the liver surface. When ascites is not present, 3D visualization of the liver surface is difficult.

diagnosis of unsuspected portal hypertension and cirrhosis or may reinforce it when suspected. Sonographic evaluation of portal hypertension includes evaluation of the portal venous system and a search for portosystemic collaterals. The normal main portal vein measures slightly more than 1 cm in diameter. It has been suggested that a portal vein diameter of more than 13 mm indicates portal hypertension but is not always useful because of variation in portal vein size related to respiration and changes in patient position.[19] Although portal vein size is not useful in diagnosing portal hypertension, a lack of respiratory variation (an increase during inspiration and a decrease during expiration) in size may be useful in diagnosing portal hypertension.[18] Normally, blood flows toward the liver (hepatopetal) in portal veins. Reversed (hepatofugal) flow, although often associated with collaterals,[18] may be the sole indication of portal hypertension. Other portal flow abnormalities include bidirectional flow and, rarely, nearly static blood flow. Sonographic contrast agents can be useful in aiding the detection of flow in the main portal vein and other hepatic vessels. The most common collaterals are left gastric (coronary) and paraumbilical (recanalized umbilical) veins (Fig. 11). Left gastric vein collaterals, although by far the most frequent portosystemic collaterals, are often difficult to image because of their deep location in the lesser omentum. Paraumbilical veins are easier to image because they are superficial. They arise from the ventral tip of the left portal vein and usually flow caudally through the ligamentum teres, where they communicate with superficial collaterals. Other types of portal collaterals occur, including retroperitoneal, splenorenal, splenoretroperitoneal, short gastric, and omental. Some collaterals cannot be detected because of overlying bowel gas.

Figure 9. Corkscrew hepatic artery: cirrhosis. Enlarged and tortuous hepatic arteries may be shown on color Doppler imaging in cirrhosis. This occurs because decreased portal flow results in increased arterial flow. Shrinkage of liver tissue foreshortens the artery, resulting in the corkscrew appearance.



Color Doppler sonography can quickly show abnormal flow reversal, sometimes the only sign of portal hypertension. It often shows collaterals invisible on gray scale images. One must be aware that transient portal flow reversal may also occur in some clinical conditions not related to liver disease; examples include tricuspid regurgitation and heart failure.

Therapeutic Portosystemic Shunts

Color Doppler sonography often shows surgical shunts,[20] even when they are inapparent on preliminary gray scale images. Failure to detect color Doppler patency within the anastomosis is the only reliable sign of surgical shunt thrombosis. Secondary signs of shunt thrombosis, such as hepatopetal intrahepatic portal flow,[20] may be misleading.[21]

Transjugular intrahepatic portosystemic stent shunts (TIPSS) can be evaluated with Doppler

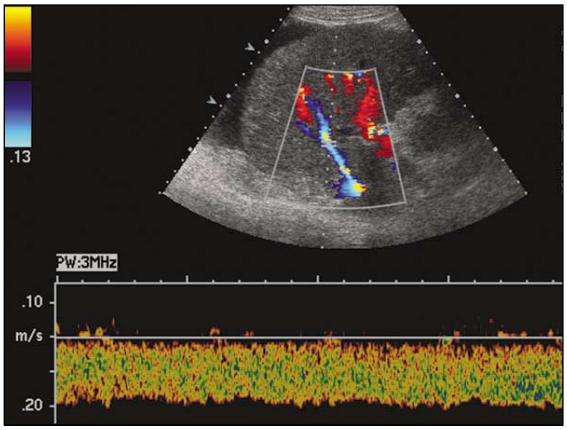
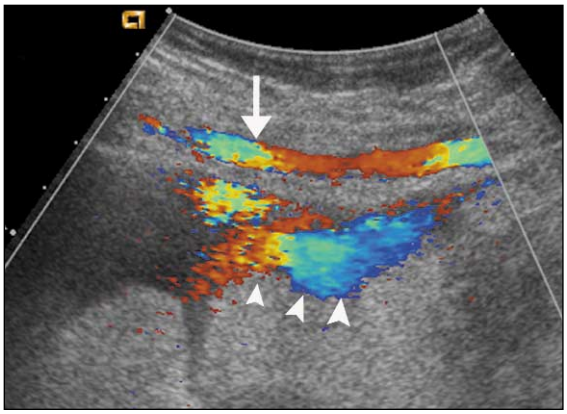


Figure 10. Narrowed hepatic vein in cirrhosis. The morphologic distortion caused by cirrhosis often causes compression of hepatic veins, which results in loss of the normal multiphasic waveform. The flattened waveform that results is similar in appearance to that seen in portal veins.

sonography. Preprocedure evaluation and post-TIPSS follow-up are useful. Intraprocedure CDS guidance is occasionally helpful. Low-frequency Doppler sonography (e.g., 2 MHz) is generally best for imaging TIPSS. Many factors influence stent velocity; most importantly is respiration, which can lead to a considerable decrease in velocity within the stent. A considerable decrease in flow velocity during inspiration compared with quiet respiration has been documented.[22] Transjugular intrahepatic portosystemic stent shunt stenosis may result in increased or decreased flow velocity within the stent (Fig. 12). We use a range of 0.9 to 2.0 m/s in the mid and distal (hepatic vein side) portions of the stent as normal. We do not use the velocity in the proximal portion of the stent (portal vein side) or any of the many other reported flow parameters. These intrastent velocity parameters are fairly sensitive for stent dysfunction, but occasional false-positive findings will result in venography in some patients with normal stents. In the normally functioning stent, blood flow in the native portal veins is almost always toward the stent; thus flow in the left and right branches of the portal vein is hepatofugal. A fairly specific but insensitive parameter for stent dysfunction is finding flow away from the stent (hepatopetal) in the branches of the native portal vein. Occasionally, poor acoustic access, a poor Doppler angle, and other technical factors may result in nonvisualization of flow, even when the shunt is patent.

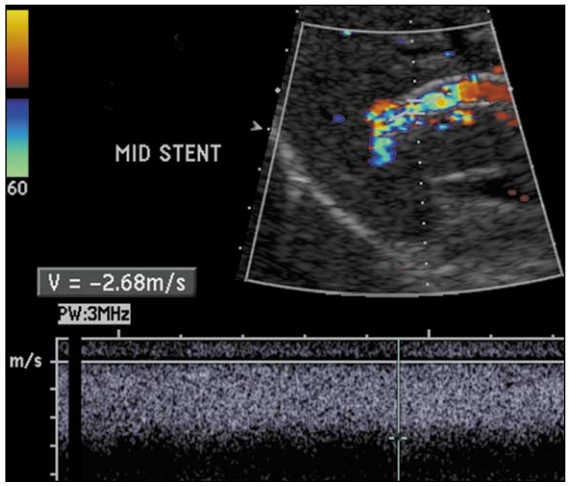
Figure 11. Recanalized paraumbilical vein. This color Doppler image shows an enlarged paraumbilical vein (arrowheads). This portal collateral is diagnostic for severe portal hypertension. In this case, the paraumbilical vein communicates with the internal mammary vein (arrow), which will drain into the subclavian vein.



Venous Thrombosis

Complete and partial venous thromboses are easily diagnosed with color Doppler imaging. Intraluminal echoes on gray scale images support but do not definitively establish the diagnosis of a clot. When hypoechoic or anechoic, a clot may be virtually invisible without color Doppler imaging. Color Doppler sonography highlights a clot by displaying flow around it (Fig. 13). An anechoic thrombus is rare in neoplastic invasion, but it is common in bland portal vein clots, espe-

Figure 12. High velocity in dysfunctional TIPSS. The midstent velocity is 2.68 m/s. Velocity greater than 2 or less than 0.9 m/s in the mid or distal (hepatic vein–end) stent usually means stent dysfunction is present.



cially when thrombosis is acute. Color Doppler sonography highlights the clot by displaying flow around the clot and in adjacent patent vessels.[18] Portal venous thrombosis can be accurately diagnosed with CDS when no intravascular color-coded flow is detected, an adequate Doppler scan angle and acoustic access are obtained, and flow is shown in other vessels at a comparable depth.

Portal venous thrombosis can result from neoplastic invasion, septic portal thrombosis (pylephlebitis), pancreatitis, portal hypertension, trauma, and endoscopic esophageal sclerotherapy. We think that thrombosis in patients with portal hypertension[23] (Fig. 13) is considerably more common than the 1% previously reported.[24] Color Doppler sonography is unrivaled in showing partially occluded vessels. Small residual flow channels are automatically displayed in color. Clot resolution or progression can be documented on serial examinations. Serial CDS can document resolution or progression of a clot. Contrast agents may be useful in difficult cases.

Cavernous transformation of the portal vein, resulting from a main portal vein clot, consists of prominent hepatopetal portal collaterals. Associated findings may include gallbladder varices and lesser omentum collaterals. In acute portal vein thrombosis, CDS may reveal small acute hepatopetal portal collaterals, invisible on gray scale sonography. This may be a precursor to cavernous transformation of the portal vein.

Hepatocellular Carcinoma

Sonography plays an important role in the evaluation and follow-up of patients with known liver cirrhosis. Focal fatty infiltration, hepatocellular carcinoma (HCC), regenerating nodules (adenomatous hyperplastic nodule), or other focal lesions may occur in a cirrhotic liver. Because of the increased risk of developing cancer, a liver mass in a cirrhotic patient strongly suggests the possibility of an HCC.

Hepatocellular carcinoma is the most common primary liver cancer. In the United States, 85% of HCCs occur in patients with cirrhosis or precirrhotic conditions. Advanced HCC is almost always multifocal, making it difficult to distinguish from metastatic disease. Small HCCs (<5 cm) are often (75%) hypoechoic.[25] As HCCs grow, they tend to develop hypoechoic peripheral rims.[25] With further progression, lesions become more numerous and heterogeneous (Fig. 14). Fatty metamorphosis may cause increased echogenicity and confusion with hemangioma.[26] Likewise, echogenic nodules are fairly common in multifocal HCC. Hepatic or portal venous invasion should suggest the diagnosis of HCC (Fig. 15), although other liver tumors may invade veins.[27] Rarely, HCC invades the bile duct. About three fourths of HCCs have identifiable internal color flow versus one third of metastases.

Figure 13. Bland clot: cirrhosis. Anechoic clot in the left portal vein. Note the arteries (arrows) on either side of the clotted portal vein clot. A bland thrombus is often anechoic or hypoechoic, whereas a tumor thrombus is usually echogenic and enlarges the vein.

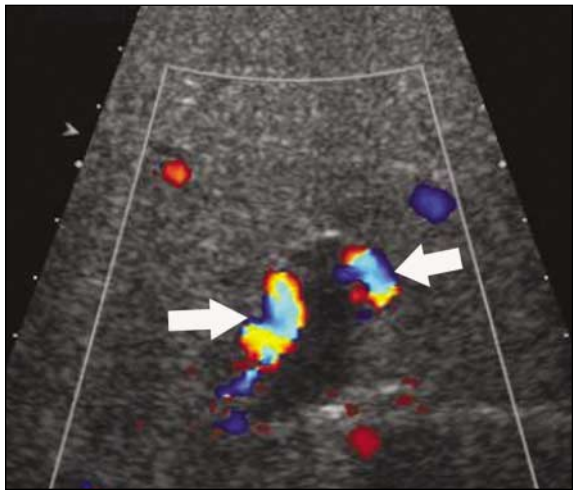


Figure 14. Multifocal HCC. This transverse sonogram of the liver shows multiple masses consistent with a multifocal HCC. The tumor has invaded the inferior vena cava (arrow). The masses range from echogenic to hypoechoic. Hepatocellular carcinomas that arise in patients with diffuse liver disease are usually multifocal at the time of initial appearance.

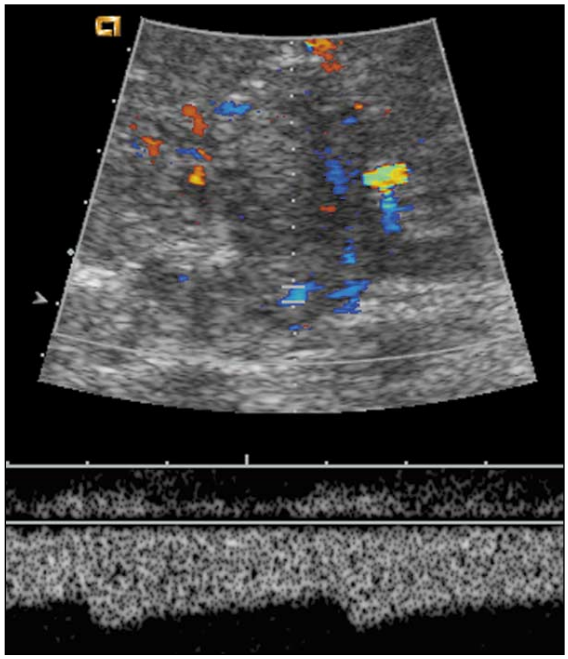


Figure 15. Flow in HCC tumor thrombus. A color Doppler sonogram of the left portal vein is shown. The normally anechoic vein is filled with echogenic tumor thrombosis. The spectral tracing from this vessel shows an arterial flow signal essentially diagnostic for a tumor thrombus.

Fibrolamellar HCC, which accounts for 2% of HCC cases overall but 25% to 50% of HCC cases in young adults, is typically a single well-circumscribed lesion in an otherwise normal liver. Other features include a "central scar" and calcification (25%), both of which also occur in other subtypes of HCC and lesions of other histologic types.

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