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CAQ CORNER

# Abdominal Imaging and Intervention in Liver Transplantation

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**Diagnostic imaging and interventional radiology play key roles in the evaluation and management of patients who are being evaluated for potential liver transplantation (LTX) and of those who have received a transplanted liver. Technical advances in imaging equipment and technique allow more accurate assessment and often obviate unnecessary or nontherapeutic surgery or invasive techniques such as catheter angiography.** *Liver Transpl 12: 184-193, 2006.* © 2006 AASLD.

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A prior review article in this series[1] provides an excellent overview of the surgical principles and techniques involved in liver transplantation (LTX), as well as the clinical presentation and management of postoperative complications. The purpose of our review is to discuss the role of imaging and intervention in potential LTX recipients (and donors) and in posttransplantation patients.

## EVALUATION OF THE POTENTIAL TRANSPLANT RECIPIENT

The goals of imaging patients with liver failure are to help to evaluate the severity of cirrhosis and portal hypertension, to identify conditions that may complicate or preclude LTX, and to identify and stage tumor within the cirrhotic liver or extrahepatic malignancy.

The major imaging tools used in these tasks are ultrasonography, computed tomography (CT), magnetic resonance imaging (MRI), and angiography. Continuous and dramatic improvements in these modalities over the past few years, especially in CT and MRI, have expanded their role and improved their performance substantially. As might be expected, variations in the availability of advanced equipment and personal expertise will be reflected by variable enthusiasm and employment of one technique over another. By virtue of its availability and ability to address most of the important issues cited above, CT is the dominant tool in evaluating patients with advanced liver disease. Sonography, MRI, and angiography maintain important screening and "problem solving" roles.

### Cirrhosis and Portal Hypertension

The extent of cirrhosis and portal hypertension are well evaluated by CT, providing important information to supplement clinical and biochemical measures, such as the Child-Turcotte-Pugh classification. Depending on the etiology of cirrhosis, some variations in hepatic morphologic changes can be expected and identified on CT. For example, primary sclerosing cholangitis often leads to hepatic damage and fibrosis that is more prevalent in peripheral segments of the liver, with sparing or even hypertrophy of the central and caudate segments. This, along with the presence of irregular strictures of the biliary tree and prominent porta hepatis lymphadenopathy, often allows for a confident CT-based diagnosis of cirrhosis due to primary sclerosing cholangitis.[2] In contrast, primary biliary cirrhosis is characterized by hepatic enlargement (until end-stage disease has developed), even more prominent lymphadenopathy, and a lace-like pattern of hepatic fibrosis that is most apparent on noncontrast CT imaging[3] (Fig. 1).

While distinguishing among various etiologies of cir-

**Abbreviations:** CT, computed tomography; MR, magnetic resonance; MRI, magnetic resonance imaging; HCC, hepatocellular carcinoma; LTX, liver transplantation; PTLD, posttransplantation lymphoproliferative disorder; HA, hepatic artery.

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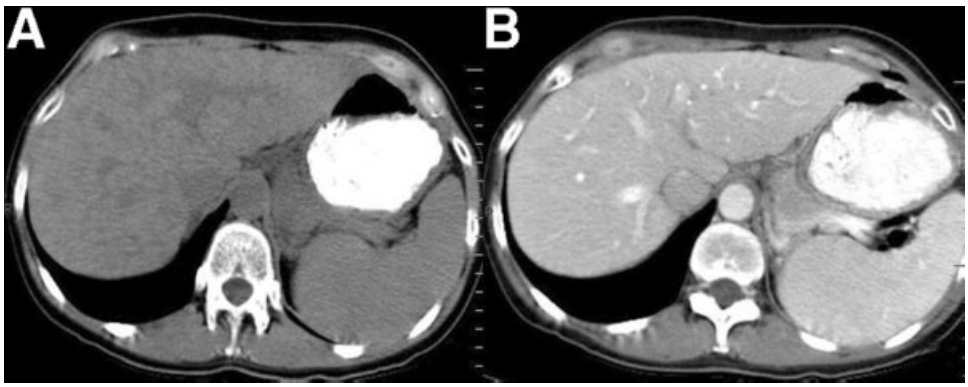


**Figure 1. (A) Primary biliary cirrhosis: Noncontrast CT image. Note the lace-like low density pattern of fibrosis throughout the liver, corresponding to fibrosis. (B) Primary biliary cirrhosis: Portal venous phase of enhancement on CT. The lace-like pattern is no longer apparent.**

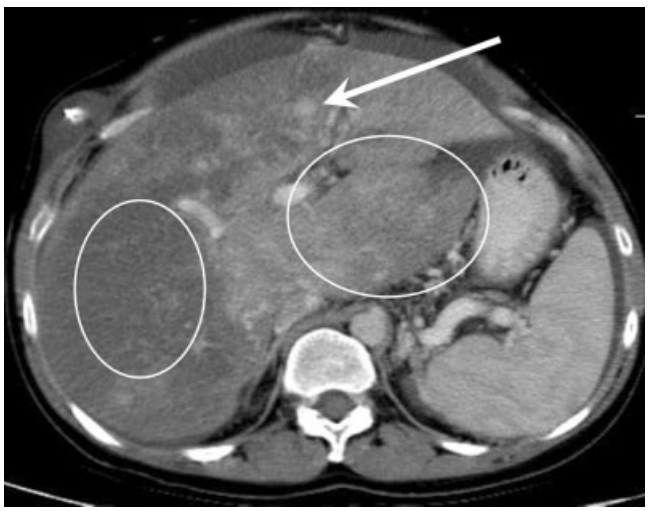

**Figure 2. Budd Chiari Syndrome: Early portal venous phase of enhancement on CT, demonstrating hypervascular, large, regenerative nodules (arrow), hepatocellular necrosis (oval), and caudate hypertrophy (circle).**

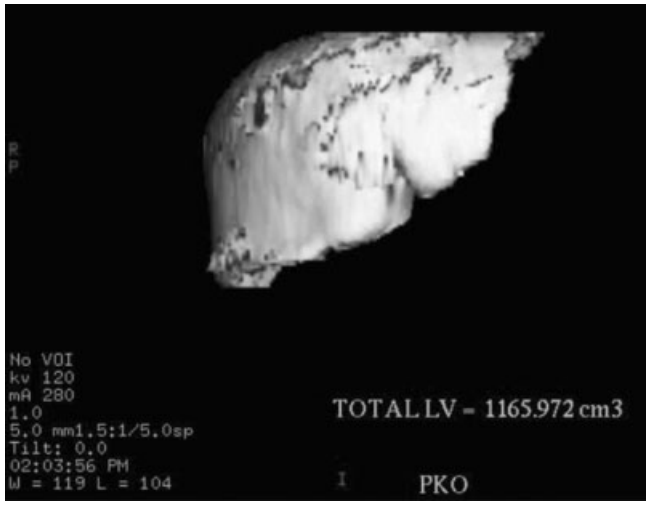


**Figure 3. Semiautomated, volume-rendered image obtained following routine CT scanning.**

rhosis is not usually a primary role for imaging, important exceptions do occur. The morphologic changes of cirrhosis may be simulated or caused by a variety of other processes that may first be identified on imaging. Examples include liver damage due to passive hepatic congestion, which may be identified with confidence by the criteria of cardiac chamber enlargement, heterogeneous enhancement of the liver and dilated hepatic veins. Conversely, Budd-Chiari syndrome is characterized by occlusion or narrowing of the hepatic veins, hypertrophy of the caudate and central right lobe, large regenerative nodules that are hypervascular on arterial phase imaging, and peripheral hepatic atrophy and fibrosis (Fig. 2). Various infiltrative processes, including sarcoidosis and metastatic breast carcinoma, may result in liver damage that closely simulates, or even results in, cirrhosis. Assessment of the hepatic and extrahepatic manifestations of these diseases often facilitates accurate diagnosis.

Regardless of the etiology of cirrhosis, the end-stage liver is usually characterized by volume loss and signs of portal hypertension. CT scanning, along with simple software programs, allows for accurate, semiautomated calculation of the volumes of the liver and its major segments, which is helpful in pre-LTX planning[4] (Fig. 3).

Signs of portal hypertension, including ascites, portosystemic collaterals (varices) and splenomegaly, are easily detected and quantitated. The presence of spontaneous (or surgically-created) shunts such as splenorenal venous is evident on CT.

One major role of pre-LTX imaging is to identify vascular anomalies or pathology that may complicate or preclude transplantation. Experienced transplant surgeons have developed techniques to address many of these vascular challenges, but preoperative imaging identification makes their task much easier. Congenital variations in hepatic arterial supply to the cirrhotic liver, for instance, are very common. Patients with anomalous arterial anatomy may not have a large enough common hepatic artery for inflow using an end-to-end arterial anastomosis. Newer "multislice" CT

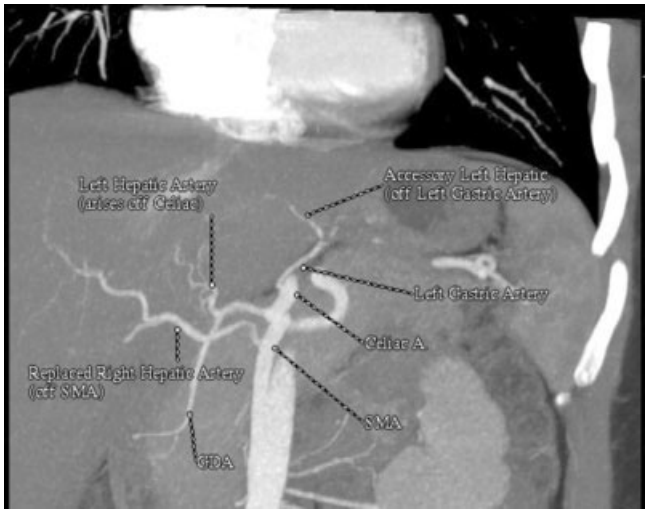


**Figure 4. Multiplanar reformat obtained from a CT arteriogram of a donor displaying several normal variants in the same patient. The right hepatic artery (RHA) arises off of the superior mesenteric artery rather than off the celiac artery. There is an accessory left hepatic artery (LHA) that arises off of the left gastric artery. The remainder of the left hepatic arterial supply arises from the common hepatic artery. GDA, gastroduodenal artery; SMA, superior mesenteric artery.**

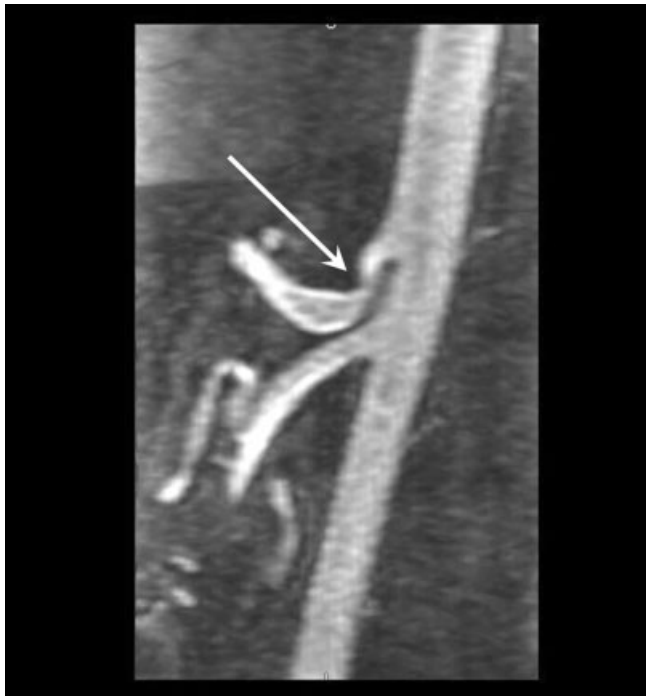

**Figure 5. Sagittal reformatted image from an MRI arteriogram depicting celiac arterial narrowing (arrow) secondary to median arcuate ligament syndrome with poststenotic dilation.**

scanners allow precise depiction of the vascular anatomy of the liver with only an intravenous injection of contrast material and a computer workstation reconfiguration of the routine transverse CT "slices" into any combination of multiplanar or "3-dimensional" images that may be desired (Fig. 4).

Patients with celiac axis stenosis or compression by a median arcuate ligament (Fig. 5) must and can be identified by CT angiography so that appropriate steps may be taken before or during surgery, such as harvesting and insertion of a donor iliac artery conduit or releasing the median arcuate ligament.

Similarly, pre-LTX recognition of portal vein thrombosis, fibrosis, or calcification is critical. Portal venous thrombus (bland) does not preclude transplant. Depending on the extent of the venous disease, thrombectomy, interposition jump venous graft or even cavoportal transposition may be required, or LTX may not remain a therapeutic option. Increasing clot burden increases the surgical risks, particularly if there is involvement of splanchnic veins.[5]

## Detection of Malignancy

The most challenging and controversial aspect of pre-LTX imaging is the detection and staging of hepatic malignancy. The cirrhotic liver usually harbors numerous focal lesions, varying from benign (cysts, hemangiomas) to hyperplastic (focal nodular hyperplasia, regenerating nodules), to dysplastic and frankly malignant. Distinguishing among these requires optimal imaging, often requiring multiple modalities, and considerable expertise, and despite that an accurate diagnosis may not be possible in some cases.

Cysts and hemangiomas are very common in the general population, affecting at least 20% of individuals. In early cirrhosis, these lesions can usually be identified with confidence due to their characteristic appearance on imaging. In more advanced cirrhosis, these "soft" lesions become compressed and obliterated by the surrounding fibrotic pathology and are difficult to detect or characterize.

## Regenerating Nodules

While all patients with cirrhosis have micronodular or macronodular regenerative nodules, most of these are not detectable on imaging due to their small size or insufficient difference from hepatic parenchyma in terms of echogenicity (sonography), density (CT), or intensity (MR). Some regenerating nodules contain excess iron (siderotic nodules), rendering them detectable on CT and MR. Unenhanced CT depicts siderotic nodules as hyperdense spherical lesions, usually 0.5 to 1.5 cm in diameter. Siderotic and other cirrhotic nodules usually show similar enhancement patterns as the remaining liver, and are less apparent on contrast-enhanced images. The excess iron in siderotic nodules results in characteristic MRI features, including decreased signal intensity on T2-weighted pulse sequences. They do not enhance appreciably following contrast administration[6] and may exhibit visible "blooming" effect (magnetic susceptibility) on gradient echo pulse sequences depending on the amount of iron within them (Fig. 6).

## Dysplastic Nodules

Dysplastic nodules are divided into low- and high-grade types and are considered a precursor to development of

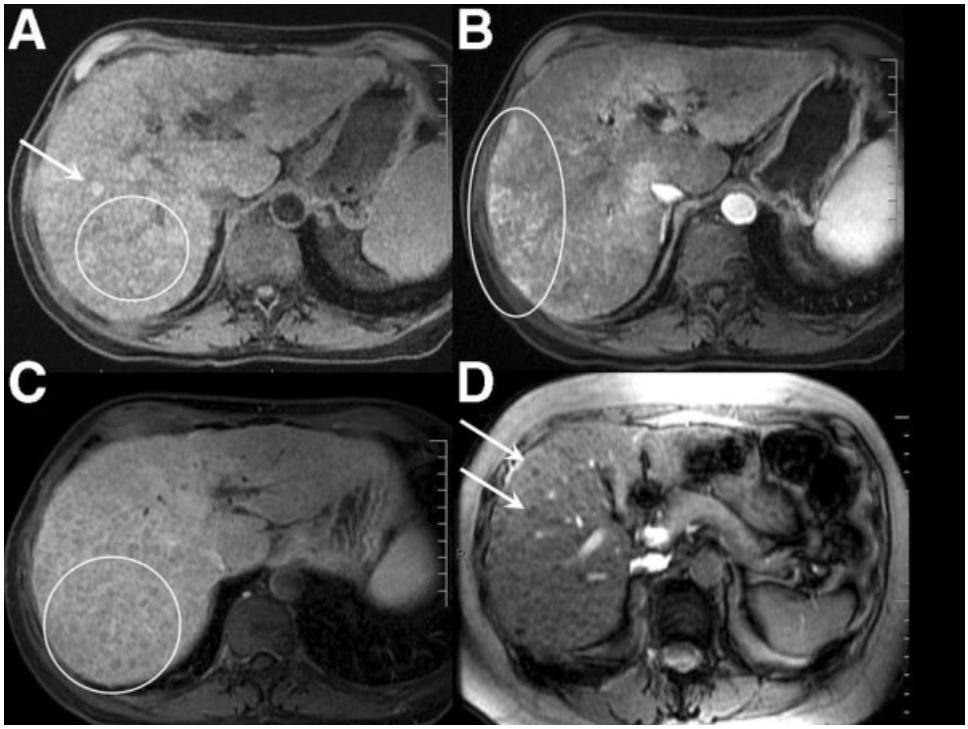


**Figure 6. Regenerative nodules. (A) Unenhanced T1-weighted (T1W) MR image reveals multiple, small (<2 cm) nodules that are hyperintense to the background liver. This depicts a larger nodule (arrow) and smaller nodules (circle). (B) T1W image in the arterial phase following administration of gadopentate dimeglumine (Gd-DTPA). Notice the nodules do not enhance to the same degree as the background liver. Peripherally, intrahepatic arterio-portal shunting is present (circle). (C) T1W image on "portal venous" phase, post-Gd-DTPA. The nodules enhance less than the background liver making them hypointense and darker than the background liver (circle). (D) Siderotic nodule on a gradient recalled echo image that has been optimized to emphasize magnetic susceptibility. The increased iron content in siderotic nodules makes them appear dark as a result of the magnetic susceptibility (arrows).**

hepatocellular carcinoma (HCC). As a general rule, lesions at the benign end of the nodular spectrum tend to maintain a portal venous blood supply, while high-grade dysplastic and malignant lesions lose portal venous supply and attain a predominant arterial blood supply. This principle underlies CT and MR imaging protocols, which must include the acquisition of non-enhanced, arterial (portal inflow), and portal venous (hepatic parenchymal) phases of imaging.

Existing imaging techniques are neither sensitive nor specific in identification of dysplastic nodules. The performance of CT exceeds that of sonography, and a diagnosis of dysplastic nodule may be suggested by a size greater than 2 cm and slight arterial phase hyperenhancement. MRI is the best technique currently available, but reports indicate that only 5 to 15% of dysplastic nodules are detected accurately.[7] The imaging characteristics on MRI are variable, but generally dysplastic nodules are bright on T1-weighted images, dark on T2-weighted images, and enhance variably on arterial phase.[11] It can be difficult to distinguish siderotic regenerative nodules from siderotic dysplastic nodules.

MR and CT may depict a "nodule-within-nodule" pattern in a focal lesion, indicating a focus of HCC within a dysplastic nodule. The focus of HCC might demonstrate enhancement on the arterial phase images, and high-signal intensity on T2-weighted MR images.

The rate of development of HCC depends on many factors including the etiology and severity of the cirrhosis, and the age and gender of the patient. A 50-year-old woman with primary biliary cirrhosis, for instance, is at a much lower risk than a 55-year-old man with chronic hepatitis B or C.[8] The method and frequency of surveillance techniques should take these factors into consideration.

In general, patients with cirrhosis are monitored by a combination of clinical and biochemical measures of liver function, serum tumor markers such as alpha fetoprotein, and imaging.

Owing to its lack of ionizing radiation, availability, and low cost, sonography is used liberally and frequently in surveillance for HCC, and is often repeated every few months in practices around the world. Greater success has been reported by investigators in Asia, though CT, MRI, and angiography are all utilized frequently in this same population, in which HCC is endemic. In North America and Europe, sonography rarely depicts HCC at a "curable" stage. This may be due in part, to the greater prevalence of larger patients and alcoholic liver disease, which compromise the quality of sonography. In all major centers, focal lesions detected by sonography are further evaluated by CT, MRI, or image-guided biopsy. With the caveats noted

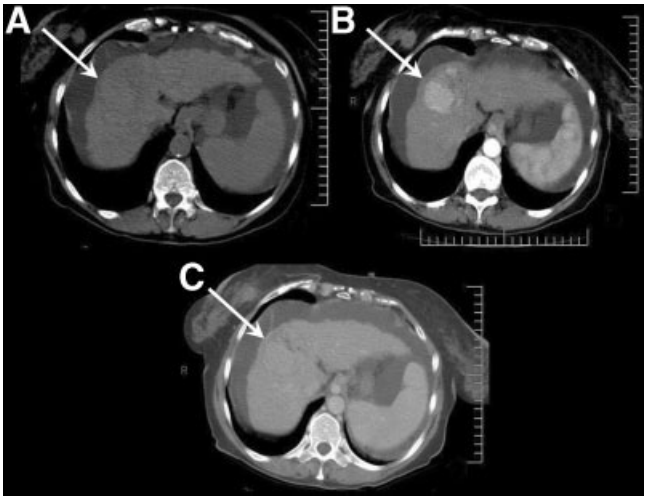


**Figure 7. HCC. (A) Noncontrast CT with the segment 8/4A lesion not particularly apparent (arrow). (B) Arterial phase CT image: the tumor (arrow) is very apparent and is hypervascular relative to the background, cirrhotic liver. (C) Portal venous phase CT image: the tumor becomes less conspicuous compared to the background (arrow).**

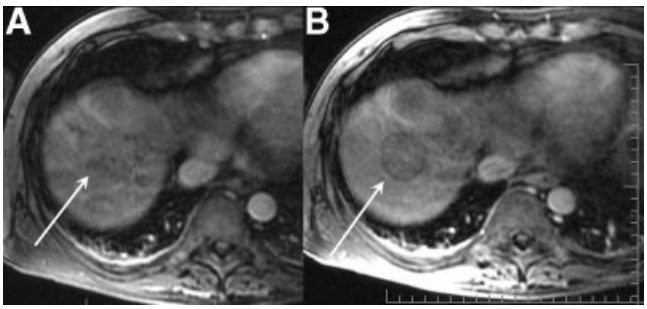


**Figure 8. HCC. (A) Arterial phase T1-weighted (T1W) image. On this phase, the tumor is difficult to discern. (B) Portal venous phase T1W image. The hypovascular, moderately differentiated HCC becomes more conspicuous on portal venous phase of imaging (arrow).**

above, most patients with established cirrhosis are evaluated by CT (or MRI) at intervals of 6-12 months.

CT and MRI are well-suited to detecting and characterizing focal masses, including HCC, that are greater than 2 cm in diameter. On CT, the usual appearance of HCC is a mass that is hypodense to liver on the nonenhanced images, transiently and heterogeneously hyperdense on arterial phase, and is iso- to hypodense on portal venous and delayed phase imaging (Fig. 7). The degree of tumor differentiation affects the imaging appearance of HCC. A well-differentiated tumor is more often hypovascular and encapsulated (Fig. 8), and would be detected best on portal venous phase imaging, while arterial phase imaging is superior for detection of the more common moderately or poorly differentiated HCC.

Dynamic MRI reveals the same hemodynamic characteristics of HCC. HCC has variable signal intensity on T1-weighted images, while being reliably hyperintense on T2-weighted sequences. Variations in signal intensity may result from focal necrosis, fat, calcification, or encapsulation, but this imaging spectrum of HCC should be familiar to experienced abdominal imagers.

The use of hepatic targeted MRI contrast agents has not proved as useful in detecting HCC as was anticipated. Well-differentiated tumors may show uptake of hepatocyte-specific agents such as mangafodipir, while agents designed to maximally enhance the contrast between liver and tumor (such as ferumoxide agents) tend to show heterogeneous and variable uptake within the cirrhotic liver.

Accurate staging of HCC has become an important goal of imaging as more effective therapies become available, especially for small tumors detected in an effective surveillance program. Small tumors may be amenable to resection or ablation, the latter using image-guided alcohol injection or radiofrequency probes. Multifocal or large tumors, or those with vascular invasion are not candidates for resection, but may have improved survival with chemoembolization or transcatheter delivery of radioactive microspheres.

It should be noted that the diagnosis of HCC and the size of lesions may result in false negatives (no tumor on imaging) or discrepancy in the size or number of lesions and therefore the staging of patients who are imaged more than two months prior to transplant.[8] Ideally, patients should be imaged within this time frame to increase sensitivity for detection of HCC and to increase accuracy in staging.

CT and MRI are quite accurate in depicting large HCC lesions and complications such as portal or hepatic venous invasion or biliary ductal obstruction. Venous tumor thrombi, for example, are detected as vessel expansion, enhancing tumor thrombi, and contiguity with a parenchymal mass.[9]

Further emphasizing the need for accurate staging of HCC is the recent recognition that patients with limited tumor respond well to liver transplantation. A patient with a single tumor less than 5 cm in diameter, or several lesions each less than 3 cm, and no evidence of venous invasion or extrahepatic tumor has an excellent prognosis for tumor-free survival following LTX.[10]

While CT and MRI detect large tumors accurately, they fare less well for tumors less than 2 cm in diameter. Using the most rigorous and "worst case" analysis, which is to compare the results of pre-LTX imaging with the pathological findings in the explanted liver on a lesion-by-lesion basis, similar disappointing results have been reported by several teams of investigators, including our own.[11] CT and MRI detect about 35 to 50% of all HCC lesions and only 50 to 65% of patients with HCC found at the time of transplantation.

## EVALUATION OF THE POTENTIAL LIVER DONOR

The goals of imaging the potential living liver donor are to evaluate the liver parenchyma, provide data for split-liver volume assessment, and to depict the intra- and extrahepatic biliary and vascular anatomy. Both CT[11] and MRI are well-suited to these tasks, although de-

tailed depiction of donor biliary anatomy remains a challenge.

The presence of unsuspected liver disease is well evaluated by CT and MR. Steatosis is especially important to detect and quantitate, as transplanting half of a liver damaged by steatosis endangers both the recipient and the donor.

With 16- and 64-channel multislice CT scanning, it is now possible to provide multiplanar and 3-dimensional images that display the hepatic arterial and venous anatomy in exquisite detail. Common variants, including hepatic arterial anomalies, trifurcation of the portal veins, or large accessory or anomalous hepatic veins may preclude the use of a potential donor liver, or may mandate alternate surgical approaches.

Noninvasive imaging of the biliary tree of a potential living donor remains challenging. MR cholangiography has been reported to provide excellent anatomic definition of the biliary tree, but experience with operative correlation is limited to small case series.[12] Moreover, the best results have been achieved by utilizing a hepatobiliary MR contrast agent that is not available for general use in North America.

Similarly, one group of investigators has reported excellent results[13,14] with CT cholangiography in which 3-dimensional depictions of the biliary tree are constructed in a manner similar to those methods used for CT angiography and is done following the administration of a hepatobiliary contrast agent. As with MR cholangiography, this contrast agent is not in widespread use and the safety and applicability of this technique awaits further validation.

## POSTOPERATIVE IMAGING OF THE LTX RECIPIENT

Because of the debilitated condition of most patients awaiting LTX, the complex nature of the LTX procedure itself, and the effects of postoperative immunosuppressive therapy, many patients develop postoperative complications. Clinical signs and symptoms are often nonspecific, leaving a major role for imaging in this setting.

### Vascular Complications

Vascular complications develop in some 8% of LTX cases and should be considered in patients with evidence of graft failure, biliary strictures and leaks, gastrointestinal bleeding or septicemia.[15,16] Hepatic artery stenosis or thrombosis is the most common and significant vascular complication and accounts for approximately 60% of LTX vascular complications.[17] Clinical presentation varies from delayed, intermittent episodes of septicemia or abnormal liver function to fulminant hepatic failure.

Doppler sonography is able to detect up to 92% of cases of arterial thrombosis,[18] demonstrating absent arterial flow, or markedly diminished flow in the presence of arterial collateral development. In cases of arterial stenosis, which usually occurs at the anastomosis, sonography demonstrates focally accelerated velocity (greater than 2 m/second) and turbulent flow at and beyond the anastomosis. A tardus-parvus waveform is seen downstream from the stenosis with a sensitivity and specificity of about 75%, and consists of a low systolic acceleration time (greater than 0.08 seconds) and a low resistive index, $<0.5$ (Fig. 9).[19]

Hepatic artery (HA) stenosis may progress to thrombosis and either may be manifested by signs of biliary ischemia, with bile duct abnormalities reported in more than 60% of patients with HA stenosis.[20] In any patient with a focal hepatic parenchymal abnormality and graft dysfunction in the early post-LTX period, HA thrombosis or stenosis should be suspected.

Definitive diagnosis of HA stenosis requires CT, MR, or catheter angiography. CT angiography and MR angiography reliably demonstrate HA stenosis and other arterial lesions, such as pseudoaneurysms. Depending on the degree of arterial stenosis and graft injury, the lesions may be amenable to angiographic catheter balloon dilatation or stenting. Surgical revision of the arterial graft or even retransplantation is required in more advanced injury.

### Venous Complications

Venous complications are less common after LTX, occurring in fewer than 2% of cases. As with all technical complications, the prevalence is increased in split liver LTX due to the smaller size of the bile ducts and vessels at the anastomoses.

Portal vein stenosis is uncommon and may occur in 1 to 2% of patients.[10] Sonography may demonstrate venous anastomotic stenosis directly or show indirect signs such as luminal thrombosis, slow flow, or turbulent flow across the anastomosis. In the setting of supra-hepatic caval stenosis, suggested by a 3- to 4-fold velocity gradient, there may be decreased phasicity in the hepatic veins or even reversal of flow.[15] CT and MR can confirm venous complications, and transhepatic portography may confirm a physiologically significant flow gradient as well as allowing for possible thrombectomy, balloon dilatation or stent placement.[21] Surgical revision may be necessary in some cases.

### Biliary Complications

Most LTX procedures result in a duct-to-duct anastomosis which is bridged by a T-tube, allowing for easy access for postoperative cholangiography. Duct-enteric anastomosis requires transhepatic cholangiography for direct biliary visualization.

Bile duct complications are reported in up to 25% of LTX procedures,[22] and most (up to 80%) occur within the first 3 months following transplantation.[23] Bile leaks may occur from the T-tube entry site, the ductal anastomosis, or in the intrahepatic ducts, and these 3 patterns have different clinical implications. Leaks from a T-tube entry site are usually recognized soon after the removal of the T-tube several months following LTX. These respond well to endoscopic papil-

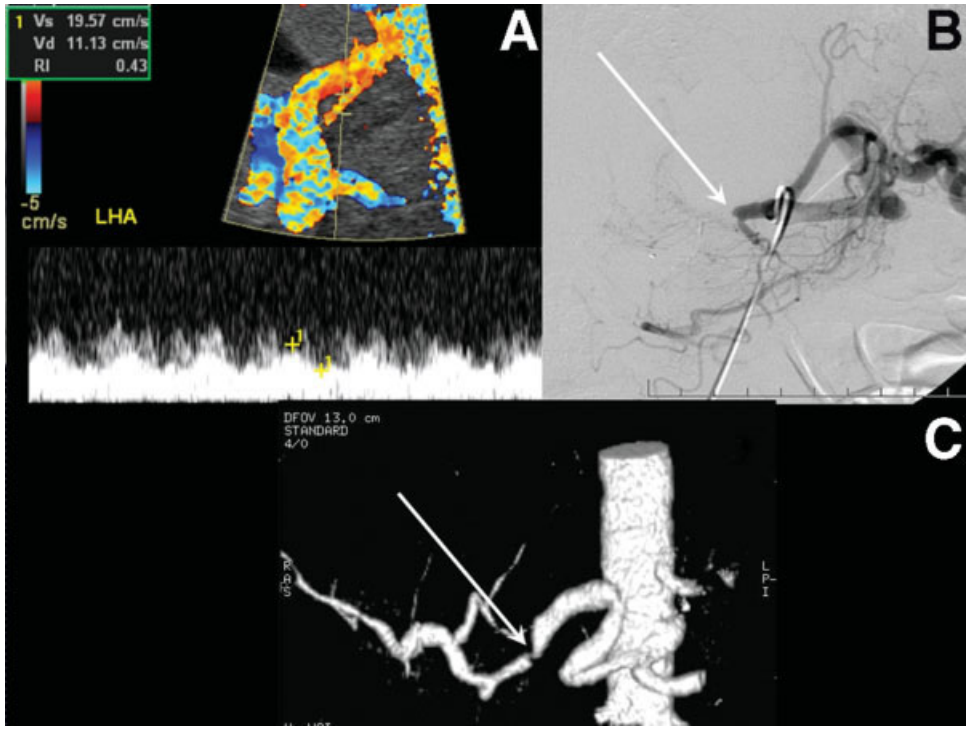


**Figure 9. (A) Color Doppler ultrasound depicting hepatic artery stenosis as seen by the "parvus tardus" waveform and the lower resistive index of 0.43 (normal > 0.5). (B) Hepatic artery thrombosis on catheter angiography. No opacification of the hepatic artery on celiac artery injection (red arrow). (C) Volume-rendered image from a CT arteriogram that also depicts hepatic artery stenosis (red arrow).**

lotomy and placement of a temporary biliary stent. Anastomotic leaks represent a surgical mishap and usually require surgical revision of the anastomosis. More proximal, nonanastomotic biliary leaks or strictures are often (approximately 89%) the result of hepatic artery stenosis or thrombosis leading to biliary ischemia[24] (Fig. 10). In addition to the ductal injury, these patients often develop intrahepatic bilomas, which frequently become infected, especially in patients with duct-enteric anastomosis and biliary contamination by enteric organisms occurs.

Less common biliary complications including biliary stenosis or sludge may result in partial ductal obstruction. Biliary casts may result from a variety of injuries including graft ischemia, HA stenosis, infection, or anastomotic stricture, though some cases have no clear etiology. These are detected best by cholangiography and may be amenable to endoscopic papillotomy and balloon sweeping of the bile ducts (Fig. 11).

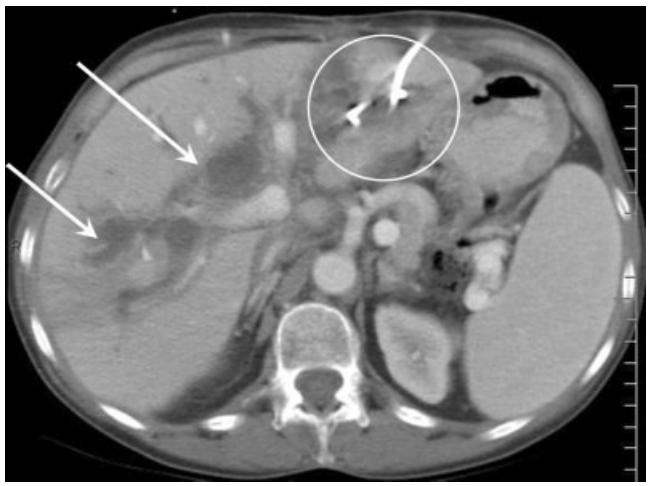

**Figure 10. Contrast-enhanced CT demonstrating the biliary necrosis secondary to hepatic artery thrombosis (HAT). The arrows depict dilated, ischemic bile ducts. A drainage catheter (circle) is present within the dilated left ducts. The drainage from this was purulent.**

## Abscess and Fluid Collection

Loculated ascites occurs commonly in the post-LTX period and is easily identified by sonography or CT. Distinction from abscess or biloma often requires aspiration and analysis and is best achieved by sonographic real-time guidance of needle aspiration or catheter placement.

Percutaneous catheter drainage is also appropriate and effective for diagnosis of most abscesses and bilomas. Patients who have received partial liver LTX are especially prone to bile leaks, due to the large surface area of transected ducts and the small size of the ducts being anastomosed (Fig. 12).

Patients who have infected intrahepatic bilomas may benefit by catheter drainage of the infected fluid. Since this is usually due to hepatic artery stenosis or throm-

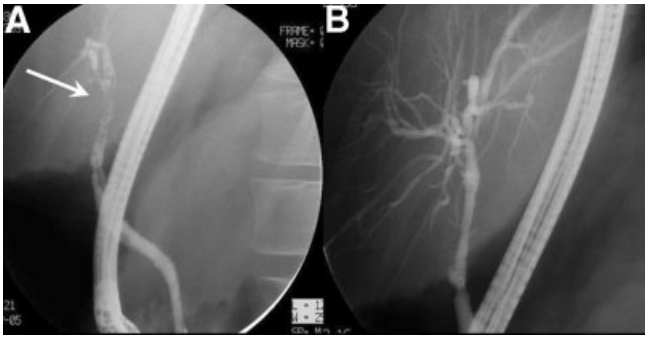


**Figure 11. (A) Endoscopic cholangiogram depicting biliary casts (arrow) within common duct post-LTX (duct-to-duct anastomosis). (B) Endoscopic cholangiogram performed following balloon sweeping to clear the casts.**

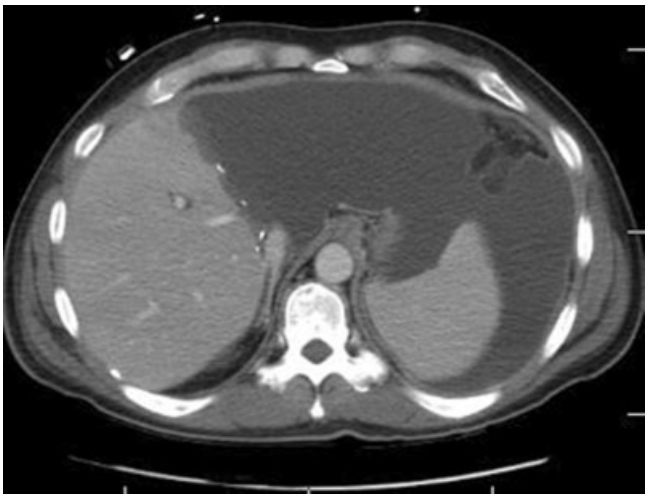

**Figure 12. Enhanced axial CT image depicting a large biloma following living-related right lobe hepatic transplant. This arises along the cut edge of the transplanted lobe.**

bosis, most adults will ultimately require surgical intervention or retransplantation.

## Recurrent Disease

Even with a technically successful LTX, patients are at a lifetime increased risk of complications, including recurrence of their primary disease. Recurrent damage to the transplanted liver by viral hepatitis, primary biliary cirrhosis, or primary sclerosing cholangitis is well documented. Imaging assumes the same role in this setting as it does for the initial evaluation of the liver.

## Posttransplantation Malignancy

All organ transplant recipients are at increased risk for the development of various malignancies, largely related to the need for long-term immunosuppression. Some 5% of LTX recipients develop malignant tumors, especially skin cancers and lymphomas.

Posttransplantation lymphoproliferative disorder (PTLD) is related to the weakened immune system of these patients and the presence of Epstein-Barr viral

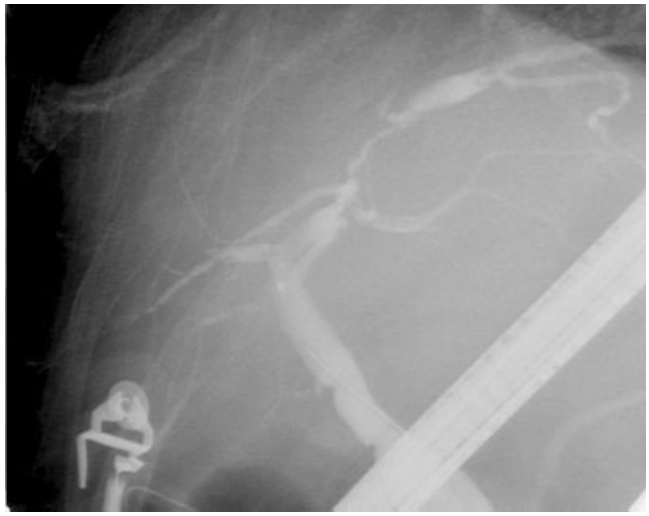

**Figure 13. Recurrent primary sclerosing cholangitis: Endoscopic cholangiogram reveals that the intra- and extrahepatic ducts have a beaded appearance with several strictures involving the intrahepatic ducts. This cholangiogram was performed approximately 6 months after LTX. The patient had recurrent primary sclerosing cholangitis.**

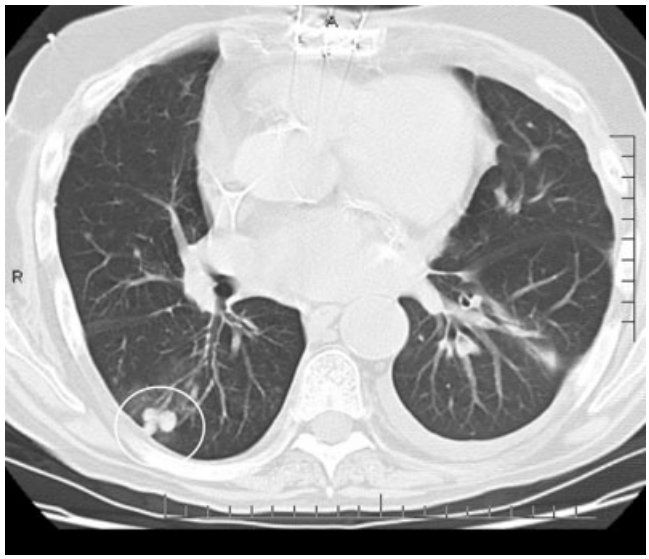


**Figure 14. PTLD manifesting as pulmonary nodules (circle) on CT scan.**

infection. The viral infection is thought to induce a polyclonal proliferation of B-lymphocytes which may progress to a monoclonal B-cell lymphoma.

PTLD develops in about 2 to 8.4%[25] of LTX patients, although the prevalence may be decreasing with newer anti-rejection medication regimens. PTLD usually becomes clinically evident within 4 to 12 months following LTX[26] and may involve almost any part of the body (Figs. 14 and 15). While lymph node involvement is common, PTLD differs from lymphoma in the nontransplant population due to its propensity to involve the bowel and abdominal viscera, including the liver. Focal soft-tissue density masses are characteristic findings,

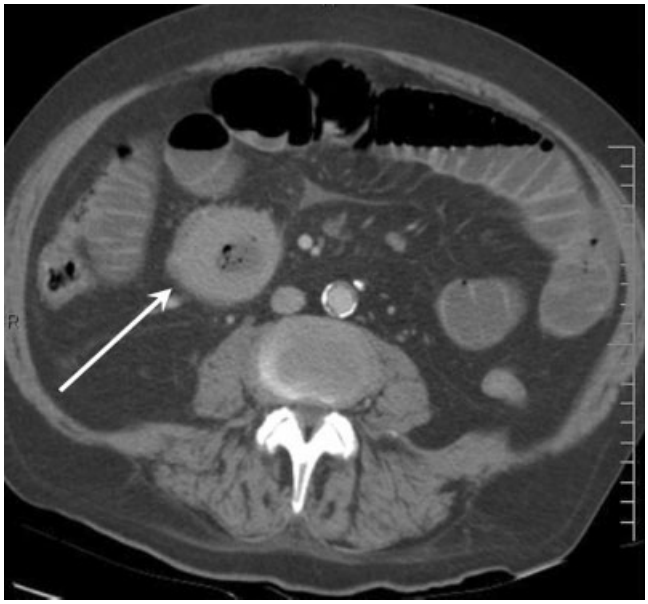

**Figure 15. PTLD manifesting as bowel wall thickening (arrow) on CT.**

and the liver masses are more homogeneous and hypovascular than hepatocellular carcinoma. CT is very accurate in diagnosing PTLD and the recent introduction of combined CT and positron-emission tomography is especially useful in detecting multifocal disease and in assessing response to therapy.

Recurrence of primary hepatic malignancy is well recognized in those patients who have had orthotopic LTX for known tumors.[27] In the case of HCC, this usually will occur in the lungs first and then is seen in the graft. Recurrence of cholangiocarcinoma is especially common and often occurs within 2 years, making LTX an ineffective treatment for cholangiocarcinoma in most cases.

Patients who have early-stage HCC usually do well after LTX with a low rate of recurrence. Factors that predict a high rate of recurrence, and which usually preclude LTX include large solitary mass (larger than 5 cm), multiple smaller masses (more than 3 lesions, larger than 3 cm),[6] vascular invasion, or extrahepatic malignancy.

## SUMMARY

Imaging and image-guided interventions play a vital role in the evaluation of potential transplant recipients, partial liver living donors, and posttransplantation complications. Appropriate use of the different modalities, optimal imaging technique, and expert interpretation are crucial in providing these roles.

## REFERENCES


1. Eghtesad B, Kadry Z, Fung J. Technical considerations in liver transplantation: what a hepatologist needs to know (and every surgeon should practice). Liver Transpl 2005; 11:861-871.
2. Dodd GD III, Baron RL, Oliver JH III, Federle MP. End-stage primary sclerosing cholangitis: CT findings of hepatic morphology in 36 patients. Radiology 1999;211:357-362.
3. Blachar A, Federle M, Brancatelli G. Primary biliary cirrhosis: clinical, pathologic, and helical CT findings in 53 patients. Radiology 2001;220:329-336.
4. Hermoye L, Laamari-Azjal I, Cao Z, Annet L, Lerut J, Dawant B, Van Beers B. Liver Segmentation in living liver transplant donors: comparison of semiautomatic and manual methods. Radiology 2005;234:171-178.
5. Manzanet G, Sanjuan F, Orbis OP, Lopez R, Moya A, Juan M, et al. Liver transplantation in patients with portal vein thrombosis. Liver Transpl 2001;7:125-131.
6. Hussain SM, Zondervan P, IJzermans J, Schalm S, de Man R, Krestin G. Benign versus malignant hepatic nodules: MR imaging findings with pathologic correlation. Radiographics 2002;22:1023–1036.
7. Krinsky GA, Lee VS, Theise ND, Weinreb JC, Rofsky NM, Diflo T, Teperman LW. Hepatocellular carcinoma and dysplastic nodules in patients with cirrhosis: prospective diagnosis with MR imaging and explantation correlation. Radiology 2001;219:445-454.
8. Peterson MS, Baron RL, Marsh J, Oliver JH, Confer SR, Hunt LE. Pretransplantation surveillance for possible hepatocellular carcinoma in patients with cirrhosis: epidemiology and CT-based tumor detection rate in 430 cases with surgical pathological correlation. Radiology 2000;217:743-749.
9. Tublin M, Dodd GI, Baron R. Benign and malignant portal vein thrombosis: differentiation by CT characteristics. Abdom Imaging 1997;168:719-723.
10. Mazzaferro V, Regalia E, Doci R, Andreola S, Pulvirenti A, Bozzetti F, et al. Liver transplantation for the treatment of small hepatocellular carcinomas in patients with cirrhosis. N Engl J Med 1996;334:693-699.
11. Schroeder T, Nadalin S, Stattaus J, Debatin JF, Malagó M, Ruehm S. Potential living liver donors: evaluation with an all-in-one protocol with multi-detector row CT. Radiology 2002;224:586-591.
12. Lee VS, Krinsky GA, Nazzaro CA, Chang JS, Babb JS, Lin JC, et al. Defining intrahepatic biliary anatomy in living liver transplant donor candidates at mangafodipir trisodium-enhanced MR cholangiography versus conventional T2-weighted MR cholangiography. Radiology 2004;233:659-666.
13. Wang ZJ, Yeh BM, Roberts JP, Breiman RS, Qayyum A, Coakley FV. living donor candidates for right hepatic lobe transplantation: evaluation at CT cholangiography—initial experience. Radiology 2005;235:899-904.
14. Yeh BM, Breiman RS, Taouli B, Qayyum A, Roberts JP, Coakley FV. Biliary tract depiction in living potential liver donors: comparison of conventional MR, mangafodipir trisodium-enhanced excretory MR, and multi-detector row CT cholangiography—initial experience. Radiology 2004; 230:645-651.
15. Katyal S, III JO, Buck D, Federle M. Detection of vascular complications after liver transplantation; early experience in multi-slice CT angiography with volume rendering. AJR Am J Roentgenol 2000;175:1735-1739.
16. Langnas A, Marujo W, Stratta R. Vascular complications after orthotopic liver transplantation. Am J Surg 1991; 161:76-82.
17. Nghiem HV. Imaging of hepatic transplantation. Radiol Clin North Am 1998;36:429-443.
18. Flint E, Sumkin J, Zajko A, Bowen A. Duplex sonography of hepatic artery thrombosis after liver transplantation. AJR Am J Roentgenol 1988;151:481-483.
19. Dodd GD III, Memel DS, Zajko AB, Baron RL, Santaguida LA. Hepatic artery stenosis and thrombosis in transplant

recipients: Doppler diagnosis with resistive index and systolic acceleration time. Radiology 1994;192:657-661.

20. Orons P, Sheng R, Zajko A. Hepatic artery stenosis in liver transplant recipients: prevalence and cholangiographic appearance of associated biliary complications. AJR Am J Roentgenol 1995;165:1145-1149.
21. Olcott EW, Ring EJ, Roberts JP, Ascher NL, Lake JR, Gordon RL. Percutaneous transhepatic portal vein angioplasty and stent placement after liver transplantation: early experience. J Vasc Interv Radiol 1990;1:17-22.
22. Letourneau JG, Castaneda-Zuniga WR. The role of radiology in the diagnosis and treatment of biliary complications after liver transplantation. Cardiovasc Intervent Radiol 1990;13:278-282.
23. Everson GT, Kam I. Immediate postoperative care. In: Maddrey WC, Schiff ER, Sorrell MF, eds. Transplantation of the Liver, 3d ed. Philadelphia: Lippincott Williams & Wilkins, 2001:131-162.
24. Nghiem HV, Tran K, Winter TC III, Schmiedl UP, Althaus SJ, Patel NH, Freeny PC. Imaging of complications in liver transplantation. Radiographics 1996;16:825-840.
25. Wu L, Rappaport DC, Hanbidge A, Merchant N, Shepherd FA, Greig PD. Lymphoproliferative disorders after liver transplantation: imaging features. Abdom Imaging 2001; 26:200-206.
26. Strouse PJ, Platt JF, Francis IR, Bree RL. Tumorous intrahepatic lymphoproliferative disorder in transplanted livers. AJR Am J Roentgenol 1996;167:1159-1162.
27. Ferris JV, Baron RL, Marsh JW Jr, Oliver JH III, Carr BI, Dodd GD III. Recurrent hepatocellular carcinoma after liver transplantation: spectrum of CT findings and recurrence patterns. Radiology 1996;198:233-238.