Complications of Orthotopic Liver Transplantation: Spectrum of Findings with Helical CT[1]

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CME FEATURE

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LEARNING OBJECTIVES FOR TEST 1

After reading this article and taking the test, the reader will be able to:

- List the most common surgical techniques used in OLT.
- Describe the helical CT appearance of the normal transplanted liver.
- Recognize a wide range of vascular and nonvascular complications of OLT.

Orthotopic liver transplantation has become the treatment of choice for patients with end-stage nonmalignant liver disease. The surgical techniques and immunosuppressive therapy for this procedure have improved considerably. Nevertheless, there are still significant complications, particularly those of vascular origin, which can lead to graft failure and require retransplantation unless prompt treatment is instituted. These complications include arterial and venous thrombosis and stenosis; arterial pseudoaneurysm; biliary leakage, stricture, and obstruction; liver ischemia, infarction, and abscess; fluid collections and hematomas; lymphoproliferative disorders; recurrent tumors; hepatitis C virus infection; and splenic infarction. Since the clinical presentation of posttransplantation complications is frequently nonspecific and varies widely, imaging studies are critical for early diagnosis. Helical computed tomography (CT) is a valuable complement to ultrasonography (US) in the postoperative period and is a safe, accurate, and noninvasive method of demonstrating hepatic vessels (hepatic artery, portal vein, hepatic veins, and inferior vena cava) and evaluating nonvascular complications (in the hepatic parenchyma and bile duct abnormalities) and extrahepatic tissues. Knowledge and early recognition of these complications is essential for graft salvage, and CT can provide valuable information, particularly for patients with indeterminate US results or in whom US examination is difficult.

Abbreviations: IVC = inferior vena cava, MPR = multiplanar reconstruction, OLT = orthotopic liver transplantation, SSD = shaded-surface display

Index terms: Computed tomography (CT), helical, 76.12115 • Liver, CT, 76.12115 • Liver, transplantation, 76.451, 76.458

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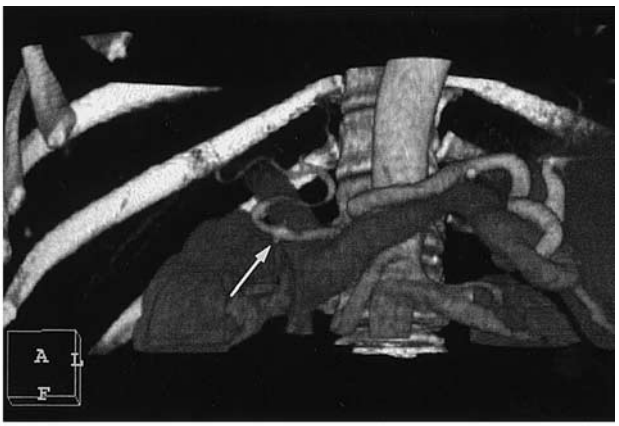

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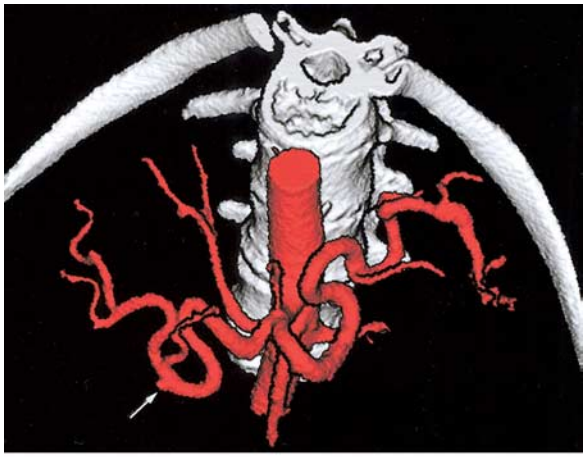


3.

Figures 1–3. **(1)** Volume-rendered reconstruction image shows a normal hepatic artery in which the area of arterial suture (arrow) can be identified. *A* = anterior, *F* = feet, *L* = left. **(2)** Volume-rendered reconstruction image clearly shows the typical fish-mouth appearance of the arterial anastomosis (arrow). *A* = anterior, *F* = feet, *L* = left. **(3)** Shaded-surface display (SSD) reconstruction image (anterosuperior view) shows a normal hepatic artery and the anastomotic site (arrow).

Introduction

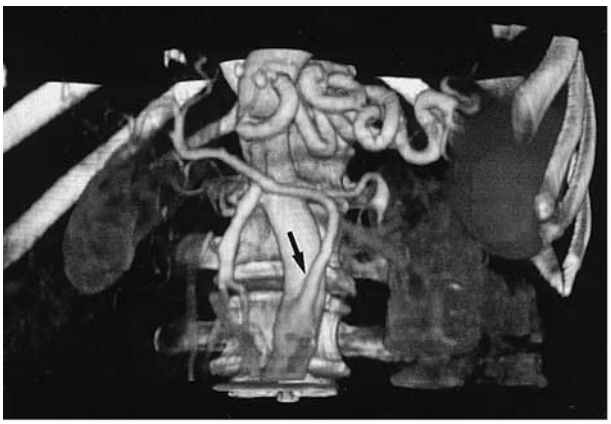
Orthotopic liver transplantation (OLT) is currently the treatment of choice for patients with severe acute or chronic liver failure for which no other therapy is available (1–4). Liver failure can have a number of causes, including autoimmune hepatitis; chronic viral hepatitis; alcoholic liver disease; metabolic diseases (α_1-antitrypsin deficiency, hemochromatosis, Wilson disease); cholestatic liver disorders (primary biliary cirrhosis, primary sclerosing cholangitis, biliary atresia); and severe acute liver failure due to viral hepatitis, drug-induced hepatitis (eg, by acetaminophen or isoniazid), or hepatotoxins (eg, mushrooms) (3). Patients with hepatocellular carcinoma, cholangiocarcinoma, or inoperable neuroendocrine metastases are also potential candidates for OLT. The absolute contraindications for OLT include acquired immunodeficiency syndrome, extrahepatic malignant tumors, and active intravenous drug use or alcohol abuse. The care of these critically ill patients has relied heavily on cross-sectional imaging, and there is a greater demand for accurate evaluation of complications because early diagnosis is critical for graft salvage.

Ultrasonography (US) is the initial imaging technique used for the detection of complications in the early posttransplantation phase, since it can be performed at the bedside and is capable of demonstrating the hepatic parenchyma and bile ducts. Doppler US allows detection of vascular abnormalities, but it is associated with a significant frequency of false-negative results (5,6). In cases where US results are inconclusive, confirmation is required, or clinical suspicion of a complication persists despite normal US results, helical computed tomography (CT) should be performed.

This pictorial essay reviews the results of 142 helical CT studies of 91 liver transplant recipients and illustrates the normal postoperative findings and complications of liver transplantation. Specific topics discussed are helical CT technique, normal appearance after OLT, vascular complications, biliary complications, liver ischemia or infarction, fluid collections and hematomas, malignancy, and other complications. The helical CT findings that facilitate diagnosis of these complications are highlighted and illustrated herein.

Helical CT Technique

All helical CT examinations were performed with a Twin Plus II CT scanner (Elscint, Haifa, Israel) by using a dual-section spiral technique (dual detector ring). After thoroughly reviewing the surgical technique used in the patient, particularly the



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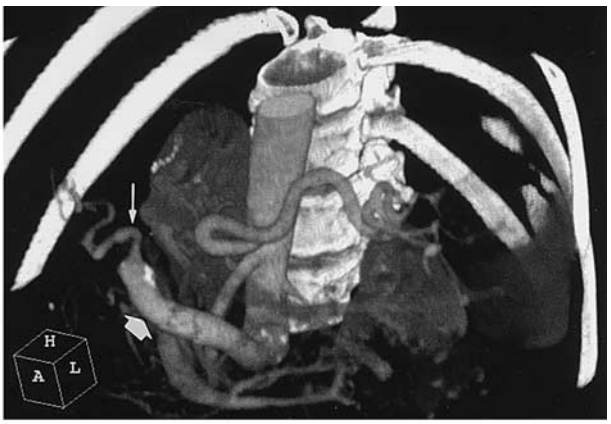


5.

Figures 4, 5. **(4)** Volume-rendered reconstruction image (anterior view) shows an infrarenal graft of the donor iliac artery (arrow) without features of stenosis. **(5)** Volume-rendered reconstruction image shows a donor iliac artery (thick arrow) grafted to the infrarenal aorta. Note the marked difference in caliber between the iliac artery and the donor celiac trunk (thin arrow). *A* = anterior, *H* = head, *L* = left.

configurations and locations of the vascular anastomoses, we first obtained nonenhanced, nonhelical, contiguous 10-mm-collimation scans to determine the appropriate coverage. The helical CT acquisition was designed to cover the entire craniocaudal extent of the liver and vascular anastomoses and was performed during the hepatic arterial phase under the following conditions: 2.5-mm section thickness, pitch ratio of 1, and reconstruction at 1.3-mm intervals. A total of 100 mL of nonionic contrast material (350 mg of iodine per milliliter) was injected via an antecubital vein at a flow rate of 3 mL/sec. The delay time was 25 seconds after the start of contrast material administration. Test bolus injections were not performed except in young patients and in patients with decreased cardiac output. A second helical sequence was performed during the portal venous phase under the following conditions: 5-mm section thickness, pitch ratio of 1, and reconstruction at 5-mm intervals, with a delay of 65–90 seconds from the start of contrast material administration. Water was used as a negative contrast material to distend the stomach and duodenum, since it permits detailed demonstration of the gastrointestinal wall during bolus injection of contrast material and does not interfere with visualization of the vascular system. Multiplanar and three-dimensional reconstruction was performed in all patients by a radiologist (S.Q., M.C.S.).

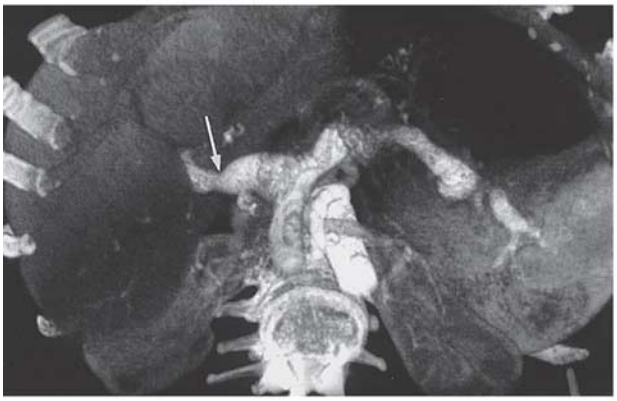
Normal Appearance after OLT

CT is not routinely performed in a regular posttransplantation course. Nevertheless, the radiologist should be familiar with the normal postoperative helical CT findings of the transplanted liver to avoid misdiagnosis and detect complications. OLT requires grafting of one arterial anastomosis (hepatic artery), at least two venous anastomoses (portal vein and inferior vena cava [IVC]), and a biliary anastomosis.

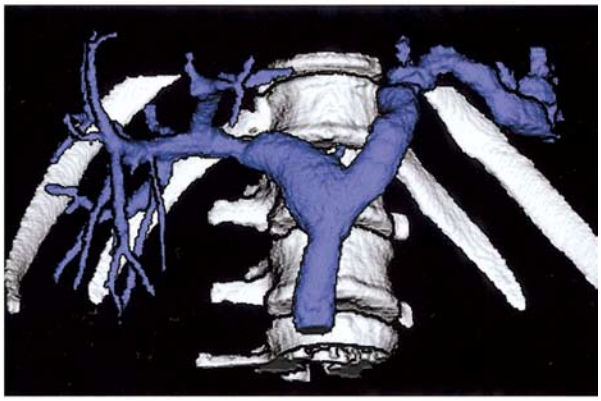
Hepatic Artery

The hepatic artery is typically reconstructed with a "fish-mouth" anastomosis between the donor and recipient arterial anastomotic sites (Figs 1–3), usually between the celiac axis of the donor and the bifurcation of the right and left hepatic arteries of the recipient or the branch point of the gastroduodenal and proper hepatic arteries of the recipient (3,7). In addition, the surgeon can decide on several variations of this procedure during the operation for patients with anatomic variants of hepatic vascularization, such as a replaced right hepatic artery from the superior mesenteric artery, and in those with small diameter of or little flow from the native hepatic artery (3,7). In the latter group, a donor iliac artery interposition graft anastomosed directly to the supraceliac or infrarenal aorta (Figs 4, 5) is often used (8). It is extremely important to know which surgical technique was used in each patient before planning the helical CT examination so that all of the anastomoses will be included in the study. The radiologist should also have a clear idea of the typical morphology of the fish-mouth anastomosis to avoid erroneous diagnoses.

6.

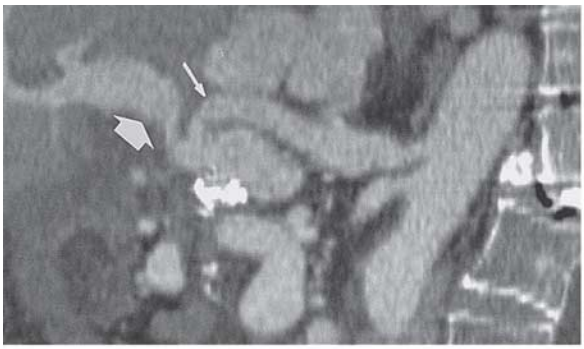


7a.



7b.

Figures 6, 7. **(6)** SSD venous reconstruction image (anterior view) clearly shows the area of portal venous suture (arrow). **(7a)** Volume-rendered reconstruction image (anteroinferior view) clearly shows the area of portal vein anastomosis (arrow). Note the marked difference in caliber between the donor and recipient portal veins. **(7b)** SSD reconstruction image (anterior view) shows the difference in caliber between the two portal veins, which is a risk factor for portal vein thrombosis.



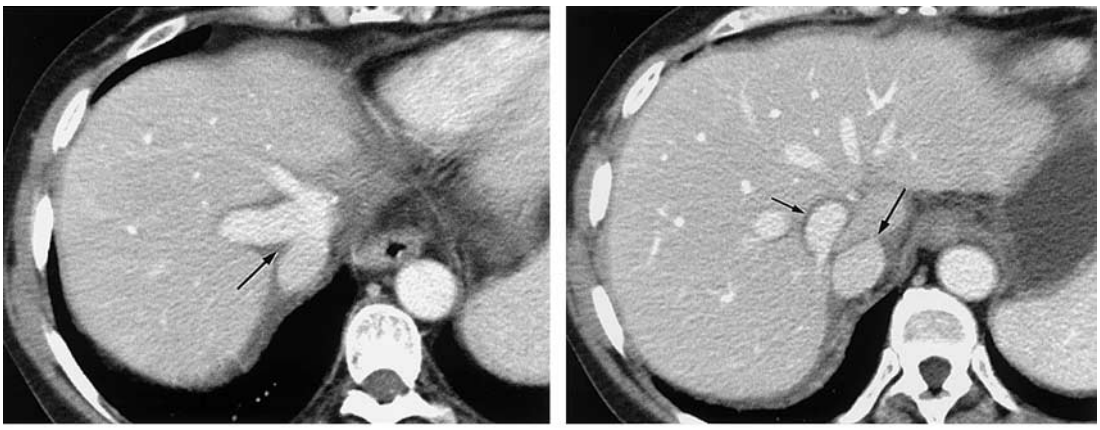
a.

b.

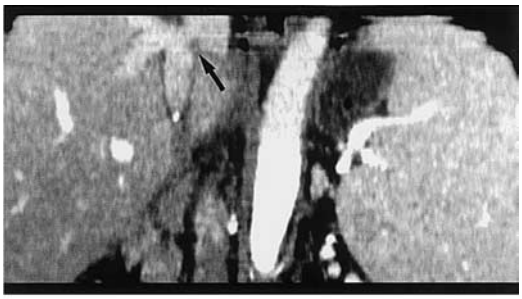
Figure 8. **(a)** Multiplanar reconstruction (MPR) image shows an end-to-side anastomosis between the recipient hepatic artery (thin arrow) and the donor portal vein (thick arrow). **(b)** SSD reconstruction image (anteroinferior view) shows the portal vein anastomosis (arrow).

Portal Vein

The portal vein anastomosis is typically an end-to-end type between the two portal veins (Figs 6, 7). In cases of extensive portal vein thrombosis or previous portal vein surgery, a venous jump graft from the donor portal vein or iliac vein may be needed (3,7). Again, knowledge of the surgical technique is crucial for the radiologist to plan the CT study and read the images. Arterialization of the portal vein (ie, creation of anastomoses between both the portal vein and hepatic artery of the donor and arterial vessels of the recipient) is occasionally used as a last resort when a portal-visceral (splenic vein, superior or inferior mesenteric vein) venous anastomosis cannot be performed because of extensive venous thrombosis



a. b.



c.

Figure 10. **(a)** CT scan shows an end-to-end anastomosis between the donor IVC and the stump of the recipient hepatic veins (arrow), which was created with the piggyback technique. **(b)** CT scan obtained at the caudal level shows the donor IVC (small arrow) and recipient IVC (large arrow). **(c)** MPR image shows the IVC anastomosis (arrow).

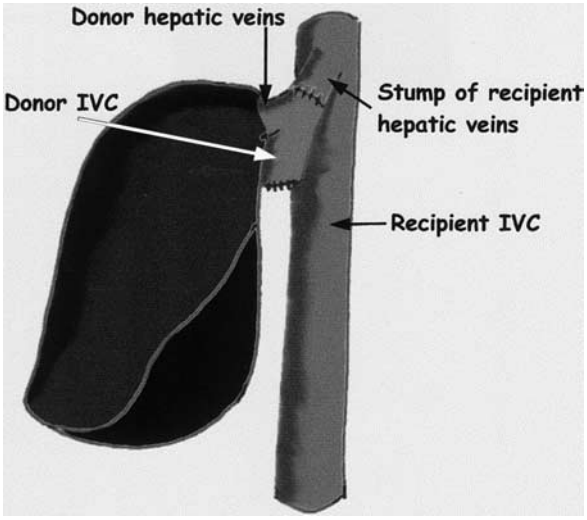
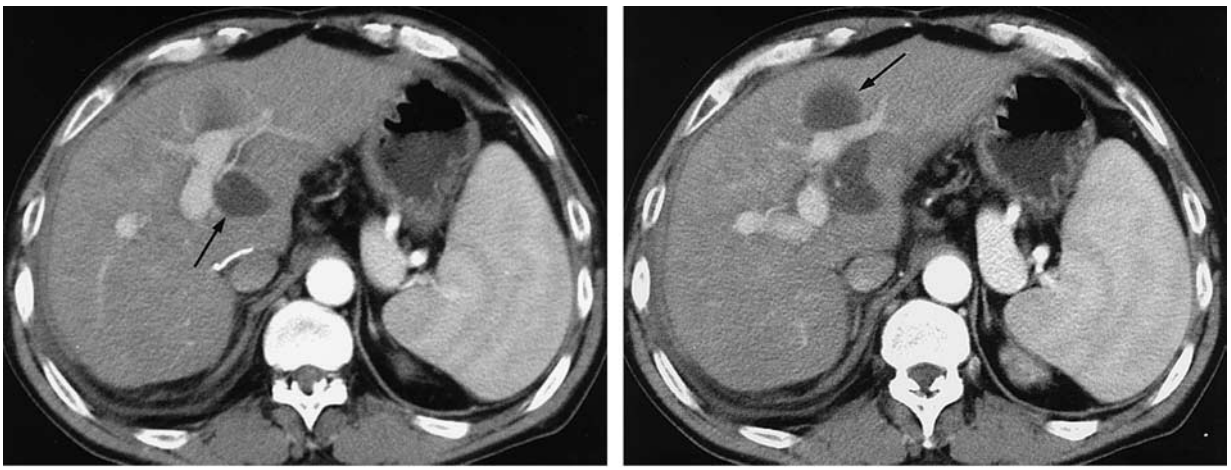


Figure 9. Schematic shows an anastomosis between the donor IVC and a common stump of the recipient's three hepatic veins, with preservation of the recipient IVC.

(Fig 8). The radiologist should be aware of this possibility. Anastomotic narrowing of the portal vein can be due to the smaller caliber of the donor portion than of the recipient portion (Fig 7) (4), and there can also be transient portal vein narrowing caused by surrounding edema or fluid collections (4). The typical end-to-end venous anastomosis is often difficult to identify on axial CT sections, although sometimes the area of anastomosis is visible on vascular reconstruction images.

Inferior Vena Cava

During hepatectomy, the retrohepatic IVC of the recipient is usually resected and the IVCs of the recipient and donor are sutured twice, with end-to-end anastomoses (3,7). New techniques have recently appeared, with preservation of the recipient retrohepatic IVC and creation of anastomoses between the donor and recipient IVCs in an end-to-side or side-to-side configuration or an end-to-end anastomosis between the donor IVC and a common stump of the three hepatic veins (the piggyback technique) (Figs 9, 10) (9,10). This last technique has gained increasing acceptance in OLT because the IVC flow is not interrupted throughout the vast majority of the operation, the problem of graft outflow is avoided, and most of the IVC flow is preserved. This is the technique used at our institution.



a. b.

Figure 11. CT scans show postoperative fluid collections in the area of the venous ligament (arrow in **a**) and the fissure for the ligamentum teres (arrow in **b**).

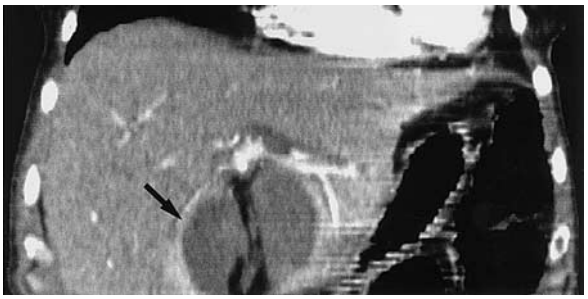


Figure 12. MPR image shows a fluid collection in the fissure for the ligamentum teres (arrow), which compresses the adjacent hepatic parenchyma.

Biliary Anastomosis

The biliary anastomosis is made between the donor common bile duct and the recipient common hepatic duct, usually after a cholecystectomy. This technique avoids intestinal surgery, preserves the sphincter of Oddi, and reduces the risk of enteric reflux into the biliary tree (11). A T-tube is left in place for cholangiography. If there is a problem with the common bile duct of the recipient, such as a diseased recipient common hepatic duct (eg, primary sclerosing cholangitis) or one that is too short, too small, or absent, choledochojejunostomy is usually performed (3).

Other Findings

Other normal findings after OLT include right pleural effusion and a small amount of free intraabdominal fluid or hematomas in the perihepatic region, especially in the hepatic hilum, adjacent to the IVC anastomoses, or in the fissure for the ligamentum teres (Figs 11, 12) (4,12). These usually resolve within a few weeks, although infiltration of the hepatic hilum fat can sometimes persist for months.

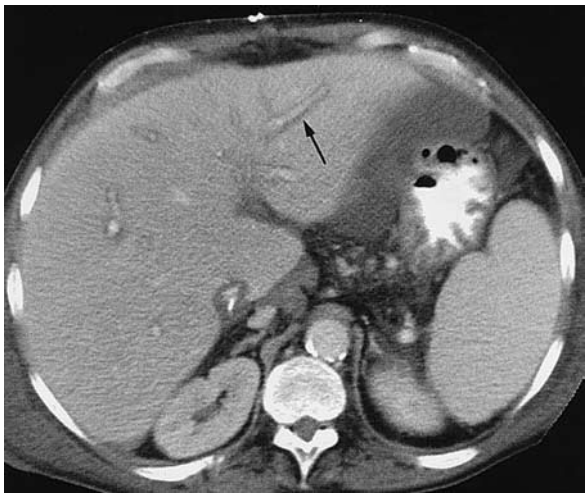
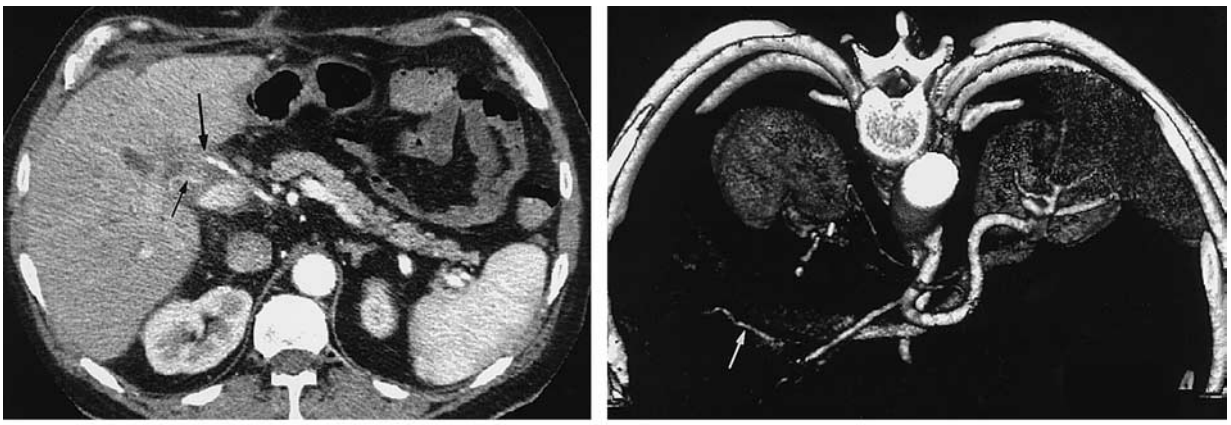


Figure 13. CT scan from an early postoperative study of an OLT patient shows periportal edema (arrow).

Finally, a periportal area of low attenuation is often seen (Fig 13). This finding is attributed to dilatation of lymphatic channels due to lack of normal lymphatic drainage into the extrahepatic lymphatic system (4,12,13). The periportal halo resolves within weeks following transplantation (possibly due to development of alternative pathways), although it can persist for months; it should not be confused with dilatation of the intrahepatic biliary pathway. This periportal edema was once considered a sign of graft rejection, but later studies have ruled out this relationship.

Vascular Complications

Vascular complications are estimated to occur in 9% of patients (14) and are a primary diagnostic consideration in OLT patients with liver failure, bile leak, abdominal bleeding, or septicemia (3). Vascular complications are the most frequent cause of graft loss. Most transplantation centers perform routine postoperative Doppler US as the



a. b.

Figure 14. **(a)** CT scan shows hepatic artery thrombosis at the area of anastomosis (large arrow) with patency of some distal vessels (small arrow), probably due to formation of collateral vessels. **(b)** Volume-rendered reconstruction image (superior view) shows similar findings, with patency of small intrahepatic arterial vessels (arrow).

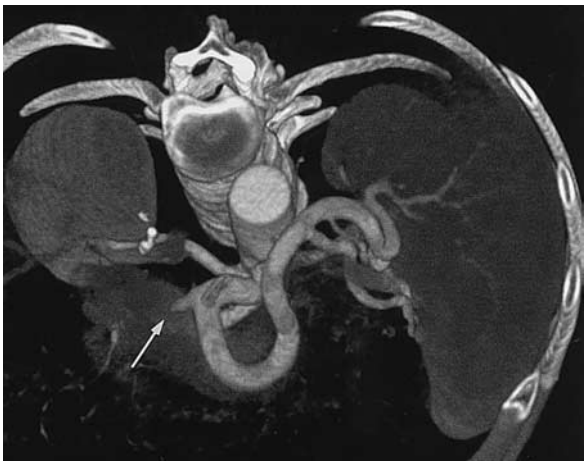


Figure 15. Volume-rendered reconstruction image (anterosuperior view) shows hepatic artery thrombosis (arrow).

initial imaging study to evaluate the integrity of the graft vasculature, since it can be performed with portable equipment and has a high sensitivity and specificity for detection of arterial and venous thrombosis (7,15). However, extensive bowel gas or extrahepatic fluid collections and hematomas, which are frequent in the postoperative period, can pose problems for US study. These factors do not impede vascular study with helical CT, which can be an alternative noninvasive technique for evaluating the hepatic vasculature. The most important vascular complication, with a potential to cause graft failure, is thrombosis of the hepatic artery or portal vein. Vascular complications related to the IVC are much less frequent. Although CT requires careful use of iodinated contrast material, particularly in patients with impaired renal function, CT is now widely available and is faster than other methods, such as magnetic resonance (MR) imaging, for detection of these problems.

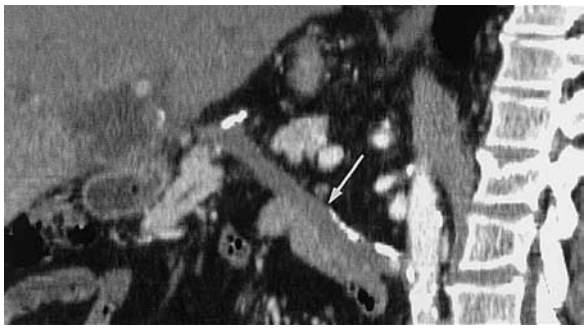
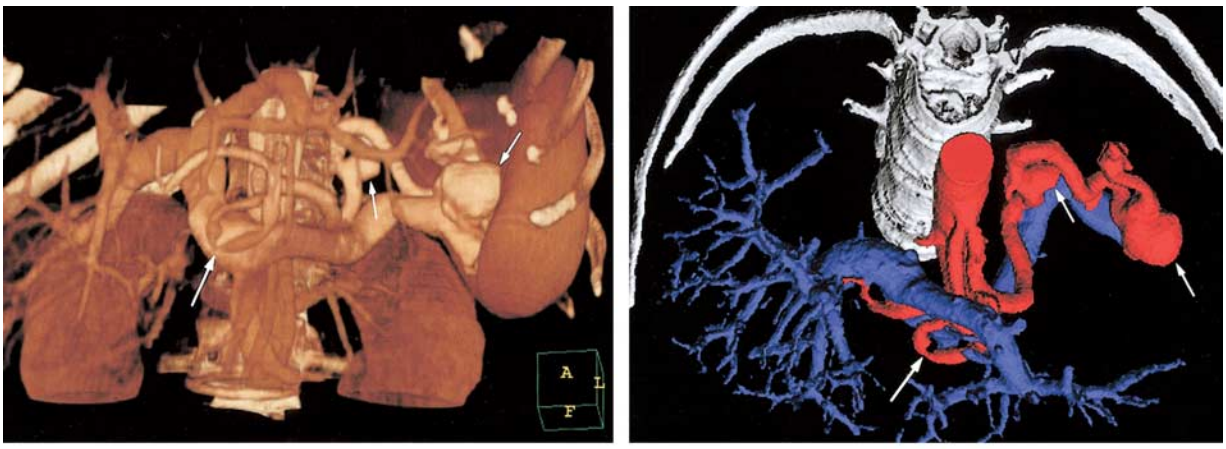


Figure 16. MPR image shows complete thrombosis of the donor iliac artery graft (arrow) to the infrarenal aorta.

Hepatic Artery Thrombosis

Hepatic artery thrombosis, the most common vascular complication of OLT, has a prevalence of 4%–12% in adult recipients and up to 40% in children (1–4,7,15–17) and a mortality rate of 50%–58% (18,19). Unless thrombectomy can be performed, most cases require retransplantation; even after retransplantation, the mortality rate is 27%–30% (18,20). Risk factors for hepatic artery thrombosis include (*a*) significant differences in caliber between the donor and recipient hepatic arterial vessels (Fig 5) or preexisting lesions such as celiac artery stenosis, (*b*) prolonged cold ischemia time of the donor liver, (*c*) ABO blood type incompatibility, and (*d*) rejection (3,4,7,18). The clinical presentation of hepatic artery thrombosis shows considerable variation, ranging from mild elevation of liver enzyme levels to delayed bile leak, bile duct stricture or ischemic changes, relapsing bacteremia, or fulminant hepatic necrosis (1,3,7). Owing to this clinical variability, imaging studies are essential for early diagnosis. Contrast material–enhanced helical CT is a useful and comparatively less invasive tool for evaluating the patency of the entire hepatic artery (Figs 14–16)



a. b.

Figure 17. Volume-rendered reconstruction image **(a)** and SSD reconstruction image (anterosuperior view) **(b)** show hepatic artery stenosis distal to the anastomosis (large arrow) in a long artery with loops. There are also aneurysms in the splenic artery (small arrows). These have a higher risk of rupture in transplant recipients and should have been ligated during the operation. *A* = anterior, *F* = feet, *L* = left.

a. b.

Figure 18. MPR image **(a)** and SSD reconstruction image (anterior view) **(b)** show marked stenosis of the transplant hepatic artery at the anastomosis (arrow).

and avoids the use of diagnostic arteriography (1,14). Even in complete arterial thrombosis, small intrahepatic arterial vessels can sometimes be identified because of extensive collateralization to the liver (Fig 14). This situation can lead to false-negative results at Doppler US (1,21,22), although in most cases a tardus-parvus arterial waveform suggests the correct diagnosis (2,3,7). Hepatic artery thrombosis is often associated with bilomas, infarcts, abscesses, or bile duct dilatation (7).

Hepatic Artery Stenosis

Hepatic artery stenosis is the second most common vascular complication of OLT, reported in about 5% of cases (1,2,16). Hepatic artery stenosis generally occurs at the anastomotic site (Figs 17–19) within 3 months of OLT. If left untreated, it can lead to hepatic artery thrombosis due to slow flow (15,18,23,24) or progress to cause liver ischemia with hepatic insufficiency, biliary strictures, sepsis, and graft loss (23). The risk factors are similar to those for hepatic artery thrombosis, and surgical problems, such as faulty technique, clamp injury, and intimal trauma caused by per-

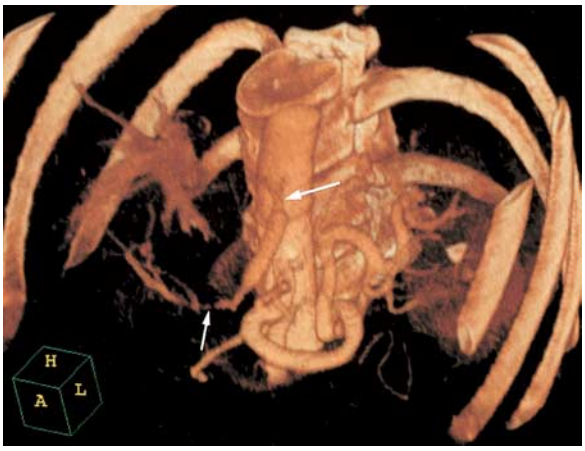
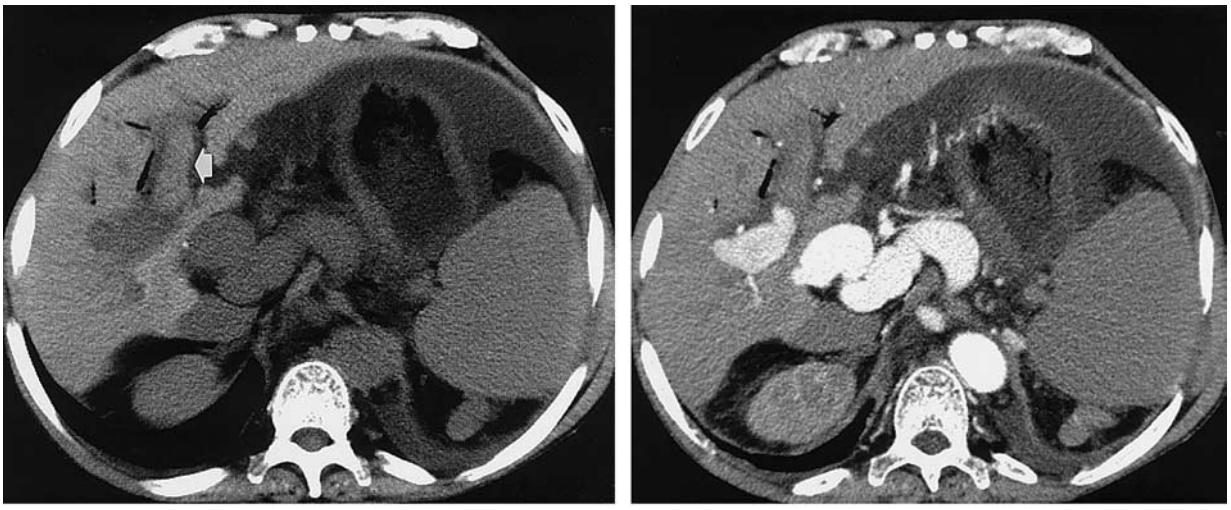


Figure 19. Volume-rendered reconstruction image shows the donor iliac artery grafted to the supraceliac aorta, with stenosis at the aortic anastomosis (large arrow) and a long, severe stenosis distal to the iliac artery (small arrow). *A* = anterior, *H* = head, *L* = left.



a. **b.**

Figure 20. **(a)** Nonenhanced CT scan shows hyperattenuating acute thrombosis of the left portal vein (arrow). **(b)** Helical CT scan also shows thrombosis of the left portal vein.

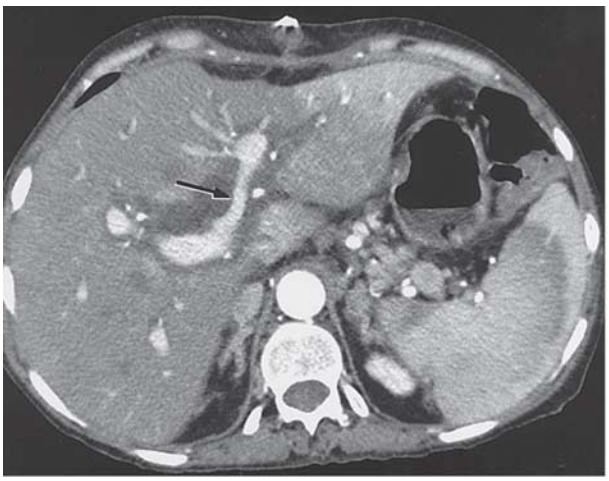
fusion catheters, are an additional cause (2,3). Early identification and reestablishment of adequate blood flow (revascularization surgical procedures or arteriography and balloon angioplasty) usually resolve the stenosis with long-term graft and patient survival (23), avoiding the need for retransplantation.

Hepatic Artery Pseudoaneurysm

Hepatic artery pseudoaneurysm is an uncommon complication that can cause major artery hemorrhage (3,7). Extrahepatic pseudoaneurysms usually develop at the vascular anastomosis or arise as a complication of angioplasty (2,25). They can rupture intraperitoneally and lead to massive hemorrhage. Treatment for extrahepatic pseudoaneurysms includes surgical resection, embolization, or exclusion with stent placement. Intrahepatic pseudoaneurysms, which can occur after percutaneous needle biopsy or local infection (3,7), are often detected incidentally. A ruptured intrahepatic pseudoaneurysm may result in portal vein or biliary fistulas (7), in the latter case manifesting as hemobilia or upper gastrointestinal bleeding. Intrahepatic pseudoaneurysms can be treated with endovascular coil embolization.

Portal Vein Thrombosis or Stenosis

Portal vein complications following OLT are relatively unusual, occurring in 1%–3% of cases (2,15,20,26), and result from faulty surgical technique, vessel misalignment, differences in caliber of anastomosed vessels (Fig 7) provoking turbulent flow, hypercoagulable states, previous portal vein surgery, or previous thrombosis in the recipient portal vein system (3). The clinical presentation includes symptoms of portal hypertension, liver failure, massive ascites, or edema (3,7). Helical CT can provide excellent visualization of filling defects within the portal vein (Fig 20) or



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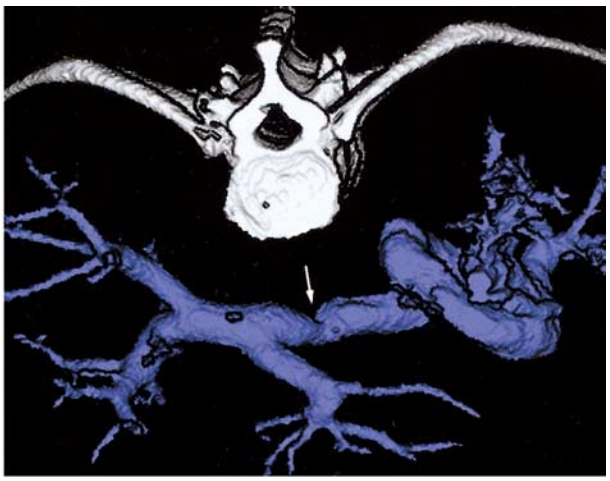

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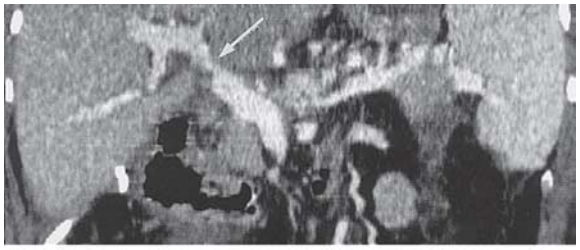


23.

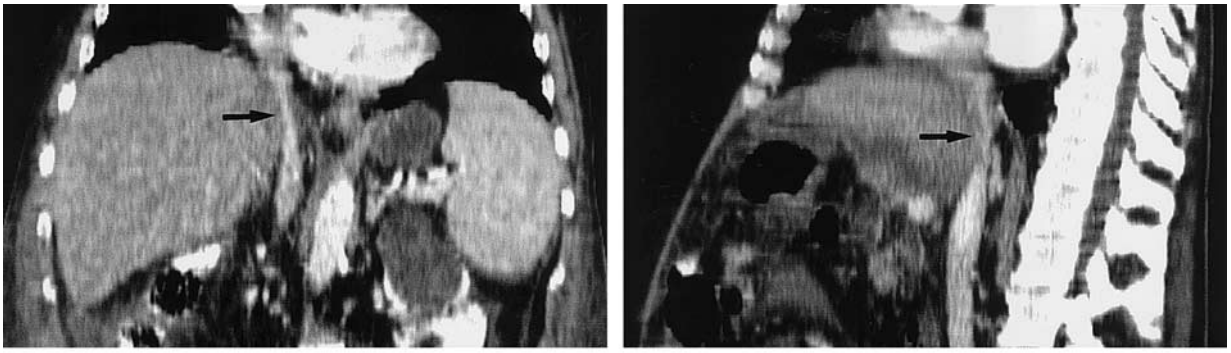
Figures 21–23. **(21)** Helical CT scan shows mild stenosis of the left portal vein (arrow) secondary to a postoperative fluid collection that affects the hepatic hilum and follows the vascular course to involve the liver. **(22)** SSD reconstruction image (superior view) shows portal vein stenosis in the area of the suture (arrow). **(23)** MPR image shows severe stenosis of the portal vein at the hepatic hilum (arrow).

focal narrowing (usually at the anastomosis) (Figs 21–23). However, such narrowing can occur naturally in patients in whom the discrepancy between donor and recipient portal vein sizes is significant (2,4). Percutaneous transhepatic direct portography allows measurement of the pressure gradient across the stenosis, with values higher than 5 mm Hg being significant (2). Treatment includes percutaneous transluminal angioplasty with or without stent placement (27), surgical thrombectomy, placement of a venous jump graft, creation of a portosystemic shunt, or even retransplantation (3,4). Arterialization of the portal vein (anastomosis of both the portal vein and hepatic artery of the donor graft with arterial vessels of the recipient) was performed in two cases at our hospital during OLT because extensive mesenteric vein thrombosis made it impossible to establish proper portal vein flow.

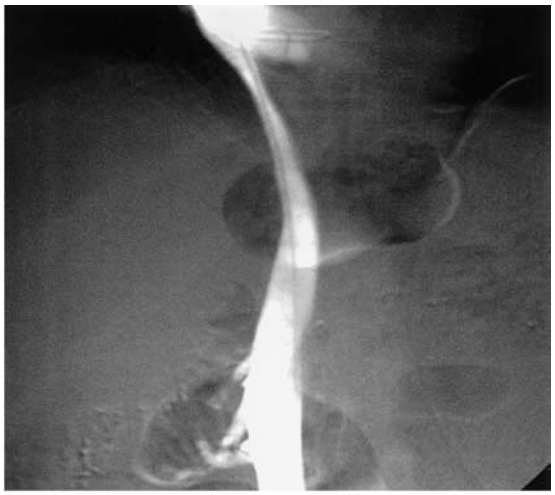
IVC Stenosis or Thrombosis

The prevalence of IVC complications is less than 1% (15,20). Stenosis of the IVC can occur at the anastomosis, but in our experience stenosis at this site is very unusual. Swelling of the graft can result in compression of the IVC (Fig 24), and sometimes a size discrepancy between the donor and recipient IVCs is misdiagnosed as stenosis (7). IVC thrombosis can be caused by surgical problems and hypercoagulable states (Fig 25). The clinical presentation includes pleural effusions, hepatomegaly, ascites, and extremity edema (7). The functional significance is unclear until the pressure gradient across the stenosis is measured and found to be significant (2). Successful balloon angioplasty and stent placement has been reported in IVC stenosis (3).

OLT with preservation of the retrohepatic IVC has gained acceptance during the past few years (10,28) and is commonly used at our hospital.



a. b.



c.

Figure 24. Coronal **(a)** and sagittal **(b)** MPR images show stenosis of the IVC in its retrohepatic course (arrow) due to swelling of the liver graft. **(c)** Correlative angiogram also shows IVC stenosis.

This procedure consists of end-to-side or side-to-side IVC anastomoses or direct anastomosis of the donor IVC with the recipient hepatic veins (the piggyback technique) (9). It avoids the anhepatic phase of standard OLT and obviates the use of venovenous shunts, which are occasionally needed to correct the decrease in venous return to the heart (10). Two types of complications have been associated with this technique: (*a*) hemorrhage (3% of cases) due to hepatic or IVC injury or release of the cavocaval suture (9) and (*b*) poor venous drainage of the graft producing Budd-Chiari syndrome (0.28%–1.5% of cases), which is related to inadequate graft size or faulty surgical technique (9,10).

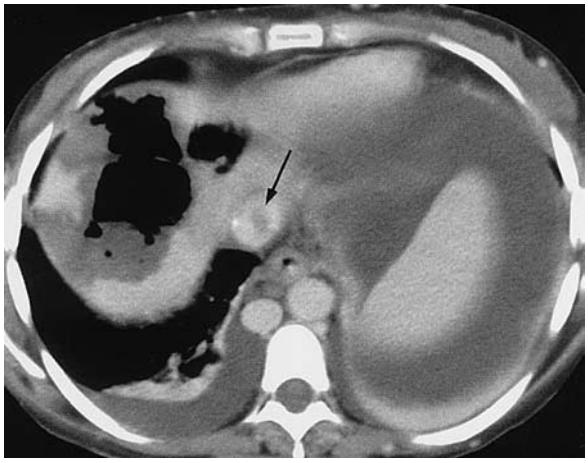
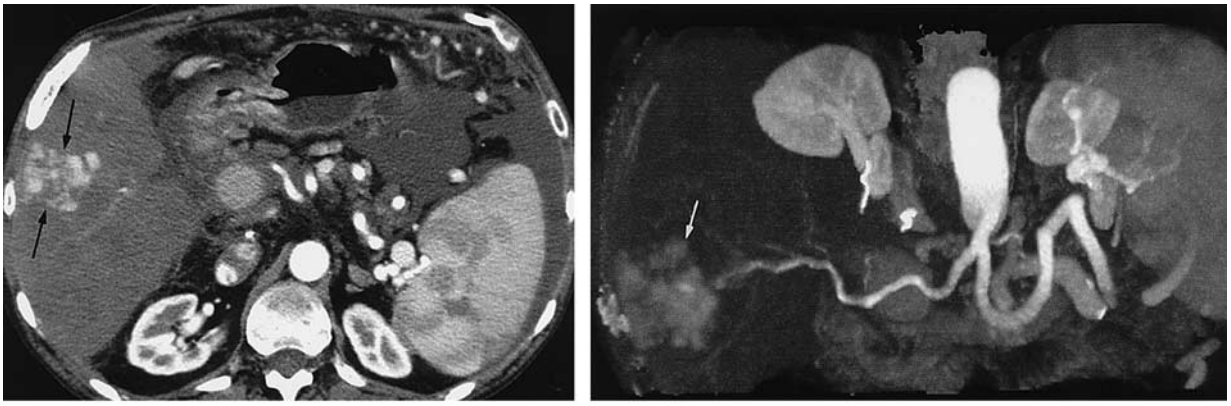


Figure 25. CT scan shows a thrombus in the recipient IVC at the suprahepatic level (arrow) in a patient with thrombosis of the infrahepatic IVC (not shown), hepatic parenchymal ischemia, and infected bilomas. There is marked subcutaneous collateral venous circulation and circulation through the azygos vein system.

Arterioportal Fistula

Intrahepatic arterioportal fistula is a relatively frequent complication of OLT following surgical or percutaneous liver biopsy performed to rule out



a. b.

Figure 26. Helical CT scan **(a)** and maximum-intensity projection reconstruction image (anterosuperior view) **(b)** show a large arterioportal fistula secondary to liver biopsy in segment V (arrows), which is seen as transient hepatic parenchymal enhancement during the hepatic arterial phase.

graft rejection (Fig 26). The helical CT findings of arterioportal fistula include (*a*) early enhancement of peripheral portal vein branches during the hepatic arterial phase and before the main portal vein is enhanced; (*b*) enhancement of peripheral portal vein branches and the main portal vein with nonenhanced superior mesenteric and splenic veins, signs that have been considered diagnostic on hepatic angiograms; and (*c*) transient, peripheral, wedge-shaped, usually straight-margined hepatic parenchymal enhancement during the hepatic arterial phase (29). This last finding usually results from a peripheral arterioportal fistula, which manifests as transient high attenuation due to passage of contrast material from high-pressure arterial blood into a low-pressure portal vein branch, thus enhancing a focal area of the liver before the adjacent parenchyma is enhanced through the portal vein system. The prevalence of arterioportal fistula secondary to liver biopsy is as high as 50% during the first week but drops to 10% after this time, since these shunts tend to close spontaneously.

Biliary Complications

Biliary complications following OLT occur in 6%–34% of cases, most of them within 3 months of transplantation (7). They are the second most common cause of liver dysfunction in OLT patients, exceeded only by rejection (30). Biliary complications include leak, stricture, obstruction, and stone formation. Transplant recipients have external biliary drainage catheters in the postoperative period, so it is fast and easy to perform cholangiography to determine the state of the biliary system when a complication is suspected (13,30). In patients in whom a T-tube is not in place, US and MR cholangiopancreatography are the best noninvasive methods of imaging the biliary tree when compared with forms of direct imaging such as endoscopic retrograde cholangiopancreatography and percutaneous transhepatic cholangiopancreatography (30,31). Helical CT, which is often used to investigate suspected vascular disease, can also demonstrate associated biliary complications, although in a less graphic manner than MR cholangiopancreatography. We include the CT findings of these complications so that they can be recognized and correctly interpreted in CT studies that may be performed for other reasons.

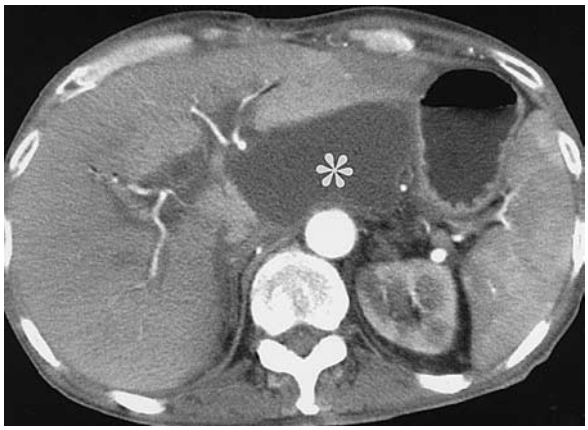
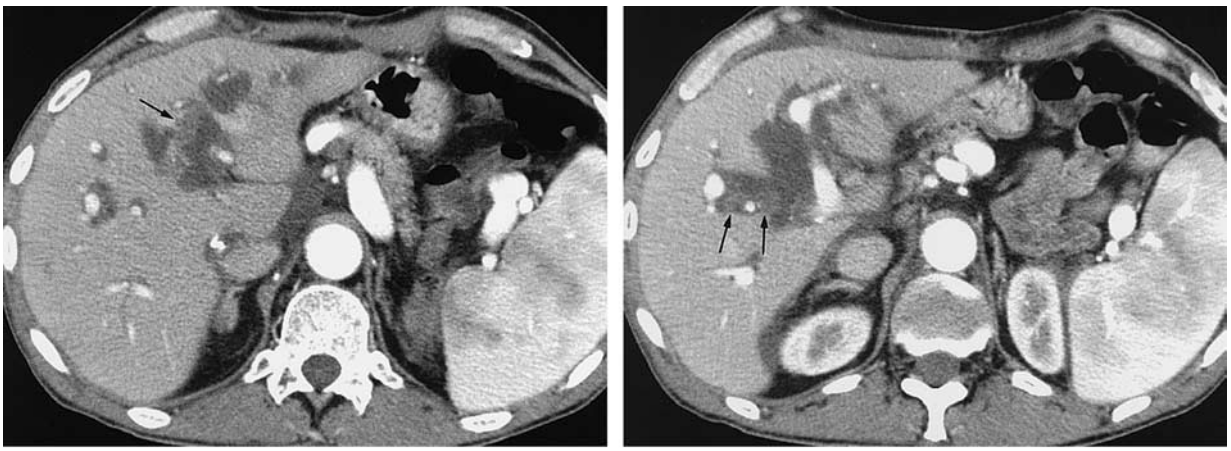


Figure 27. CT scan shows an extensive biloma at the hepatic hilum (*) in a transplant recipient with failure of the end-to-end suture between both common bile ducts.

Bile Leak

Bile leak, the most common complication of OLT in patients with a choledochocholedochostomy, is most often located at the T-tube site and rarely occurs at the anastomosis (Fig 27) (30,32). A small bile leak may close spontaneously or a stent can be placed across the site of leakage (11,32), but surgical revision of the anastomosis is often



a. b.

Figure 28. CT scans show pronounced dilatation of the intrahepatic bile ducts, with multiple intraluminal defects corresponding to biliary sludge lithiasis (arrows).

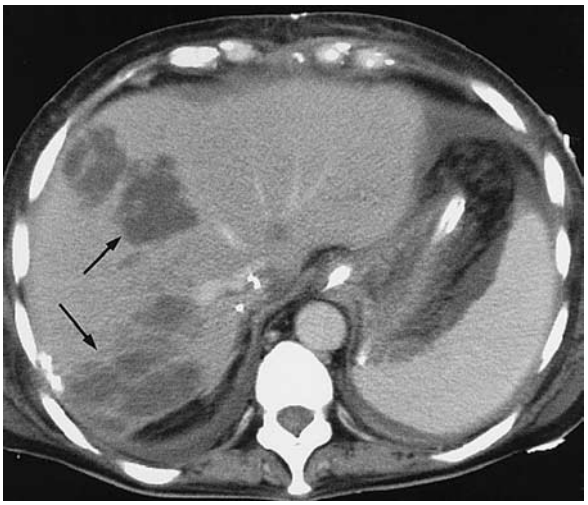


Figure 29. CT scan shows multiple areas of ischemia (arrows) in the right lobe of a patient with arterial stenosis.

necessary. Formation of a bile collection can be treated with percutaneous drainage.

Biliary Stricture

Most biliary strictures occur at the anastomotic site and may be secondary to scar formation that results in retraction and narrowing (4,7). Percutaneous dilation can be performed, although repeat surgery is occasionally required (11). Nonanastomotic strictures are probably caused by ischemia due to hepatic artery stenosis or thrombosis or preservation injury. If a biliary stricture is suspected and CT shows no dilatation, endoscopic retrograde or percutaneous transhepatic cholangiography should be performed, since many liver transplants do not develop bile duct dilatation even in high-grade stenosis.

Bile Duct Ischemia

Since the bile duct is entirely dependent on hepatic artery blood supply, nonanastomotic bile leaks and biliary stenosis can be caused by bile duct ischemia due to arterial stenosis or thrombosis (2–4,11,14,33,34). These strictures may respond to dilation, although they need frequent dilation procedures and long-term biliary drainage. Retransplantation is often necessary. Other causes of nonanastomotic bile leaks include prolonged cold ischemia and chronic ductopenic rejection leading to bile duct necrosis. Bilomas can be treated with percutaneous drainage, prolonging graft survival, but if the hepatic artery is thrombosed (almost 90% of cases) retransplantation is needed (34). Biliary strictures secondary to ischemia often start at the hilum and progress to the intrahepatic bile ducts (4,7), although ductal dilatation may be the only CT finding (Fig 28). Intrahepatic biliary strictures can also be due to recurrent sclerosing cholangitis (11,33); thus, when a peripheral biliary stricture is detected in these patients and helical CT shows a normal hepatic artery, recurrence should be suspected.

Less common complications include sphincter of Oddi dysfunction and biliary obstruction due to kinking in a redundant common bile duct or to stones or sludge caused by alterations in bile composition (7,11,14). Mucocele of the cystic duct remnant is a rare complication resulting from ligation of the cystic duct both proximally and distally. It is seen as a round fluid collection that can compress the common bile duct, producing obstruction (11,12).

Liver Ischemia or Infarction

Areas of liver ischemia or infarction are seen at CT as wedge-shaped, low-attenuation peripheral lesions (Figs 29, 30) (2,12). The larger areas of infarction may liquefy, become infected, and occasionally calcify (13). Focal abscesses within the infarcted areas of the liver can cause intermittent

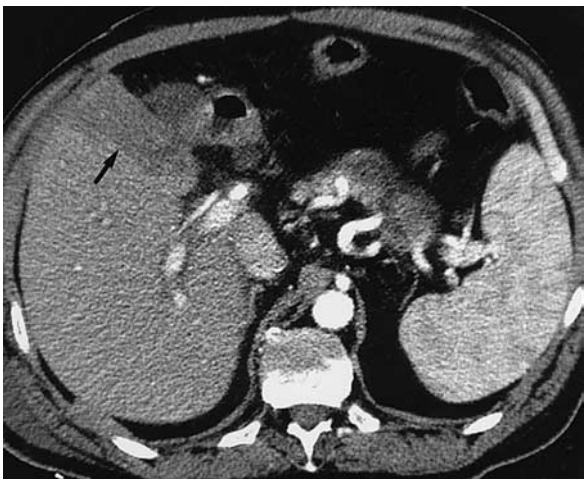


Figure 30. CT scan shows segment IV ischemia (arrow) due to absence of arterial vascularization in the graft. Segments II and III were used in transplantation for a child, and segment IV was not resected. Follow-up CT showed marked atrophy of this segment.

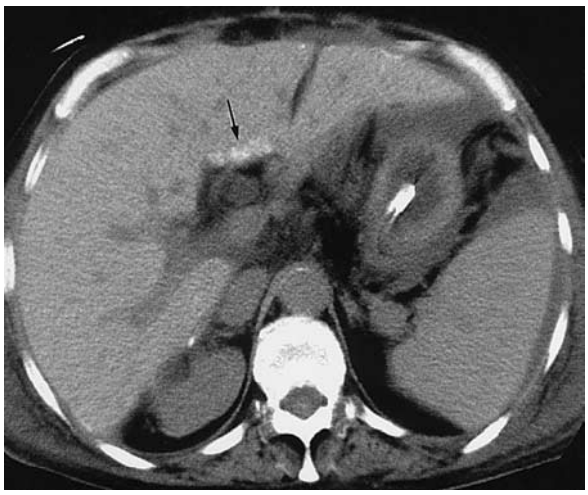
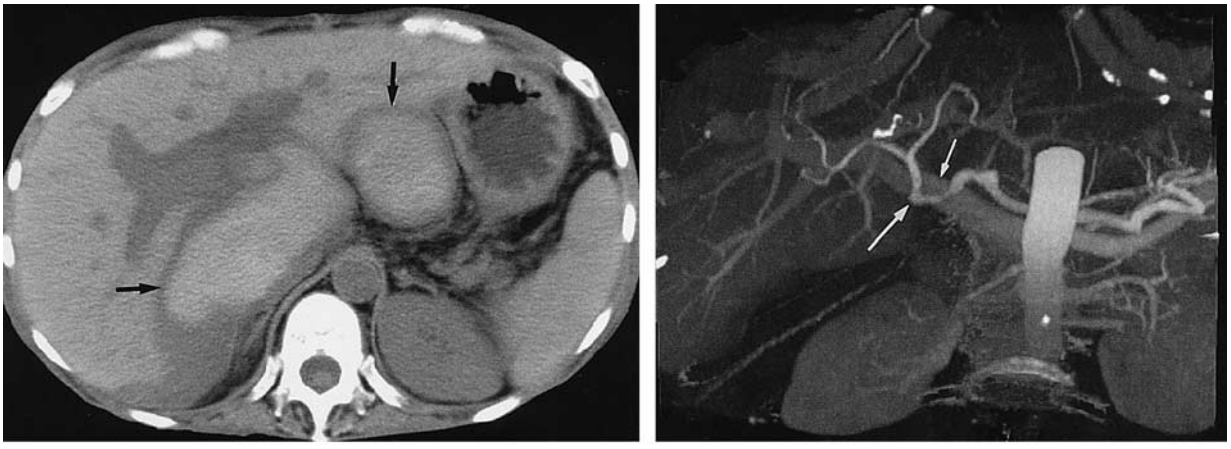


Figure 31. CT scan shows small peripheral calcifications (arrow), probably over ischemic preservation lesions.



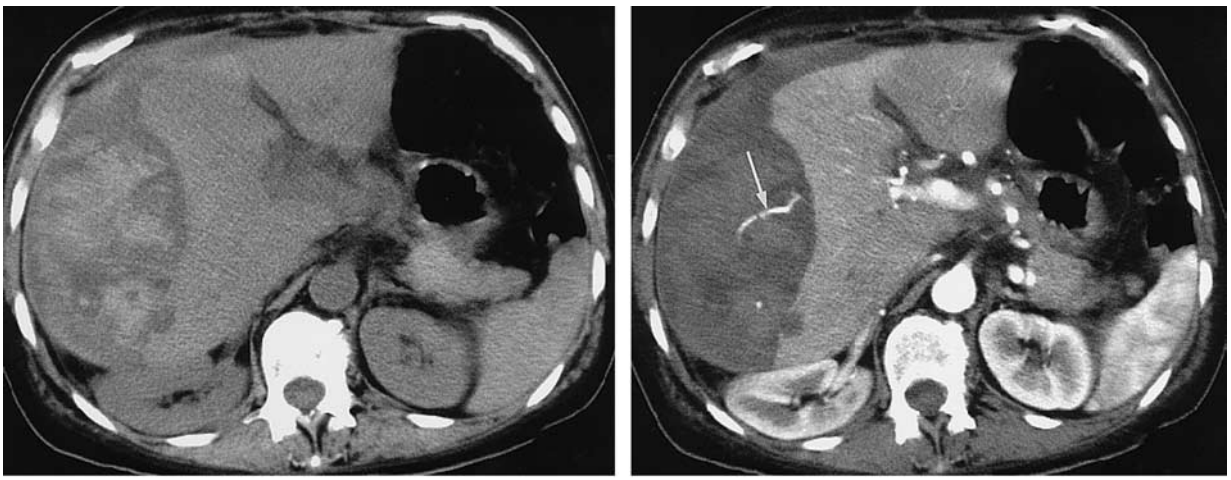
a. **b.**

Figure 32. **(a)** CT scan shows extensive peri- and retrohepatic hematomas (arrows). **(b)** Maximum-intensity projection reconstruction image (anteroinferior view) shows that the hematomas do not impede evaluation of vascular structure patency. Large arrow = hepatic artery, small arrow = portal vein.

episodes of sepsis. Puncture of the ischemic areas is sometimes necessary to rule out superinfection; when ischemia is confirmed, percutaneous or surgical drainage with excision of the necrotic tissue is performed. Most cases of liver ischemia or infarction are due to vascular problems involving the hepatic artery (85% of cases) or, less frequently, the portal vein. In these cases, besides evaluation of the hepatic parenchyma (bilomas, infarction, abscesses), helical CT can be used to visualize possible complications involving vascular structures (stenosis, thrombosis). Extensive parenchymal and bile duct necrosis can lead to graft failure and require retransplantation. In the postoperative period, one may encounter hypoattenuating areas of ischemia due to preservation lesions, which usually resolve within a few weeks. Occasionally, small residual calcifications can be observed in the hepatic parenchyma (Fig 31), particularly in patients with renal failure or abnormalities of calcium-phosphorus metabolism.

Fluid Collections and Hematomas

Fluid collections and hematomas are frequent in the areas of vascular anastomosis (hepatic hilum and adjacent to the IVC) and biliary anastomosis, as well as in the lesser sac, surrounding the ligamentum teres, and in peri- and subhepatic spaces (4,12). These usually resolve over several weeks, although they are sometimes large enough to produce IVC or portal vein compromise. Hematomas are relatively easy to differentiate from fluid collections (seromas, bilomas, abscesses) because of their higher attenuation (Figs 32, 33) (12).



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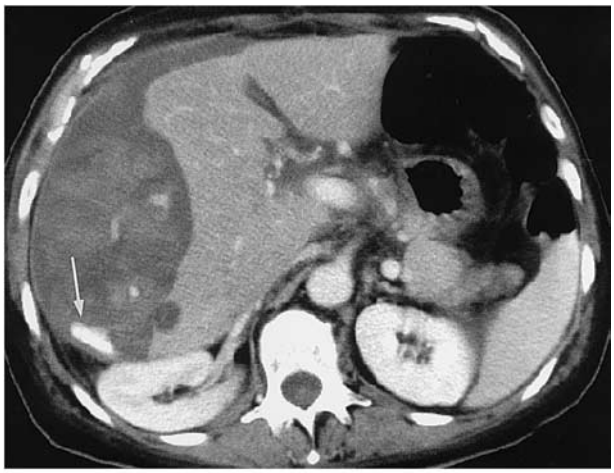

c.

Figure 33. **(a)** Nonenhanced CT scan shows a large, hyperattenuating subcapsular hematoma, which was secondary to a liver biopsy performed to evaluate the state of the graft. **(b)** Hepatic arterial–phase helical CT scan shows active bleeding (arrow). **(c)** Portal venous–phase helical CT scan shows extravasation of intravenous contrast material within the hematoma (arrow).

They can be caused by problems with vascular anastomoses, rupture of a hepatic artery pseudoaneurysm, or intraperitoneal bleeding after liver biopsy (surgical or percutaneous) to evaluate rejection (Fig 33) (13). The clinical picture, a patient who suddenly becomes hypotensive, and the descent in hematocrit are usually diagnostic. CT is useful for confirmation of doubtful cases and sometimes helps determine the focus of bleeding. Open surgical revision of the transplant is generally required in these cases; however, in postbiopsy intrahepatic arterial injury, endovascular embolization may be an alternative. Aspiration and culture of the fluid collections may be necessary to rule out superinfection. When superinfection occurs, it can be treated with percutaneous drainage.

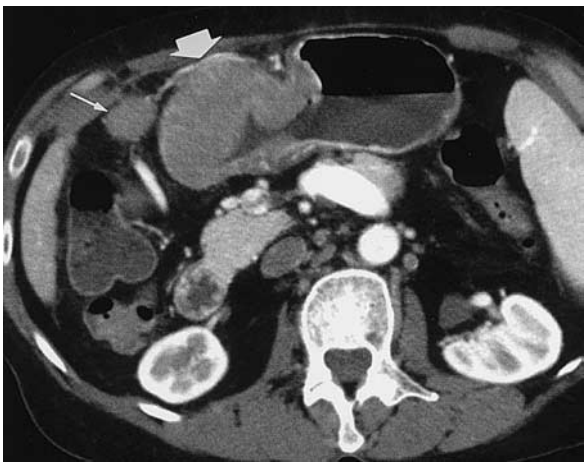

Figure 34. CT scan shows gastric lymphoma (thick arrow) with regional lymph nodes (thin arrow) in a liver transplant recipient undergoing immunosuppressive treatment with cyclosporine.

Malignancy

OLT patients are at increased risk for developing malignancy, especially non-Hodgkin lymphoma (Fig 34) and squamous cell skin cancer, because of the immunosuppressive therapy administered to avoid graft rejection (3,7). Lymphoma, which is more frequent in patients treated with cyclosporine, can involve any organ, including the liver

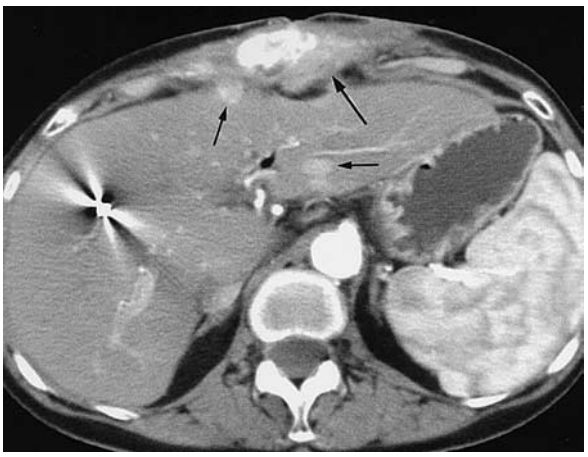

Figure 35. CT scan shows recurrence of hepatocellular carcinoma in the abdominal wall (large arrow) and liver graft (small arrows).

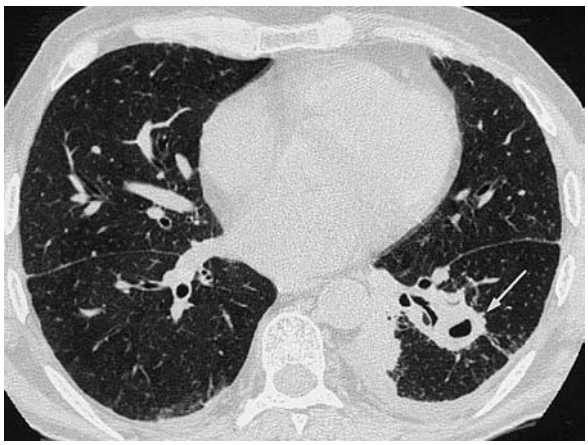

Figure 36. CT scan shows cavitated lung infiltrates (arrow) in an OLT patient with a lung infection due to *Aspergillus*.

graft parenchyma itself, where it is seen as multiple hypoattenuating nodules (3,12). Other common features include lymph node enlargement and extranodal involvement (spleen, small intestine, stomach, kidney, mesentery, and adrenal glands) (4). Epstein-Barr virus has been associated with posttransplantation lymphoproliferative disease and lymphoma in patients treated with cyclosporine (3,4,35), and several studies have also demonstrated that lymphoproliferative disorders are more frequent in transplant recipients with hepatitis C virus (36). Diagnosis is complex in cases of lymphoma with hepatic hilum involvement (37), since this area tends to show marked postoperative changes, making identification of small nodules difficult; although lymph nodes can be identified, they are not an uncommon finding and are often reactive (4).

In patients with a neoplasm treated with OLT (hepatocellular carcinoma, hepatic metastases of neuroendocrine tumors, or cholangiocarcinoma), the primary tumor can recur in the graft or at any other location (Fig 35). The most common site of recurrence of hepatocellular carcinoma is the lung, followed by the liver graft (4). Since the recurrence rate is very high in cholangiocarcinoma (13), liver transplantation is rarely performed in this pathologic condition. Finally, keep in mind that transplant recipients can develop any type of neoplasm, as in the general population.

Other Complications

Cirrhosis due to hepatitis C virus infection is now the most common indication for OLT in Western Europe and the United States. After OLT, there is persistence of viremia and reinfection of the liver; without effective prophylaxis, progression of graft disease is almost inevitable (38). Although helical CT does not allow evaluation of this aspect, it can reveal the morphologic changes produced in the graft when cirrhosis develops.

Splenic infarction can occur in OLT patients, but it is of no clinical significance unless infection ensues (13).

Patients with cirrhosis and portal hypertension are at increased risk for developing splenic artery aneurysms (7%–10% of cases), due mainly to a high flow rate in the splenic artery (Fig 17). These patients are also at higher risk for splenic artery aneurysm rupture in the posttransplantation period, especially if the aneurysm is larger than 1.5 cm in diameter (39), owing to decreased portal vein pressure and increased splenic artery flow, which may cause splenic artery aneurysms to expand and rupture (40,41). Preoperative study is necessary to detect splenic artery aneurysms, since this area is not routinely explored during transplantation surgery. If a splenic artery aneurysm is found, ligation of the artery should be performed at the time of transplantation to prevent possible rupture.

The diagnosis of acute rejection, one of the most serious complications of OLT, is established with graft biopsy and histologic study (3). The role of imaging methods consists of excluding the other complications described herein, which can have clinical signs and symptoms similar to those of acute rejection.

OLT patients are immunocompromised and prone to bacterial and opportunistic infections (42). Among them, tuberculosis, cytomegalovirus infection, and *Aspergillus* infection are not infrequent in our setting, whereas *Pneumocystis carinii* pneumonia is a rare type of infection. Nonenhanced CT of the chest could be very helpful in diagnosing early lung infections in these immunocompromised patients (Fig 36).

Conclusions

Helical CT is a valuable technique for evaluation of OLT patients. When vascular complications are suspected and US results are indeterminate, it can avoid the need for diagnostic angiography. Helical CT also reveals abnormalities of the hepatic parenchyma and, to a lesser extent, the bile ducts and allows evaluation of extrahepatic tissues.

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