Pediatric Liver Transplantation

Part II. Diagnostic Imaging in Postoperative Management[1]

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The postoperative diagnostic imaging examinations of 44 children who underwent 59 orthotopic liver transplantations were reviewed. The imaging modalities used for the evaluation of suspected complications include plain roentgenography, ultrasonography (US), computed tomography (CT), nuclear scintigraphy, arteriography, percutaneous and operative cholangiography, and endoscopic retrograde cholangiopancreatography. The main postoperative complications included ischemia, thrombosis (hepatic artery and portal vein), infarction, obstruction or leakage of the biliary anastomosis, hepatic and perihepatic infection, and allograft rejection. US, the most frequently used abdominal imaging modality, was best suited for detection of biliary duct dilatation, fluid collections in or around the transplanted liver, and hepatic arterial, inferior vena caval, and portal vein thrombosis. CT was especially helpful in corroborating findings of infection and in locating abscesses. Technetium 99m sulfur colloid (early- and late-phase imaging) provided a sensitive, although nonspecific, means of assessing allograft vascularization and morphology. Angiography showed vascularity most clearly, and cholangiography was the most useful in the assessment of bile duct patency. A diagnostic imaging algorithm is proposed for evaluation of suspected complications.

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THE resurgence of pediatric liver transplantation has been due to refinement in operative techniques, to better understanding of the body's immune system (1–5), and to the recent development of cyclosporine as a successful immunosuppressor of allograft rejection (6). Concomitantly, there has been an increase in survival, a greater awareness of early postoperative complications, and consequently, more aggressive intervention (7, 8). The diagnostic imaging modalities of computed tomography (CT), ultrasonography (US), nuclear scintigraphy, angiography, and percutaneous cholangiography are important in helping to determine the life-threatening, medically or surgically related complications (9–12). Common medical complications include hypertension, overhydration pulmonary edema, and cyclosporine-induced renal dysfunction. Infarction caused by deficient organ preservation, ischemia, hepatic arterial or portal venous thrombosis, obstruction or leakage of the biliary anastomosis, and rejection or graft infection have been the major postoperative surgical complications (13–15). Allograft rejection and graft infection are the most life threatening of all the complications.

Fever, nonspecific symptoms, and laboratory test results showing hepatic dysfunction are indicators of hepatic allograft abnormality. Recognition of allograft abnormality must be prompt, and a specific diagnosis must be reasonably accurate. Each complication merits specific treatment, and the prognosis for each is variable. For example, acute rejection requires increased doses of immunosuppressive drugs, while allograft infarction or vascular thrombosis necessitates hepatic retransplantation. To avoid the increased risk of sepsis, however, immunosuppression should be enhanced with caution when hepatobiliary reconstruction has been disrupted or when allograft ischemia is present.

Thus, from a retrospective review of diagnostic imaging methods used at the Children's Hospital of Pittsburgh for hepatic-allograft failure, we present an algorithm for standardization of postoperative liver transplantation imaging (Fig. 1). Preoperative aspects have been addressed in Part I (16).

MATERIALS AND METHODS

In the 22 months from May 1981 to March 1983, 44 children at our institution underwent a total of 59 orthotopic liver transplantations. Review of clinical data and diagnostic imaging examinations of these patients yielded the following information.

Clinical

There were 28 girls and 16 boys whose ages ranged from 7 months to 18 years. Many of these patients had been referred to our facility from other medical centers with the diagnosis of terminal liver disease. The most common causes of end-stage hepatic disease in this series were biliary atresia and inborn metabolic disorders (Table 1). Seventeen of 19 patients with biliary atresia had undergone Kasai operations. Three of 13 patients with metabolic disorders presented with superimposed liver neoplasms.

Twelve children each underwent two liver transplantations, and two other children each underwent three. Twenty-seven children were alive at the time of this review, which represents a maximal follow-up of 24 months (Table 1).

Diagnostic Imaging

Abdominal US was performed with a real-time sector scanner using 3.0- or 5.0-MHz transducers. CT examinations of the abdomen were performed with contrast-filled bowel and enhancement of other organs with contrast medium injected intravenously. Wide-field gamma camera technetium 99m (Tc-99m) sulfur colloid scintigraphy (early blood flow and delayed phases), Tc-99m PIPIDA hepatocyte derivative scintigraphy, and gallium 67 (Ga-67) scintigraphy were the types of radionuclide examinations done. Celiac axis arteriography was done when US findings were suggestive of vascular thrombosis and also to determine the extent of occlusion and the presence of collateral circulation. T-tube or percutaneous transhepatic cholangiography was performed when US showed biliary dilatation in patients with clinical signs of biliary obstruction or leakage. When biliary leakage was suspected in patients with no US evidence of biliary dilatation, endoscopic retrograde cholangiopancreatography (ERCP) was performed.

RESULTS

Five patients had no complications. Complications were multiple in 39 patients (each complication in the total number of 59 orthotopic liver transplantations did not necessarily represent a single patient).

Vascular Compromise

Vascular insufficiency of the hepatic allograft developed in 12 children (20% of 59 transplantations) (Table 2). Four patients also had hepatic artery thrombosis, three had portal vein thrombosis, and one had thrombosis of both vessels followed by hepatic necrosis. Four of the 12 children underwent a second transplantation, and three others died just prior to a second transplantation (one from hyperkalemia and the other two from hepatic insufficiency). The clinical presentation was that of hepatic failure caused by hepatic infarction with superimposed infection and/or abscess. The ischemic areas were represented by decreased radionuclide uptake on Tc-99m sulfur colloid liver scintigraphy, by increased echogenicity on sonograms, or by decreased attenuation within the hepatic parenchyma on CT scans.

US was useful in the initial demonstration of portal vein thrombosis in two children and of portal anastomotic stenosis in another. These results were corroborated with angiography and proved either at biopsy or surgery. One of these patients had an associated thrombosis of the inferior vena cava (Fig. 2). The angiographic finding of interruption of the hepatic artery was present in one patient.

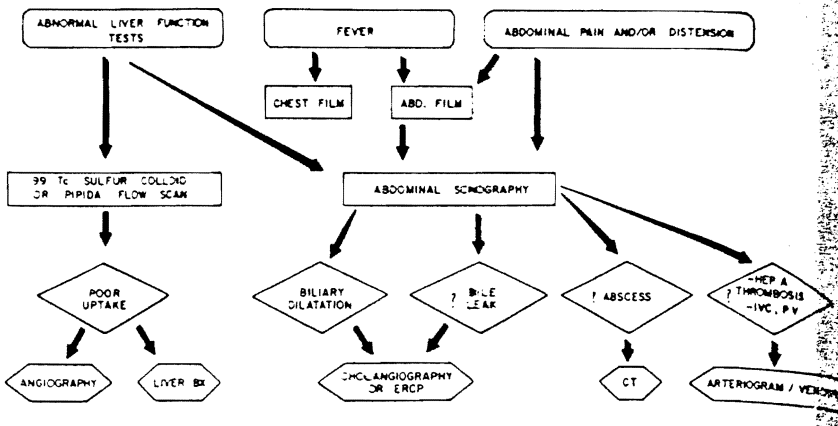

Figure 1. Algorithm summarizing the sequential use of the various imaging modalities when one or multiple problems are encountered. *IVC* = inferior vena cava, *PV* = portal vein, *ERCP* = endoscopic retrograde cholangiopancreatography, *PTC* = percutaneous transhepatic cholangiography.

Table 1
Patient Data

Diagnosis	No. of Patients	Survivors	Follow-up (mo)
Biliary atresia	19	12	1–25
Inborn error			
Alpha-1-antitrypsin deficiency	8	6	1–14
Wilson disease	2	1	22
Tyrosinemia with hepatoma	1	1	17
Type I glycogen storage disease with adenomas	1	1	18
Sea-blue histiocytes with hepatoma	1	1	15
Biliary hypoplasia	2	0	1–11
Chronic active hepatitis	3	1	1–12
Neonatal hepatitis	2	1	1–19
Familial intrahepatic cholestasis	1	1	23
Secondary biliary cirrhosis	1	0	1
Byler syndrome	1	1	10
Pseudoinflammatory tumor	1	1	6
Status after liver transplantation for Budd-Chiari syndrome	1	0	20
Total	44	27	

Biliary Tract Complications

Ten children (17% of 59 transplantations) required a second operation because of biliary complications: five for biliary obstruction and four for biliary fistula. The necrosis of the donor common bile duct in one patient caused an abscess that ruptured into the portal vein. Exsanguination curtailed the operative attempt to drain the abscess. Despite operative intervention, ascending cholangitis caused death in one patient. A higher incidence of complications was noted in patients with choledocho-ch... chostomies (eight of 19 patient... than in those with choledocho... ostomies (two of 31 patients; 6%...

Cholangiography was the p... ble diagnostic modality in this ... although US was performed in... in four of these patients. US fi... were dilatation of biliary radic... some cases of obstruction (Fig. 3... loculated fluid with bile leaka... others. Bile leakage was confi... with T-tube cholangiography, ... hepatic cholangiography, or ... (Fig. 4).

Hepatic Infections

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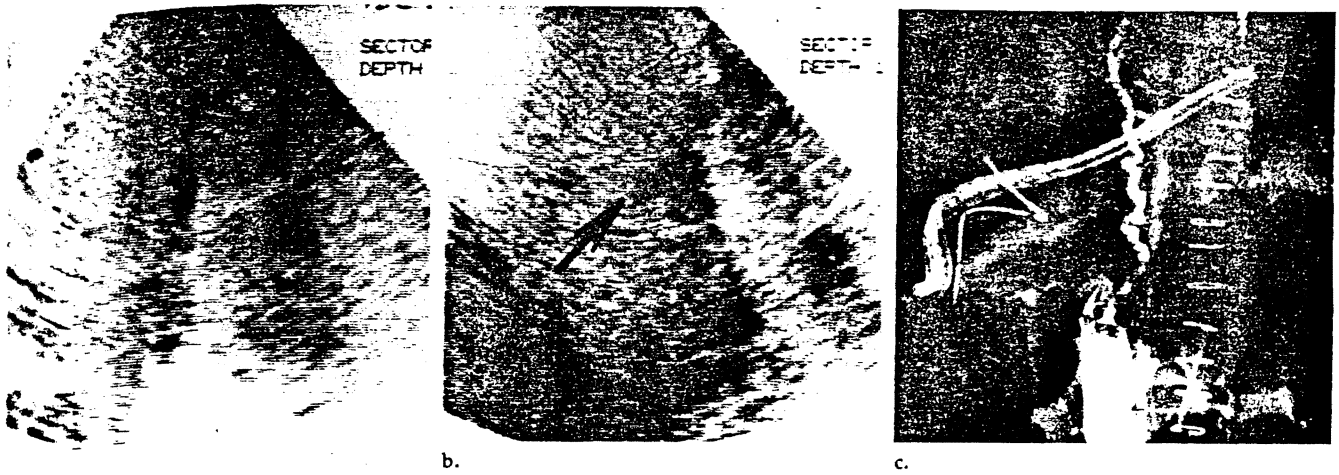


Figure 2. Sagittal (a) and oblique (b) sonograms of the liver with lack of visualization of the portal vein in a (arrow) and a clot in the distal extrahepatic inferior vena cava in b (arrow) in a child with acute hepatic failure. Confirmatory selective angiography of the hepatic artery and superior mesenteric artery did not show the opacification of the portal vein in the venous phase. (c) Cavogram shows the obstruction of the inferior vena cava, first suggested by US findings.

Table 2
Vascular Complications

Nature of Hepatic Failure	Diagnostic Procedure (findings)	Follow-up
Hepatic infarction, right hepatic artery thrombosis with concomitant abscess	Nuclear medicine, cholangiography	Retransplantation
Portal stenosis	US, angiography, venography	Reoperation
Necrotic donor common duct, hepatic abscess, hepatic artery thrombosis	US, nuclear medicine, CT, cholangiography	Exploration of intrahepatic abscess, hemorrhage, death
Allograft infarction due to pulmonary arteriovenous shunt	Nuclear medicine	First graft: graft hypoxia; second graft: graft hypoxia; third graft: graft hypoxia, cerebral hemorrhage, death
Extrahepatic portal thrombosis	Superior mesenteric angiography, transhepatic portography, US, gastrointestinal tract series (varices)	Sclerotherapy; liver function tests normal 1 year posttransplantation
Allograft ischemia due to previous portal vein thrombosis		Retransplantation, cerebral hemorrhage, death
Total allograft infarction due to mechanical compression of hepatic artery		Liver failure, death
Hepatic artery thrombosis	Abdominal film (aerobilia)	Hyperkalemia during retransplantation, cardiac arrest
Extrahepatic portal vein and infrahepatic vena cava thrombosis	US, angiography	Retransplantation
Hepatic artery thrombosis due to septic thrombovasculitis	Abdominal film (aerobilia), arteriography	Retransplantation, ischemic liver injury, septic shock, death
Multiple allograft infarctions	Nuclear medicine	Retransplantation
Thrombosis and fungal overgrowth of hepatic artery and portal vein	Laparotomy with biopsy	Retransplantation, disseminated candidiasis, death

identified with US and corroborated with hepatobiliary scintigraphy and/or CT. CT was useful not only for abscess localization but also to facilitate percutaneous drainage around interposed bowel.

Other Complications

Other complications developed in 30 patients (51% of 59 transplantations), including ten patients with complications involving the gastrointestinal tract (17% of 59 transplantations). A mycotic aneurysm developed at the site of a hepatic artery anastomosis in one child. The aneurysm ruptured and caused exsanguination. At necropsy, blood was found in the peritoneal cavity and within the bowel lumen. In four other children, massive postoperative intraabdominal bleeding occurred and required immediate exploratory surgery. A hematoma developed in one of the four, which compressed the hepatic artery and caused complete necrosis of the transplanted liver.

Melena was discovered 1 year after liver transplantation in one child with esophageal varices. A sclerosing procedure of the esophageal veins controlled the bleeding. Results of selective superior mesenteric arteriography and transhepatic portography confirmed the presence of extrahepatic portal vein thrombosis. Following sclerotherapy for varices, this child has done well, and results of liver function tests have been normal.

Acute pancreatitis occurred in one child after transplantation, and this episode was followed by formation of a pancreatic pseudocyst during the early postoperative period. The pseu-

...o postischemic injury. In two children the hepatic infection was secondary to vascular complications, which developed in necrotic allografts from postportal thrombosis, arterial occlusion, and vasculitis. In two patients with ascending cholangitis, the cholangiographic finding was biliary tract dilatation, but results of a liver biopsy established a diagnosis of abscess, and we performed operative drainage.

The abdominal roentgenographic finding of intrahepatic gas was present in two patients, both of whom died from anaerobic infection secondary to arterial thrombosis.

In general, hepatic abscesses were

docyst was identified on sonograms but was more accurately located and drained using data from CT. In one child, bowel obstruction characterized by acute abdominal distention was produced by cyclosporine-induced lymphoma 6 months after liver transplantation. At present, this child is alive and free of overt lymphoma following interruption of cyclosporine therapy and subsequent readjustment of the dosage of cyclosporine to a minimal level.

An intestinal fistula (at the allograft/recipient choledocho-cholecysto-jejunostomy) developed in another child, requiring operative correction. Diagnosis of the fistula was made with the aid of the US finding of loculated fluid between the hepatic hilus and the small bowel. Two intraabdominal abscesses were diagnosed with US, one secondary to the fistula described above and the second in the subphrenic area, which required operative drainage.

Chest roentgenography demonstrated 20 respiratory complications: three cases of barotraumatic phenomena (mainly pneumomediastinum or pneumothorax), 14 patients with transient pulmonary edema (ten with pleural effusion), two with opportunistic pulmonary fungal infection, and one with aspiration pneumonia.

One case of cyclosporine nephrotoxicity (1%) was diagnosed by the scintigraphic finding of decreased renal excretion of Tc-99m MDP.

Rejection

In our series of 44 children, irreversible rejection developed in seven (16%) after their first transplantations (44 transplants). Six of these children underwent a second transplantation; the seventh died while waiting for a second hepatic donor. Although rejection or a second process, such as infection, might have presented a similar clinical picture, liver biopsy has been the specific test to prove that the allograft was being rejected. All of these patients had normal results of abdominal CT and US examinations. Tc-99m sulfur colloid scintigrams were obtained for four patients, two of which showed normal hepatic parenchyma, while the scintigrams of the other two showed decreased hepatic radionuclide uptake, which was consistent with, but not diagnostic of, rejection.

DISCUSSION

Complications

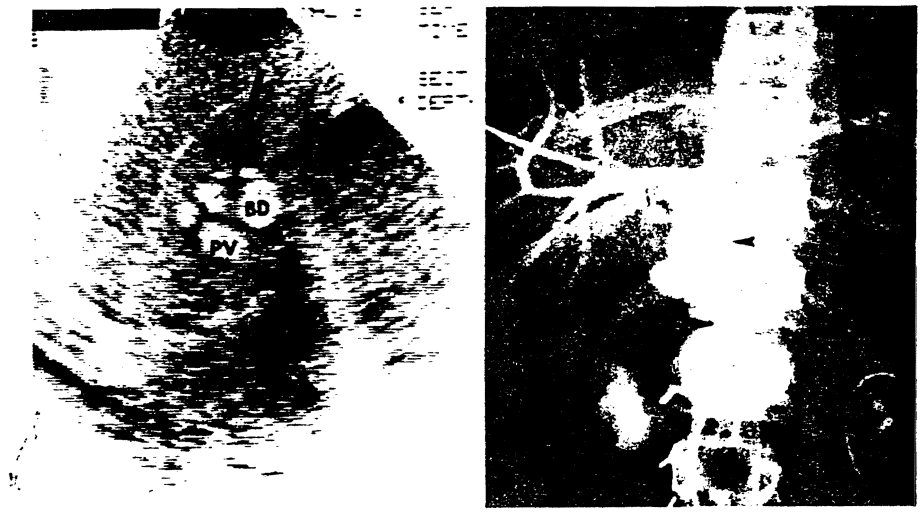
Complications occurring immediately after liver transplantation can be very serious and must be carefully watched for. Each of the following complications is uniformly lethal: total occlusions of both the hepatic artery and portal vein and an irreversibly damaged or devascularized organ secondary to deficient preservation. Attempted transplantation prior to discovery of absent superior vena cava or absent intrahepatic segment of the inferior vena cava (16–18)—dreaded complications—might also be included as "complications" (Table 3). Death usually occurs quickly from massive hepatic necrosis when any of these complications is present. Retransplantation represents the only means of allowing these patients to live. Therefore, early recognition of these complications is mandatory.

Regional hepatic infarction, necrosis, and sepsis may develop in patients with partial vascular occlusion or isloated thrombosis of the hepatic artery via passage of intestinal microorganisms through the biliary anastomosis. Among early complications of liver transplantation, sepsis is one of the most frequent causes of death (Table 3). This outcome is even more likely to occur in immunosuppressed patients. In other words, hepatic or perihepatic abscess usually follows a more serious cause of allograft deterioration (e.g., uncontrolled hepatic rejection and bacterial colonization) as shown by clinical experimentation and laboratory research data (3). Abscess represents the "tip of the iceberg" and warrants further investigation. The problems that may arise from the reconstruction of the biliary duct—obstruction and fistula—have been other important sources of infection in patients already immunosuppressed (19).

The lymphoproliferative growth and renal toxicity secondary to cyclosporine therapy may be reversed by decreasing the dose of the immunosuppressive agent (20–23), as was evident in this series.

Diagnosis

An algorithm is presented for expeditious diagnostic imaging of the most commonly encountered clinical problems in the period immediately after liver transplantation (Fig. 1). In the early detection of posttransplantation complications, serial biochemical, bacteriologic, and immunologic tests may contribute corroborative



a.

b.

Figure 3. (a) Longtudinal sonogram of the liver shows the dilated bile duct (*BD*) with the metallic stent (arrow) within it. The portal vein (*PV*), posterior to the bile duct, is patent. (b) Percutaneous transhepatic cholangiogram of the same liver transplant recipient with a balloon (arrow) dilating the stenosis at the site of biliary reconstruction. Arrowhead shows prestenotic dilatation of anastomosed duct.

Figure 4. Image from ERCP shows main branches of the biliary tree and patic intraperitoneal loculated fluid beled vertical arrow). *PD* = pancreatic *CBD* = common bile duct.

findings that are equally important in the diagnosis and management of each complication.

Liver function tests, along with abdominal US, are the best early indicators of allograft dysfunction. Abnormal liver function tests may reflect parenchymal dysfunction caused by vascular compromise and/or rejection or biliary duct malfunction from leakage, obstruction, or infection. Abdominal US offers an easy method of examining the transplanted liver in the immediate postoperative period, even at the bedside. Patency and size of the portal vein, inferior vena cava, hepatic artery, and biliary anastomoses can be monitored, and the amount of perihepatic postoperative fluid can be determined. Loculated fluid may represent biloma, hematoma, or abscess. Fluid within thick walls is even more suggestive of abscess. Loculated fluid in the falciform ligament, however, should not be considered an abscess (16, 24). Parenchymal mixed hyperechogenicity suggests hepatic infarction.

Doppler US, if available, may be used to confirm vascular patency and may obviate the need for angiography (12, 25). Doppler US was not used in the 59 transplantations in 44 pediatric patients in this series. If hepatic infection or ischemia was suspected clinically or by US, hepatic scintigraphy, angiography, and/or dynamic CT were performed. An intravenous bolus of contrast medium with angiography or dynamic CT, or Tc-99m sulfur colloid scintigraphy was used to evaluate parenchymal perfusion or vascularity of the new liver. In evaluating liver transplantation complications, these methods continue to be accurate.

Tc-99m PIPIDA liver scanning is also a valuable screening modality for the assessment of hepatobiliary function. Hepatobiliary scintigraphy also may be used in the evaluation of biliary obstruction, leakage, or abscess. However, the nonspecificity of the results and the urgency to establish a definitive diagnosis have limited the frequent use of radionuclide scintigraphy in our patients. In this series, when acute hepatic deterioration ensued, arteriography was the definitive procedure for the diagnosis of thrombosis—the main cause of liver anoxia and its complications. One can confirm the degree of residual obstruction and evaluate the extent of collateral circulation using angiography.

When persistent or progressive fever and sepsis develop, and if the abdominal viscera are not involved, the lung is the next most common focus of infection. In addition to the possibility of pneumonia and/or atelectasis, chest radiographs should bring attention to the possibilities of pulmonary edema, pleural effusion, and air-leak phenomena in the immediate postoperative period. Edema and effusion may occur because of the enormous shift of fluids as a consequence of the newly implanted liver.

Careful inspection of the portable abdominal radiograph for aerobilia—a sign of acute hepatic failure—is especially important. Intrahepatic gas, pneumatosis intestinalis, or a mass suggest anaerobic infection or abscess formation. Pneumoperitoneum is not an unusual finding in patients who have just undergone transplantation, or in patients with indwelling drainage tubes or open operative wounds. In patients with abdominal pain or distension, signs of intestinal obstruction, ascites, or an unusual location of the metallic biliary stent should be looked for on radiographs.

When the diagnostic imaging examinations, including angiography and Tc-99m sulfur colloid scintigraphy, are normal or nonspecific, needle or intraoperative biopsy is the definitive procedure to exclude or diagnose liver rejection (5, 14). There are no specific diagnostic imaging findings of rejection.

Table 3
Major Causes of Death

Age (yr)	Sex	Disorder before Transplant	Major Cause of Death	Interval between Surgery and Death (mo)
4	M	Alpha-1-antitrypsin deficiency disease	Hemorrhage during exploration of intrahepatic abscess	2
18	M	Alpha-1-antitrypsin deficiency disease	First graft: hypoxia due to pulmonary AV shunt; second graft: hypoxia, rejection; third graft: hypoxia, cerebral hemorrhage	4
2⅔	M	Biliary hypoplasia	First graft: chronic rejection; second graft: moderate rejection, staphylococcal sepsis	12
2	F	Biliary atresia; Kasai operation	Chronic rejection, liver failure, sepsis	4
8	F	Secondary biliary cirrhosis, choledochal cyst, portal vein thrombosis	First graft: necrosis; second graft: cerebral hemorrhage	1
16	F	Budd-Chiari syndrome	First graft: chronic rejection; second graft: acute rejection; third graft: liver failure, renal failure	20
3	M	Biliary hypoplasia, absent hepatic artery, hypoplastic portal vein	Sepsis, ascending cholangitis	1
18	M	Chronic agressive hepatitis	First graft: chronic rejection; second graft: disseminated aspergillosis	4
6	F	Biliary atresia, Kasai operation, absent inferior vena cava, malnutrition	Total hepatic infarction due to mechanical compression	1
2½	F	Biliary atresia, Kasai operation, absent superior vena cava with innominate drainage into inferior vena cava	Severe cerebral edema, perioperative brain death	1
17	M	Chronic aggressive hepatitis	Hemorrhage, ischemic infarction of the liver	1
3½	F	Biliary atresia, Kasai operation	First graft: necrosis, fungal overgrowth; second graft: gastrointestinal bleeding, disseminated candidiasis, pseudomona septicemia	1
1	M	Biliary atresia, Kasai operation	Ischemic injury of allograft, liver failure	1
5½	F	Biliary atresia, Kasai operation	Allograft necrosis, hepatic artery thrombosis	1
1½	F	Biliary atresia, Kasai operation	First graft: severe ischemic injury; second graft: severe ischemic injury	1
3	F	Neonatal hepatitis, micronodular cirrhosis	First graft: septic thrombovasculitis due to candida of allograft; second graft: septic shock, ischemic injury	1
9	F	Wilson disease	First graft: severe ischemic injury; second graft: bilateral uncal and cerebellar tonsillar herniation	1

Acute rejection consists of portal or lobular inflammatory infiltrates, disruption of the limiting plate, and cellular injury along the bile ducts with occasional portal and central venous thickening. Chronic rejection is a vascular injury of medium-size hilar arteries that show subendothelial foam cells, fibrinoid necrosis, and intimal hyperplasia, as well as extensive periportal fibrosis and absence of bile ductules (5). The clinical picture of chronic rejection is similar to that of chronic liver failure with end-stage disease of any other cause, and therefore, it can only be assumed. Fever, anorexia, depression, and abdominal pain are the early clinical manifestations. Increasing serum alkaline phosphatase and serum transaminase values and prolonged prothrombin time are among the laboratory findings. Hepatic scintigraphy may show the nonspecific finding of decreased uptake.

Either cholangiography or ERCP is a definitive procedure in the evaluation of biliary leakage, the site of biliary stenosis, or in determining complete biliary obstruction.

The algorithm we propose is a working one. We anticipate appropriate modifications as diagnostic imaging and management methods advance. ■

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