Biliary Complications in Liver Allografts After Hepatic Artery Occlusion: A 6½-Year Study

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HEPATIC artery thrombosis, the most common vascular complication in liver allograft recipients, is a major cause of morbidity and mortality.[1] Because this complication usually requires liver retransplantation for survival, prompt diagnosis is important. Because the principal site of ischemic injury is the allograft biliary tree, arterial occlusion (thrombosis or severe stenosis) may be manifested clinically as a biliary complication. Cholangiography may be the initial test performed before the diagnosis of hepatic artery occlusion is known.

In this study, we present the biliary complications, as diagnosed by cholangiography, in 30 liver transplant patients with hepatic artery occlusion. Liver allograft recipients who develop such complications should be evaluated for hepatic artery occlusion as the probable underlying etiology.

MATERIALS AND METHODS

During the 6½-year period ending June 30, 1987, 869 patients received 1,139 orthotopic liver transplants. Transhepatic or T-tube cholangiograms were performed in 35 transplant patients with proved thrombosis (32 cases) or severe stenosis (three cases) of the allograft hepatic artery. The biliary complications diagnosed by these cholangiograms form the basis of this study.

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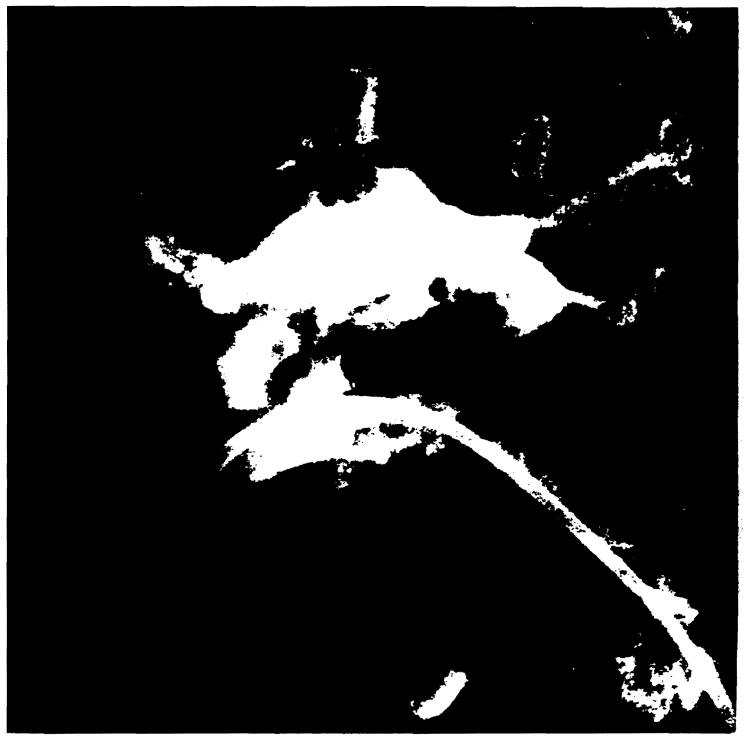

Fig. 1. Hilar biloma after hepatic artery occlusion. Cholangiogram shows a large central biloma cavity. The peripheral intrahepatic biliary tree fills from the central cavity; however, there is no filling of the main right or left hepatic ducts. Histological examination of the hepatectomy specimen after retransplantation showed necrosis of hilar structures including the main right and left hepatic ducts.

RESULTS

Biliary complications were observed in 30 (86%) of the 35 patients with hepatic artery occlusion (thrombosis in 27 and severe stenosis in three). The most common cholangiographic finding, seen in 18 patients, was nonanastomotic leakage of contrast material from the allograft biliary tree. Such bile leakage resulted in a hilar biloma in 12 of these patients (Fig 1). Five patients had bilomas within the liver; four of these also had bilomas in the hilar area. Necrosis of the entire allograft biliary tree occurred in one patient. A bile leak from the donor extrahepatic common duct occurred in one patient.

Nonanastomotic bile duct strictures occurred in 14 patients (Fig 2), four of whom also had nonanastomotic bile leakage. These strictures involved the intrahepatic ducts in five cases, common hepatic duct in three cases, and common hepatic duct bifurcation in six cases. Biliary-enteric anastomotic strictures occurred in only two patients, one of whom had a biloma.

Of the 30 patients with nonanastomotic bile leaks or biliary strictures, 10 patients presented clinically with suspected biliary complications before the diagnosis of hepatic artery occlusion was known. The cholangiographic diagnosis of a nonanastomotic bile leak or nonanastomotic stricture prompted further evaluation for hepatic artery occlusion as the underlying etiology.

DISCUSSION

The hepatic artery provides the entire blood supply to the donor biliary system after trans-

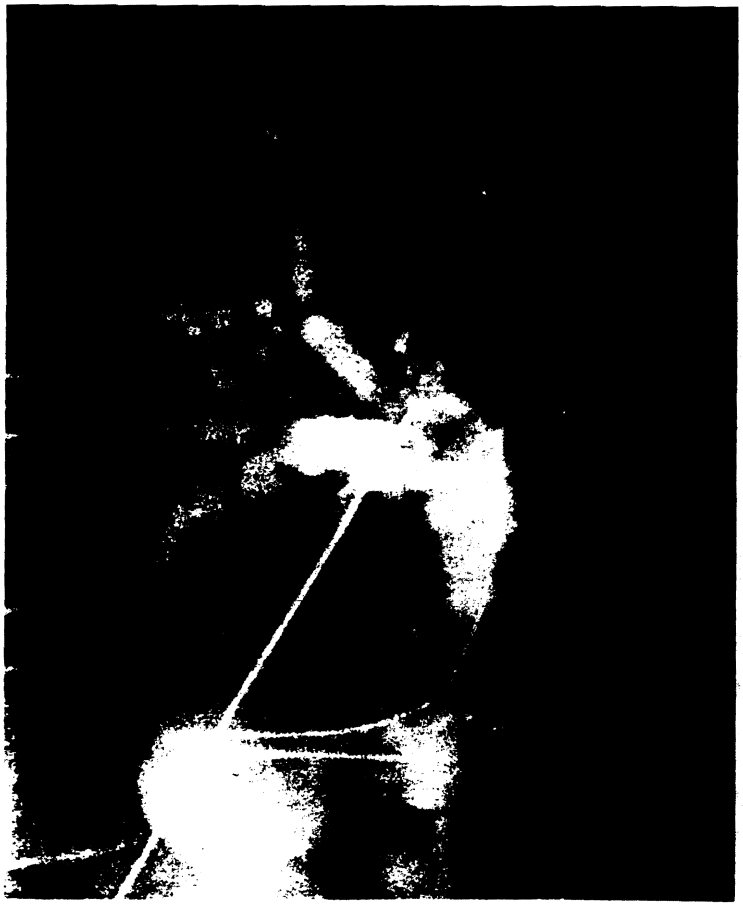

Fig 2. Nonanastomotic biliary strictures after hepatic artery occlusion. Transhepatic cholangiogram shows multiple intrahepatic biliary strictures (arrows).

plantation. Thrombosis or severe stenosis of the hepatic artery may therefore lead to ischemic necrosis of the donor intrahepatic or extrahepatic bile ducts. Clinically, the patient may present with a biliary complication.

Necrosis of the allograft biliary tree may result in nonanastomotic bile leakage. Tzakis et al[2] reported seven transplant patients with hepatic artery thrombosis and extrahepatic bile leaks; all had necrosis of the donor common duct at laparotomy. Bile leakage due to necrosis of intrahepatic bile ducts results in an intrahepatic or hilar biloma (Fig 1).

The development of nonanastomotic bile leakage strongly suggests that hepatic artery occlusion has occurred. Of 20 transplant patients with this complication seen in our institution in the past 6½ years, 18 (90%) had hepatic artery occlusion (thrombosis in 17 and severe stenosis in one).

Most allograft nonanastomotic biliary strictures (Fig 2) are probably due to ischemia. Of 23 transplants with nonanastomotic biliary strictures seen in our institution in the past 6½ years, 14 had hepatic artery occlusion (thrombosis in 12 and severe stenosis in two). In nontransplant patients, bile duct strictures have been reported to occur after peripheral hepatic artery embolization.[3] These strictures are caused by ischemic injury due to occlusion of the peribiliary vascular plexus. Because of collateral circulation, such strictures do not develop after proximal occlusion of the hepatic artery.[4] In liver allografts, hepatic artery thrombosis effectively produces complete dearterialization because of the lack of collaterals that normally exist in the attachments of the liver.

In summary, hepatic artery thrombosis or severe stenosis posttranpslant may cause ischemic necrosis of the allograft biliary tree. Clinically, the patient may present as a primary biliary complication. Biliary complications due to hepatic artery occlusion include nonanastomotic bile leakage and nonanastomotic bile duct strictures. The development of such biliary complications should be evaluated for occlusion of the hepatic artery as the probable underlying etiology.

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