Biliary Strictures Complicating Liver Transplantation

Incidence, Pathogenesis, Management, and Outcome

JOHN O. COLONNA, II, M.D.,* ABRAHAM SHAKED, M.D., PH.D.,* ANTIONETTE S. GOMES, M.D.,†
STEVEN D. COLQUHOUN, M.D.,* ODED JURIM, M.D.,* SUE V. McDIARMID, M.D.,‡
J. MICHAEL MILLIS, M.D.,* LEONARD I. GOLDSTEIN, M.D.,§ and RONALD W. BUSUTTIL, M.D., PH.D.*

Six hundred sixty-six patients received 792 liver transplants between February 1, 1984 and September 30, 1991. Biliary reconstruction was by choledochocholedochostomy (CDCD) with T-tube (n = 509) or Roux-en-Y choledochojejunostomy (CDJ) (*n* = 283). Twenty-five patients (4%) developed biliary strictures. Anastomotic strictures were more common after CDJ (*n* = 10, 3.5%) than for CDCD (*n* = 3, 0.6%). Intrahepatic strictures developed in 12 patients. Six patients had occult hepatic artery thrombosis (HAT). The other six patients received grafts in which cold ischemia time exceeded 12 hours. Anastomotic strictures were successfully managed by percutaneous dilation (PD) in five patients (*n* = 10), operation in three (*n* = 6), with retransplantation required in two patients. Intrahepatic strictures were managed by PD in seven, retransplantation in one, and expectantly in four patients. Of 25 patients, 19 (76%) are alive with good graft function. In three of six deaths, the biliary stricture was a significant factor to the development of sepsis and allograft failure. The authors conclude that (1) anastomotic strictures are rare after LT; (2) the development of biliary strictures may signify occult HAT; (3) PD is effective for most strictures; and (4) extended cold graft ischemia (<12 hours) may be injurious to the biliary epithelium, resulting in intrahepatic stricture formation.

ADVANCES IN ORGAN preservation, immunosuppressive agents, and refinement of surgical techniques have lead to a dramatic increase in survival after liver transplantation over the past two decades. Although biliary reconstruction, once considered the "Achilles heel" of liver transplantation, has become standardized, biliary complications remain a significant source of complications.[1-4] Fortunately early diagnosis and prompt intervention has decreased mortality due to these complications.[3-5]

Presented at the 112th Annual Meeting of the American Surgical Association, April 6–8, 1992, Palm Desert, California.

Address reprint requests to Ronald W. Busuttil, M.D., Ph.D., Director, UCLA Liver Transplantation Program, Room 77-132, CHS, 10833 LeConte Avenue, Los Angeles, CA 90024.

Accepted for publication April 10, 1992.

From the Departments of Surgery, Radiology,† Pediatrics,‡ and Medicine,§ UCLA School of Medicine, Los Angeles, California*

Biliary leaks typically present early after transplantation and mandate immediate operative or endoscopic intervention to avoid sepsis. Biliary strictures, however, may have a more indolent course, and management options and their timing are more diverse. In this study, we review our experience with biliary strictures after liver transplantation. Factors in their pathogenesis are identified, and the results of radiologic and operative interventions are analyzed.

Methods

Patient Population

Between February 1, 1984 and September 30, 1991, 666 patients received 792 liver transplants at UCLA Center for the Health Sciences. Indications for transplantation in adults and children are given in Table 1. The hospital and outpatient records were reviewed, and patients with a biliary stricture confirmed by cholangiography were studied in further detail. Follow-up is 100% for patients in our program. Potential contributing factors such as hepatic artery thrombosis, prolonged cold ischemia time, ABO incompatibility, cytomegalovirus infection, number of rejection episodes, and type of rejection were analyzed. Treatment strategies were evaluated for initial success and long-term outcome.

Technique of Biliary Reconstruction

Orthotopic liver transplantation of a whole or reduced graft was performed using standard techniques that we have reported previously.[6-8] Choledochocholedochostomy (CDCD) is our preferred method in adult and adolescent

TABLE 1. *Indications for Transplantation in Adults and Children**

Adults		Children	
Diagnosis	Number	Diagnosis	Number
PBC	63	Biliary atresia	113
CAHC	106	Metabolic	24
CAHB	42	CAH-all	18
Alcoholic	52	Familial cholestasis	5
Fulminant hepatitis	36	Fulminant hepatitis	25
Hepatic neoplasm	39	Hepatic neoplasm	6
Sclerosing cholangitis	43	Other	19
Other	75	Total	210
Total	456		

* Data from February 1, 1984 to September 31, 1991.

patients. During the recipient hepatectomy, the bile duct is ligated and divided well above the cystic duct junction, providing sufficient length for a tension-free anastomosis with the donor bile duct. After portal and arterial reperfusion of the allograft, a cholecystectomy is performed and the donor bile duct is divided just above the cystic duct junction. An everting end-to-end anastomosis is performed with interrupted 5–0 polypropylene suture. All knots are tied on the outside. When the posterior row has been completed, an appropriate-sized T-tube (5 to 10 French) is inserted into the duct with the sidearm exiting the recipient's native bile duct well below the level of the anastomosis. The upper limb of the tube is trimmed to lie below the bifurcation of the common hepatic duct. The anterior row is completed and the exit site is reinforced with an additional suture. A cholangiogram is obtained to confirm the absence of obstruction or leak. A closed suction drain is placed posterior to the anastomosis before closing.

In most children and selected adult patients, a Roux-en-Y choledochojejunostomy (CDJ) is performed. The small diameter of the bile duct in infants and small children (1 to 2 mm) makes CDCD impractical. Choledochojejunostomy is performed in adults with diseases of the bile duct (*e.g.*, sclerosing cholangitis and cholangiocarcinoma), secondary biliary cirrhosis, excessive discrepancy in the sizes of the donor and recipient bile ducts, and in most retransplants. A standard 40-cm Roux limb is fashioned and is brought retrocolic whenever possible. If a Roux limb is already present (*e.g.*, Kasai limb), it is reused if sufficient length (>15 cm) remains after resecting any areas devitalized during the recipient hepatectomy. The anastomosis is performed with interrupted monofilament suture; 6–0 in adults, 7–0 in children. Absorbable suture is used for the posterior row with knots placed on the inside. The tails of the center suture are then tied around a short polyethylene or Silastic stent of appropriate size (2 to 6 French). External drainage is not performed routinely. The anterior row is then completed with the knots on the outside, and tension is taken off the anastomosis by tacking the Roux limb to adjacent hilar tissues. Cholangiography is not performed and a closed suction drain is placed posterior to the anastomosis before closing.

Postoperative Management

Our protocol for postoperative immunosuppression has been previously reported.[9] Most patients have received maintenance therapy with cyclosporine and prednisone with or without azathioprine. Episodes of rejection are initially treated with high-dose steroids followed by OKT3 in resistant cases. In the past 2 years, a number of patients have been treated with FK506 as part of a multicenter trial.

In patients with a CDCD, a T-tube cholangiogram is performed 7 to 10 days after operation (Fig. 1). A HIDA scan is obtained on patients with a CDJ. If there is no leak, the drain is removed and the T-tube is clamped. The T-tube is usually removed after 4 months. The patient is admitted for a cholangiogram in the interventional radiology suite. If the study is normal, the T-tube is removed over a guide wire passed into the duct. A 5-French angiography catheter is then advance over the wire and positioned with its tip just outside the former T-tube exit site. This drain then is advanced out over the next 24 hours and the patient is discharged. This technique has essentially eliminated episodes of bile peritonitis after T-tube removal. In the occasional patient who develops signs of bile peritonitis, a nasobiliary catheter is placed endoscopically. This catheter remains in place until follow-up cholangiography demonstrates no leak.

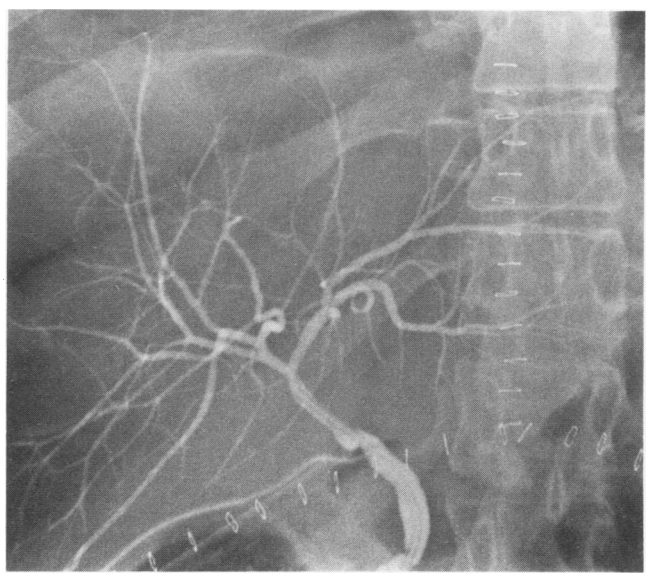

FIG. 1. T-tube cholangiogram performed 1 week after orthotopic liver transplantation.

Diagnosis of Biliary Stricture

The biliary strictures reported in this study were diagnosed either at the time of routine cholangiography as described above or through diagnostic cholangiography in the evaluation of allograft dysfunction. Patients with a cholestatic profile on liver function tests, evidence of biliary obstruction on liver biopsy, and patients with biliary dilation on ultrasound are candidates for cholangiography. T-tube cholangiography is performed when possible. Patients with a CDJ or those in whom the T-tube has been removed are studied with either transhepatic cholangiography or endoscopic retrograde cholangiopancreatography, depending on their anatomy (Fig. 2). Any patient who has a biliary stricture diagnosed is further studied to assess patency of the hepatic artery by color flow Doppler ultrasound or by hepatic arteriography in equivocal cases.

Management of Biliary Strictures

Patients with biliary strictures were managed by one of four methods: medical, percutaneous dilation, operative revision, or retransplantation. Clinically asymptomatic patients with normal graft function and minimal biliary dilation on cholangiography were observed. Ursadiol was prescribed for its choluretic effect. Patients with anastomotic strictures were treated by either percutaneous dilation and stenting or operative revision. Symptomatic intrahepatic strictures were dilated. Retransplantation was reserved for patients who developed progressive allograft dysfunction despite the above measures.

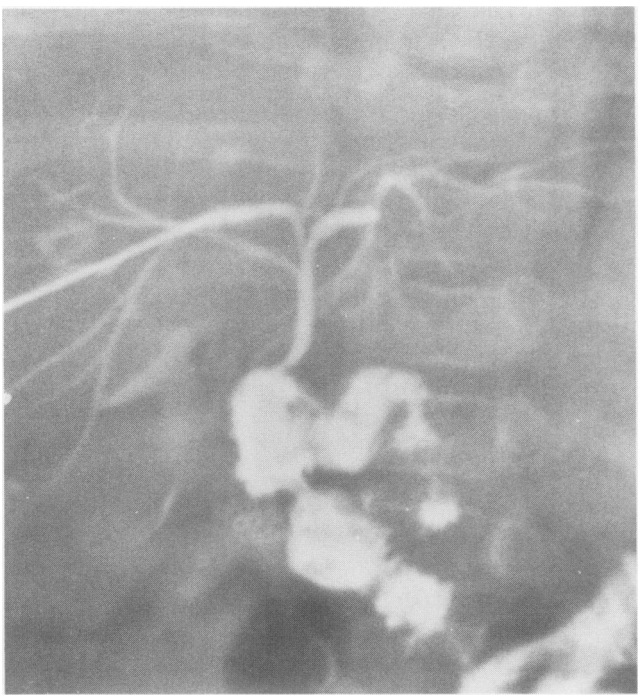

FIG. 2. Transhepatic cholangiogram demonstrating a normal Roux-en-Y choledochojejunostomy after orthotopic liver transplant.

Results

Choledochocholedochostomy was performed in 509 cases and CDJ in 283. Biliary strictures occurred in 25 patients (4%).

Anastomotic Strictures

Anastomotic strictures were more common after CDJ (n = 10, 3.5%) than for CDCD (n = 3, 0.6%). Stricture of the CDCD anastomosis was diagnosed at 3, 4, and 6 months after operation. Hepatic artery patency was documented noninvasively in all three. One patient was successfully treated by percutaneous transhepatic dilation and stenting. One patient was successfully converted to a CDJ. The final patient had one attempt at conversion to a CDJ, but restrictured and developed progressive graft dysfunction. She underwent successful retransplantation 8 months after her initial transplant.

Stricture of a CDJ anastomosis was diagnosed from 1 to 24 months after transplant. Three of these 10 patients were subsequently shown to have hepatic artery thrombosis. Percutaneous dilation was attempted in all 10 patients and was the only therapy required in four. One of the failures developed a portal-biliary fistula, which precipitated a progressive downhill course resulting in death of sepsis and multisystem failure. Two patients underwent operative revision of the CDJ anastomosis, with excellent long-term results. Two patients underwent operative revision in the face of persistent sepsis and eventually died of sepsis. The remaining patient underwent successful retransplantation without attempt at operative revision. The presence of hepatic artery thrombosis did not preclude successful management, with only one patient requiring retransplantation. One patient was treated with dilation alone, and one underwent successful operative revision.

Intrahepatic Strictures

Twelve patients developed strictures in the biliary tree at or above the level of the hepatic duct bifurcation. Six of these patients were subsequently shown to have hepatic artery thrombosis. In two of these patients, only the right hepatic artery was thrombosed and strictures were predominately in the right lobe. Of the six patients who developed intrahepatic strictures in the absence of hepatic artery thrombosis, one received an ABO-incompatible liver in the setting of fulminant hepatitis. Two of the other patients were successfully treated for cytomegalovirus hepatitis after transplantation. None of these patients had chronic rejection. Preservation of all donor livers in this group was accomplished with University of Wisconsin

(UW) solution. It is our policy to irrigate the bile duct on the back table with UW solution in addition to the *in situ* gallbladder flush. Of particular interest is that the ischemia times for the six livers that developed intrahepatic strictures in the absence of hepatic artery thrombosis were 12, 12, 12.5, 14, 14.5, and 16 hours.

The management of patients with intrahepatic strictures is outlined in Table 2. Four of these 12 patients have been treated medically and remain asymptomatic with normal graft function. Percutaneous dilation was attempted in seven patients and was successful in all. Repeated dilations were necessary in each case (mean, 2.5). Two of these six patients eventually succumbed to viral pneumonia. The other five are alive with normal graft function. The remaining patient in this group had multiple strictures due to hepatic artery thrombosis and has undergone successful retransplantation. Overall graft and patient survival for patients developing biliary strictures in this series is 64% and 76%, respectively.

It is difficult to assess the role allograft rejection may have played in the development of the biliary strictures. Each patient had at least one episode of biopsy-proven acute rejection. OKT3 was necessary in 12 patients. Two patients have developed chronic rejection, but in each case, it followed diagnosis and successful treatment of the stricture by more than 12 months.

Discussion

Numerous factors have been identified as contributing to the development of biliary strictures after liver transplantation. The association of biliary complications with hepatic artery thrombosis is well established.[4,10] Particularly in the pediatric patient, the development of biliary obstruction or leakage should arouse suspicion of hepatic artery thrombosis, even in the absence of significant hepatocellular dysfunction or a "normal" Doppler ultrasound.[11] Less-well-established risk factors include ABO incompatibility, chronic rejection, cytomegalovirus infections, and recurrence of primary ductal diseases like sclerosing cholangitis.[12]

TABLE 2. *Management of Intrahepatic Biliary Strictures in 12 Patients After Orthotopic Liver Transplantation*

Hepatic Artery	Treatment	*N*	Success	Outcome
Thrombosed	Medical	3	3	Good
(*n* = 6)	Dilation	2	2	Good
	Re-OLT	1	1	Good
Patent	Medical	1	1	Good
(*n* = 6)	Dilate	5	5	2 deaths*
	Re-OLT	0	N/A	N/A

N/A, not applicable; Re-OLT, retransplantation.
* Deaths were unrelated to stricture; outcome was good in the three others.

Primary choledochocholedochostomy is the preferred anastomosis in many large centers. The gallbladder conduit technique has been largely abandoned because of associated bile stasis with stone formation and frequent episodes of cholangitis.[13] With CDCD anastomotic stricture rates are uniformly less than 10%.[1-4] It would seem that this type of anastomosis is analogous to primary repair of an accidental injury to the common bile duct at the time of cholecystectomy. In the nontransplant setting, however, the stricture rate appears to be much higher. Csendes et al.[14] recently reported a series of 43 patients with accidental section of the common bile duct. Of 37 patients who had immediate end-to-end repair with a T-tube stent, 29 patients (78%) developed a benign stricture by 4 years after operation. Andren-Sandberg et al.[15] reported a series of 65 cases of accidental injuries to the bile duct during cholecystectomy with 38 patients undergoing immediate end-to-end repair. Of the 26 who did not have an early complication, 14 (54%) developed a stricture. These results are in sharp contrast to a 0.6% incidence in our series of 509 cases with a mean follow-up of 2.3 years. Andren-Sandberg et al. reported that 75% of the strictures in their series developed within 18 months after repair.[15]

What accounts for the differences in strictures after CDCD in the transplant and nontransplant setting? One factor may be the level of the bile duct injury. Csendes et al.[14] found that accidental bile duct injuries were three times more likely to occur above the level of the cystic duct than more distally. Injuries located near the hepatic duct bifurcation are considerably more difficult to repair. Terblanche and co-workers[16,17] have emphasized the relatively poor blood supply to the bile duct below the hilum. In the transplant setting, the anastomosis is performed at a lower level, which provides better exposure for precise suture approximation. Additionally, the use of loop magnification with transplant operations further advances the technical conduct of the anastomosis. A second factor is likely the absence of tension in the transplant anastomosis. The additional length one gains with two bile ducts allows for a completely tension-free reconstruction. The associated inflammatory disease (*e.g.*, cholecystitis) frequently present in the nontransplant setting is absent in the transplant operation.

The use of immunosuppressive agents and their attendant impairment of the healing process may further decrease the likelihood of stricture formation. The effects of glucocorticoids on wound healing have been well described and include limitation of capillary budding, decreased rate of fibroblast proliferation, and decreased rate of epithelialization.[18] Eisinger and Sheil[19] noted a similar impairment of wound healing with azathioprine, but found very little effect with cyclosporine. More recently, Holla et al.[20] have shown that cyclosporine inhibits tensile strength of healing incision wounds at a dose of 5 mg/kg

in rats. In a study more analogous to a transplant bile duct anastomosis, Recker et al.[21] have reported that cyclosporine impairs healing of ureteroneocystostomy in rats.

The development of intrahepatic strictures in the absence of hepatic artery thrombosis is a unique pathological entity whose natural history has not been fully elucidated. A report by Sanchez-Urdazpal et al.[22] described a series of 31 patients with this lesion. The salient finding in this study was the association of prolonged donor ischemia time and stricture formation. In livers perfused with UW solution, the incidence was 7% for ischemia times less than 13 hours, 52% if longer than 13 hours, and 69% if longer than 15 hours. A similar relationship was shown for livers perfused with EuroCollins solution. An ischemia time of less than 6.5 hours was associated with a 2% incidence. Ischemia times of more than 6.5 hours had a 24% incidence of ischemic-type biliary complications.

The implication that these strictures are ischemic in nature is quite plausible and is supported by their appearance on cholangiogram, which is indistinguishable from that of strictures caused by hepatic artery thrombosis (Figs. 3 and 4). Our data support the role of prolonged cold ischemia in the pathogenesis of these strictures. No intrahepatic strictures were observed with less than 12 hours' ischemia using UW solution, and moreover, none were seen in the pre-UW era, when ischemia did not exceed 6 to 8 hours. The incidence of ischemic stricture development is variable, as illustrated by our results and that reported by the Mayo Clinic.[22] In our series, there were 155 livers with cold ischemia times over 13 hours. We could identify only six patients with ischemic type biliary strictures (3.8%). There were 22 livers in which the cold ischemia time exceeded 18 hours, and none of these livers developed such strictures. It is unclear why our results are so different. Because a few of our patients have remained asymptomatic without treatment, there may be a number of patients who have simply not been diagnosed, thus underestimating the true incidence. Additionally, if hepatic artery patency was assessed only by noninvasive means in the Mayo Clinic series, it is possible that some of the strictures may have been due to branch hepatic artery thrombosis.

The blood supply to the biliary tree has been shown to be almost solely arterial, with no significant contribution form the portal vein.[17,23] The hepatic artery is in essence an end artery for the donor biliary tree, as collaterals from the lower extrahepatic biliary tree are interrupted in the process of procurement and transplantation. Portal perfusion can maintain the hepatocytes in many cases of hepatic artery thrombosis.[23] New collaterals can form after hepatic artery thrombosis, as demonstrated in Figure 4B, and may limit additional stricture formation. It is somewhat more difficult to explain the situation in cases of intrahepatic strictures that occur in the absence of hepatic artery thrombosis. The most likely explanation is in the essential difference in response to ischemic injury between a hollow viscus and a solid organ. As described above, there is compelling evidence that these lesions are ischemic in nature. This is further supported by the fact that in five of six patients in our series, the initial transaminases were greater than 2500 IU/mL. Liver biopsies in the early post-

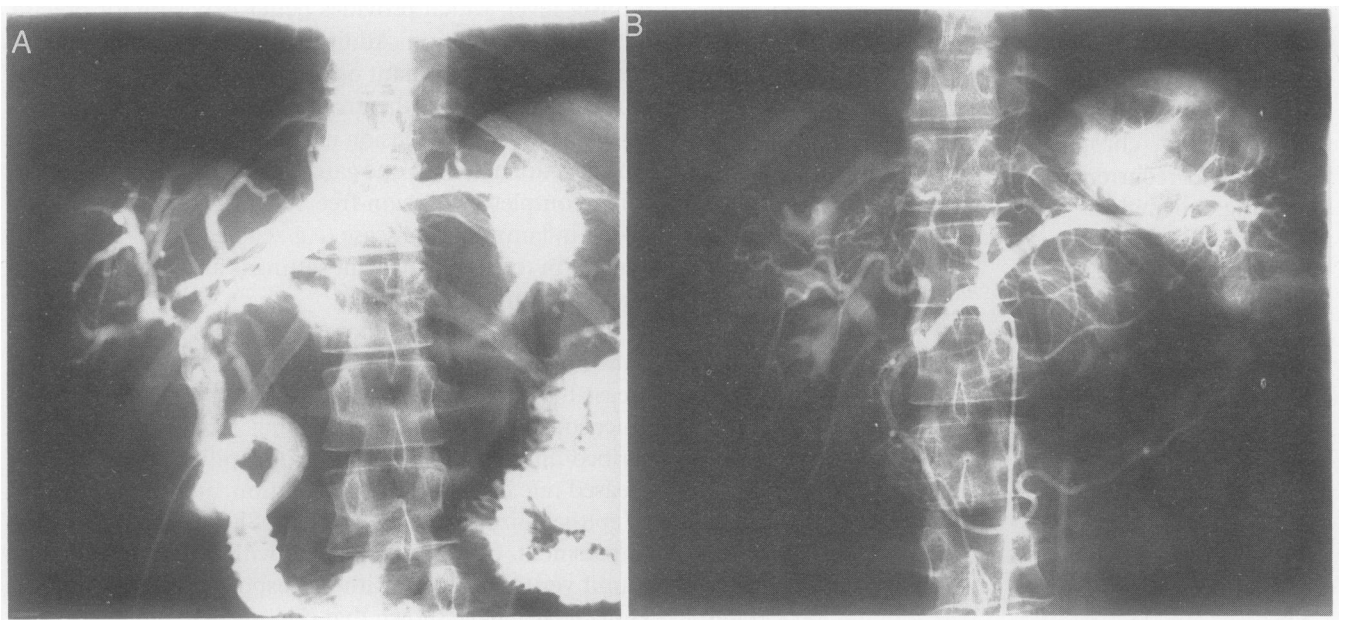

FIG. 3. (A) Transhepatic cholangiogram and (B) hepatic angiogram showing a hilar biliary stricture and a patent hepatic artery.

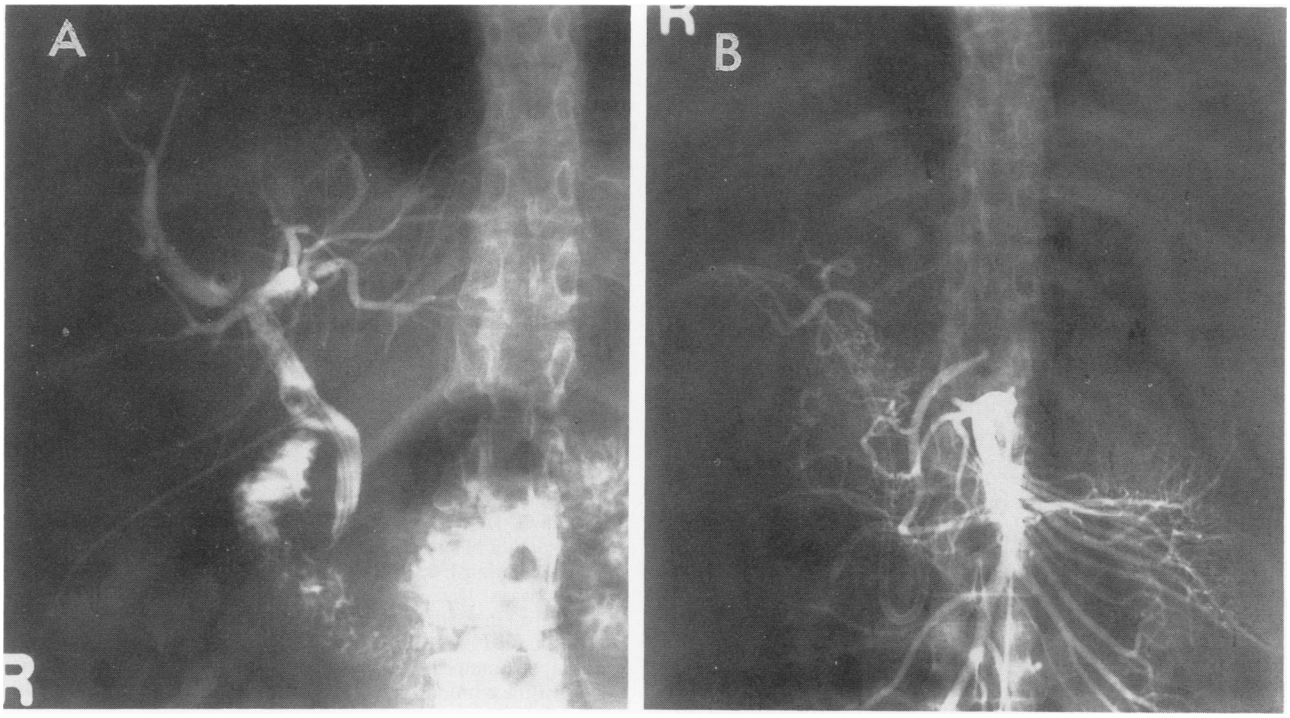

FIG. 4. (A) Transhepatic cholangiogram and (B) hepatic angiogram showing a hilar biliary stricture and thrombosis of the hepatic artery with reconstitution via collaterals.

operative course of all six patients demonstrated significant ischemic injury. We have previously shown that this type of injury often resolves on subsequent biopsies and is accompanied by a normalization in graft function.[24] Thus, the "solid organ" component of the liver can eventually recover from an ischemic insult that falls short of

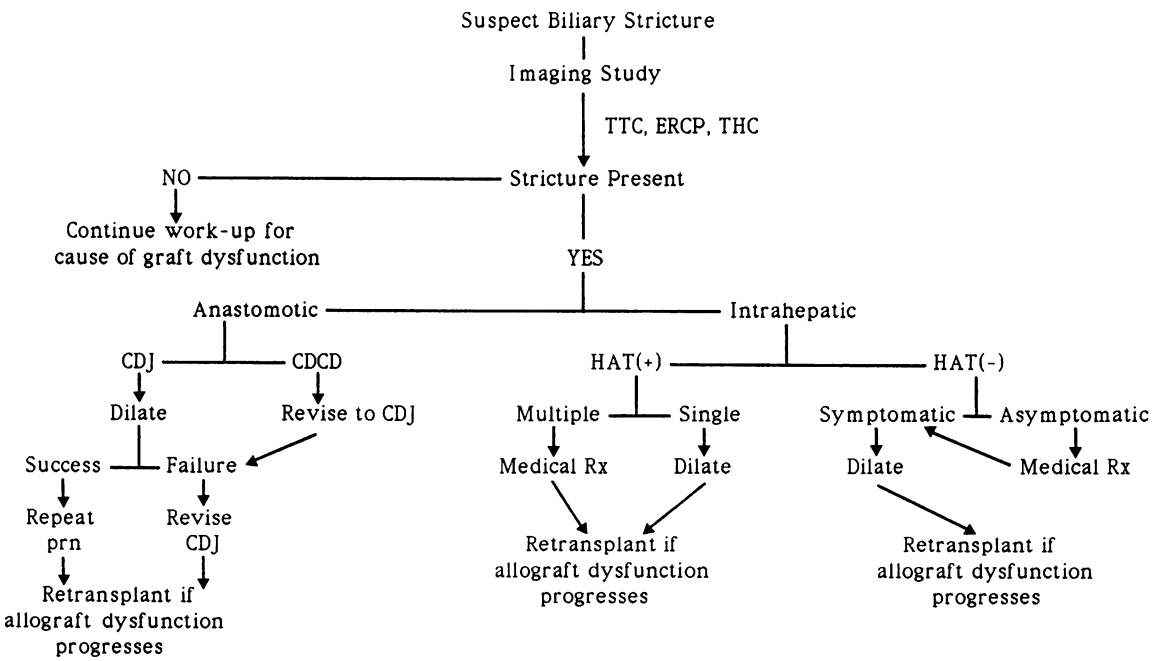

FIG. 5. Algorithm for the management of biliary strictures occurring after liver transplantation. TTC, T-tube cholangiogram; ERCP, endoscopic retrograde cholangiopancreatogram; THC, transhepatic cholangiogram; CDJ, choledochojejunostomy; CDCD, choledochocholedochostomy; HAT, hepatic artery thrombosis.

massive infarction and fulminant allograft failure. The biliary tree, however, behaves as a hollow viscus. Ischemia produces a spectrum of injury ranging from mucosal slough to full-thickness necrosis and perforation. Full-thickness necrosis can also produce stricture formation. This appears to be the explanation for intrahepatic stricture formation.

Our experience suggests that percutaneous dilation and stenting is effective therapy for most symptomatic intrahepatic strictures and for strictures of a CDJ anastomosis. It was the only therapy needed in 11 of 17 patients so treated. Three patients developed portal-biliary fistulas, one of whom eventually succumbed. This stresses the need for a radiologist who is experienced in biliary tract interventional procedures. It also emphasizes the need to restrict such procedures to patients who can potentially benefit. In patients with multiple intrahepatic strictures due to hepatic artery thrombosis, retransplantation is probably the treatment of choice (Fig. 5).

Conclusions

From our review of biliary strictures complicating liver transplantation, we conclude that (1) anastomotic strictures are quite rare, (2) the development of biliary strictures may signify occult hepatic artery thrombosis, (3) percutaneous dilation is effective for most strictures, and (4) extended cold graft ischemia (>12 hours) may be injurious to the biliary epithelium, resulting in intrahepatic stricture formation.

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DISCUSSION

DR. G. KLINTMALM (Dallas, Texas): Mr. President, members and guests: This is a very important paper and certainly the biggest series of these kind of problems documented outside Pittsburgh. We have a very similar experience at Baylor with a low complication rate from strictures. We have found that if we divide the strictures up into early—that is, those less than 3 months and those later than 3 months, we end up with two distinct populations.

The early ones are anastomotic, and are in our experience usually due to the training of our fellows. That is where we pay our dues. The second population, which is the larger one, are intrahepatic strictures, and they are, like here, seemingly ischemic in nature. In our experience, two thirds of them are ischemic, and the other third is maybe due to rejection. I have two questions for Dr. Colonna.

This remarkably low incidence of biliary strictures in liver transplantation is worth discussing. Is this due to the steroid therapy we give our patients? As we all know, patients on steroid therapy develop very small scar formations. Should we recommend steroid therapy for all biliary reconstruction to prevent stricture formation also in the nontransplant?

And the second question is, do you have any experience with the indwelling self-expanding Gianturco stents?