Hepatic Artery Reconstruction for Hepatic Artery Thrombosis After Orthotopic Liver Transplantation

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- We evaluated the efficacy of reconstruction of the hepatic artery for intraoperative or postoperative thrombosis in orthotopic liver transplantation: Of 37 grafts with artery thrombosis, 13 (35.1%, 6 intraoperative and 7 postoperative) underwent reconstruction of the hepatic artery. The arterial flow was reestablished and maintained in 5 (38.5%) of the 13. Recurrent thrombosis in the other 8 grafts developed 2 to 24 days (mean, 13.8 days) after transplantation. Reconstruction was successful in 50% (4/8) of the adults, compared with only 20% (1/5) of the children. Satisfactory results were obtained when a definitive cause of thrombosis could be identified. We conclude that early recognition and correction of the cause of hepatic artery thrombosis during or after orthotopic liver transplantation, especially in adults, is often a graft-saving and lifesaving procedure worthy of consideration.

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After orthotopic liver transplantation (OLTx), hepatic artery (HA) thrombosis (HAT) remains the most common technical complication that requires retransplantation. In children, HAT accounts for approximately 40% of the retransplantations. The incidence of this serious complication generally varies from 9% to 18%, and the mortality approaches 50%. Hepatic artery thrombosis typically occurs in the early postoperative course and is usually fatal without retransplantation. The shortage of donor organs remains a major limiting factor in clinical liver transplantation, and thus salvage of a liver allograft, if possible, is of critical importance. This study analyzes our experience with 13 patients in whom HA reconstruction (HAR) was attempted during or after OLTx.

PATIENTS AND METHODS

During the 1-year period between January 1 and December 31, 1987, 323 patients underwent 389 OLTx procedures (282 in adults and 107 in children) at the University of Pittsburgh (Pa). Of these, 37 grafts (9.5%) (16 in adults and 21 in children) developed HAT during or shortly after OLTx. Hepatic artery reconstruction was carried out in 13 patients (4.0%), during OLTx in 6 and 4.7 ± 3.5 days (mean ± SD) later (range, 1 to 12 days) in 7 patients. In those who developed HAT postoperatively, the diagnosis of HAT was established by arteriography in 3, by Doppler ultrasonography in 1, and at the time of semissec- tive exploratory laparotomy for HA stricture on either Doppler ultrasonography or arteriography in 1 each and for intra-abdominal bleeding in 1 patient. Reconstruction of the HA among these patients was carried out as an emergency procedure immediately after the diagnosis of HAT was established. The age among the patients studied ranged from 17 months to 58 years, with a mean of 22.0 years; 6 (46.1%) were male. Charts of these patients were reviewed to evaluate the possible cause of HAT, the technique and timing of HAR, and the outcome.

Baseline immunosuppression was achieved with cyclosporine and corticosteroids. Episodes of allograft rejection were treated with a bolus of intravenous methylprednisolone and/or steroid recycle, and steroid-resistant rejection was treated with OKT3 (Orthoclone; Ortho Pharmaceutical Co, Raritan, NJ). Immediately postoperatively, the pediatric patients received low-molecular-weight dextran intravenously at 5 to 10 mL/h for 4 days; heparin, 50 U/kg subcutaneously every 12 hours throughout the hospital stay (approximately 4 weeks); aspirin, 20 to 40 mg/d by mouth or per nasogastric tube for at least 3 months; and dipyradimole (Persantine), 12.5 to 25 mg/d by mouth for at least 3 months. The above-therapy was discontinued if the patient demonstrated clinical or laboratory evidence of coagulopathy. All adult patients who underwent OLTx for Budd-Chiari syndrome received anticoagulation with heparin (5000 U subcutaneously, three times a day), followed by warfarin sodium to maintain the prothrombin time around 18 seconds.

RESULTS

Table 1 lists the clinical data of the patients who developed HAT during the actual transplant procedure. The possible cause of HAT among these patients included poor inflow related to a triple arterial supply of the native liver in one (patient 1), rotation of the aortohepatic interposition graft in the retroperitoneal tunnel in one (patient 2), disseminated intravascular coagulation of the donor in one (patient 3), intimal dissection of the recipient common HA due to excessive traction in one (patient 6), and unknown in two patients.
Of these, 3 of HAT during was carried out (mean ± SD) of recivered HAT by arteriography of semielastic Doppelt intra-abdominal these patients after the mean 22.0 years. We reviewed the timing of closporine and treated with a diet recycle, and orthotolx; Operative: dextran intrasubcutaneousadmittedly 4 weeks: be for at least and the patient's angulopathy. Allevi syndrome temporarily, then the prothrombin (patients 3 and 5). In patient 4, the donor had died of a gunshot wound to the head, and a biopsy of the kidney revealed extensive microthrombosis of the glomeruli. The liver, however, looked grossly normal and was transplanted into a recipient without technical difficulty. The HA became thrombotic despite good inflow and a technically sound anastomosis. For these patients with infraoperative HAT, the HA anastomosis was taken down and inspected, and the donor HA was flushed with heparinized saline solution (10 U/mL). The method of revision of the thrombotic HA consisted of the placement of the aortohepatic interposition graft (patients 1 and 6), untwisting of the aortohepatic interposition graft and revision of the anastomosis (patient 2), revision with (patient 4) or without (patient 5) systemic heparinization, and the replacement of the donor celiac axis and the proximal common hepatic artery with an interposition graft with the use of the donor iliac artery (patient 3).

The HA flow was reestablished successfully in two patients (patients 1 and 2) (33.3%), after the placement or revision of the aortohepatic interposition arterial graft in one. Patient 2

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<tr>
<th>Table 1.—Clinical Data of Patients Who Underwent HAR During OLTx*</th>
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<td><strong>Patient No./ Age/Sex</strong></td>
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<tr>
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<tr>
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<td>5/17 mo/M</td>
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<td>6/56 y/M</td>
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*HA indicates hepatic artery; HAR, HA reconstruction; HAT, HA thrombosis; CAH, chronic active hepatitis; RHA, right HA; PHA, proper HA; LHA, left HA; CA, celiac axis; CHA, common HA; AHIG, aortohepatic interposition graft; IG, interposition graft; IV, intravenous; OLTx, orthotopic liver transplantation; CBA, congenital biliary atresia; and DIC, disseminated intravascular coagulation.

†Numbers in parentheses are posttransplant day.

<table>
<thead>
<tr>
<th>Table 2.—Clinical Data of Patients Who Underwent HAR After OLTx*</th>
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<td>13/36 y/F</td>
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*HA indicates hepatic artery; HAR, HA reconstruction; POD, postoperative day; HAT, HA thrombosis; PBC, primary biliary cirrhosis; RHA, right HA; PHA, proper HA; CA, celiac axis; AHIG, aortohepatic interposition graft; OLTx, orthotopic liver transplantation; CBA, congenital biliary atresia; CHA, common HA; IG, interposition graft; SpA, spenic artery; CBD, common bile duct; FLF, fulminant liver failure; PSC, primary sclerosing cholangitis; and SMA, superior mesenteric artery.

†Numbers in parentheses are posttransplant day.
died of rupture of an infected aortic-donor iliac arterial anastomosis site on posttransplant day 15. In the other four patients, the HA occluded 1 to 8 days (mean, 4.3 days) after transplantation and required retransplantation.

Table 2 lists the clinical data of the patients who developed HAT after OLTx. Possible causative factors included poor inflow related to double arterial supply of the native liver in patients, the HA occluded 1 to 8 days (mean, 4.3 days) after transplantation and required retransplantation. The clinical course of the 5 patients after HAT was indistinguishable from that of patients without HAT, and none developed biliary stricture or liver abscesses after reconstruction. Of these 5 patients, 3 were alive and well with patent HA on repeated Doppler ultrasonography and had normal graft function. The other 2 died of infectious complications with patent HA.

Table 3 lists the correlation between various clinical variables and the outcome of HA among the patients studied. Resection of the HA was successful in 50% (4/8) of adults. In children, on the other hand, only one (20%) of five grafts could be salvaged, after incidental detection of HAT on exploratory laparotomy and revision with administration of streptokinase intravenously and through the HA (patient 12). Although no obvious relationship was observed between the outcome of HAT and the timing of HAR or methods of HAR, grafts with HAT due to mechanical causes responded well to HAR.

**COMMENT**

As a general rule in vascular surgery, embolectomy for arterial thromboembolic disease is often satisfactory, whereas thrombectomy is seldom effective. In solid-organ transplantation, little is known about revascularization of the graft with acute arterial occlusion.

For kidney transplants in which the renal artery is the only inflow for the graft, Melzer et al. in 1982 reported successful recovery of autograft function with a revision of the arterial anastomosis after 3 hours 15 minutes of warm ischemia. The cause of arterial occlusion was kinking of the recipient internal iliac artery. Okiye and Zincke in 1983 described a patient whose arterial thrombosis was successfully treated after 5 hours 30 minutes of warm ischemia, with repositioning of the kinked autograft renal artery, intra-arterial injection of heparin, and postoperative systemic heparinization. Acute tubular necrosis in this patient resolved in 3 days.

For liver transplants, Klintmalm et al. in 1988 described three patients with HAT after OLTx in whom attempts were made after transplantation to salvage the grafts by immediate vascular reconstruction. Two patients with kinking of the HA distal to the anastomosis were treated with thrombectomy, a flush of the HA with heparinized saline, and a revision of the HA anastomosis; this resulted in complete recovery of graft function in one, whereas in the other, biliary stricture and hepatic abscess ensued and HAT was confirmed at the time of retransplantation 5¾ months after the initial transplant. In another patient in whom intimal dissection of the donor HA was the cause of HAT, the aortohepatic graft was placed after thrombectomy and a flush with heparinized saline. This patient developed biliary stricture and a hepatic abscess that required surgical drainage. The patency of the HA after the reconstruction was not documented in the two patients who did not undergo retransplantation in that report. The authors stressed the importance of early detection and intervention of HAT after OLTx.
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In this series, however, no evidence of rejection could be demonstrated among the patients with HAT.

For insufficient arterial inflow due to recipient HA anomaly or hypoplastic recipient artery, an aorto-hepatic interposition graft has been placed through a retroperitoneal route from the infrarenal abdominal aorta, with the use of a donor iliac artery. Since inflow problem is a correctable cause of HAT, and since palpation of the HA is not a reliable method in the assessment of the adequacy of blood flow through the HA, measurement of the HA blood flow with a flow meter seems important to avoid HAT.

The study described herein seems to indicate that an early recognition of HAT followed by an attempt at reconstruction of the HA for HAT during or after OLTx, especially in adults, is often satisfactory and graft-saving as well as lifesaving.

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References


Invited Commentary

Thrombosis of the HA remains a significant problem after liver transplantation. The vast number of liver transplantations performed in Pittsburgh has provided Yanaga et al with a unique experience of this complication. The clinical outcome after HAT largely depends on the age of the patient and the timing of occurrence of HAT. Pediatric patients may tolerate the insult when it occurs after transplantation, and some adult patients presenting late after the transplantation procedure, when a collateral blood supply has developed, have been successfully treated conservatively. However, the vast majority of patients with HAT require urgent retransplantation. This is not always possible, as donor organs are often in short supply. Furthermore, retransplantation for HAT is associated with significant morbidity and mortality.

Reconstruction of the hepatic artery, as reported by the group from Pittsburgh, therefore provides a valuable alternative option in the treatment of patients with HAT. The success rate of 88.5% is encouraging. Causative factors, other than inadequate surgical technique, that are associated with the occurrence of HAT include the presence of multiple arteries in either the donor or the recipient, small-diameter vessels, particularly in pediatric liver transplantation, severe rejection, and a raised hematocrit. The treatment of choice for any complication is prevention. Thus, the anastomosis of the HA, especially in pediatric recipients, should be performed by an experienced surgeon using meticulous technique and adequate magnification. Two additional techniques aimed at prevention that may be worthy of further evaluation are the role of ligation of the splenic artery, which supposedly prevents a steal phenomenon, and the possible value of routine intraoperative measurement of blood flow by means of a flow meter. Furthermore, continuous or repeated postoperative monitoring of HA blood flow to detect HAT as early as possible also needs evaluation.

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