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Operative Procedures

Hepatic Artery in Liver Transplantation

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ADVANCES in surgical techniques have contributed to the improved results of orthotopic liver transplantation (OLT_X) that have been realized over the last few years.[1] Examples include better understanding of anomalies of the hepatic arterial supply, development of methods of reconstructing them, and the use of vascular grafts and other innovative techniques of revascularization. Nonetheless, ~10% of liver grafts still are lost from arterial complications.[2] In this report, we describe our experience with the management and complications of the hepatic artery (HA) in the donors and recipients of 231 liver grafts procured and transplanted at the University of Pittsburgh.

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MATERIALS AND METHODS

Two hundred thirty-one hepatic grafts were procured between August 1, 1985 and April 30, 1986. Of the 231 grafts, 2 were sent to other centers and 1 was transplanted as an auxiliary graft. The remaining 228 grafts, consisting of 141 adult and 87 pediatric livers, were transplanted orthotopically into 192 patients. In 182, these were primary liver transplants (first OLT_X) (115 adult and 67 pediatric), whereas 46 were used for retransplantation ($ReOLT_X$) (26 adult and 20 pediatric). All recipients were treated with cyclosporine (Cs) and steroids. Monoclonal antilymphocyte globulin (ALG) (OKT_3, Orthelone) was administered to patients with intractable rejection. A chi-square test was used to compare statistically the rate of hepatic artery thrombosis with donor anomalies and type of reconstruction.

The technical details of the donor and recipient procedure performed at our institutions have already been reported.[1,3,4] In this report, the management of the HA during the liver transplant procedure is briefly outlined.

Donor Operation

Identification of an anomaly of the HA is the initial step of the operation. A left hepatic artery (LHA) arising from the left gastric artery (LGA) is easily visualized in the gastrohepatic ligament. A right hepatic artery (RHA) arising from the superior mesenteric artery (SMA) lies behind the portal vein (PV). Once the HA anatomy has been identified, careful dissection and preservation of the entire arterial system is begun. The normal HA (NHA) is mobilized by dividing the right gastric artery (RGA), the gastroduodenal artery (GDA), and more proximally, the LGA and the splenic artery (SPA). A LHA arising from the LGA is preserved by dividing the ascending and descending branches of the LGA adjacent to the gastric wall. A RHA arising from the SMA is preserved by tracing it back to its origin at the SMA and then dissecting the SMA from its aortic origin to several centimeters distal to the RHA. In all cases, it is desirable to remove the HA with a patch of aortic wall for the arterial reconstruction or, in the case of a double arterial system arising from the celiac axis (CA) and the SMA, to create a common trunk arterial system.[5] The iliac arteries (IA) are always removed for possible use as arterial grafts (AG). The abdominal aorta (AA) and/or the thoracic aorta (TA) may be removed in continuity with the HA in pediatric cases to serve as arterial conduits.

Preparation of the Liver at the Back Table

The entire HA is skeletonized only when the surgeon is at the back table for final preparation. It is then also examined for unexpected injuries. In the case of a RHA arising from the SMA, the common aortic patch that includes both the origins of the SMA and the CA is folded and anastomosed to create a single trunk.[5]

Recipient Operation

Generally, the existence of an anomaly in the recipient HA is not important if it has appropriate size and flow. In some cases with a recipient double hepatic arterial supply, however, branches may be inadequate to sustain flow to the graft if used alone. Arterialization of the hepatic graft is usually achieved by end-to-end anastomosis (E-E) between a small Carrel patch of the graft CA on the CA itself to the recipient HA. The bifurcation of the recipient GDA at the CHA is one preferred location for the E-E anastomosis (Fig 1). The GDA and the proper HA (PHA) are split and converted into a patch to obtain a suitable recipient for anastomosis. The bifurcation of the RHA and LHA at the PHA or the take-off of the SPA from the CA can similarly be used. The common HA (CHA) and CA trunks are also suitable sites. If the recipient HA is not appropriate for E-E anastomosis (eg, because of inadequate flow or a technical problem) a donor hepatic artery is anastomosed to the AG placed between the infrarenal AA and the hepatic hilum (Fig 2). The AG may be passed through one of three retropancreatic tunnels (Fig 3). The "short tunnel" is created behind the pancreatic neck to the left of the SMA and exits to the left of the PV. The "long tunnel" runs behind the head of the pancreas and exits to the right of the PV. The "intermediate tunnel" is located between the AA and the inferior vena cava to the right of the SMA and exits to the left of the PV.

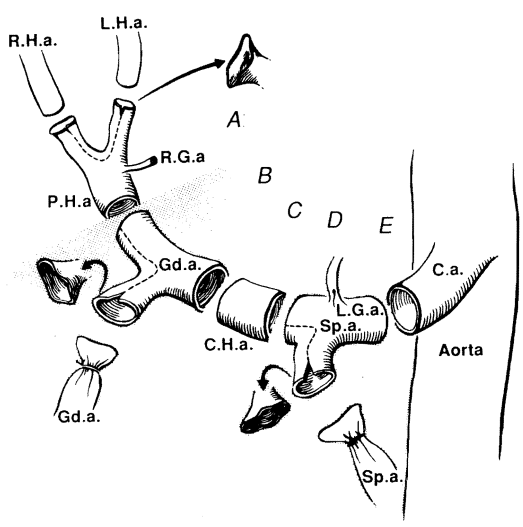

Fig 1. Site (A–E) and preparation of recipient hepatic artery for end-to-end anastomosis with a hepatic artery of the allograft.

RESULTS

Anomalies of the Donor Artery

Of the 231 procurements, 20 grafts (8.6%) had a single artery, but the determination of accompanying anomalies was incomplete during the donor operation. No hepatic allografts were discarded because of the presence of an anomaly. Variations in the HA anatomy of the remaining 211 grafts are depicted in Fig 4. A NHA was found in 136 donors (64.5%) (Fig 4A). Twenty-seven livers had a LHA arising from the LGA (12.8%) (Fig 4B), and 21 had a RHA arising from the SMA (9.9%) (Fig 4C). Both these anomalies occurred simultaneously in seven donor grafts (3.2%)

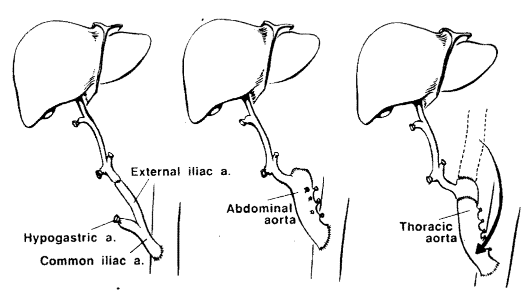

Fig 2. Use of iliac arterial graft, abdominal aortic, and thoracic aortic grafting between recipient aorta and allograft hepatic artery.

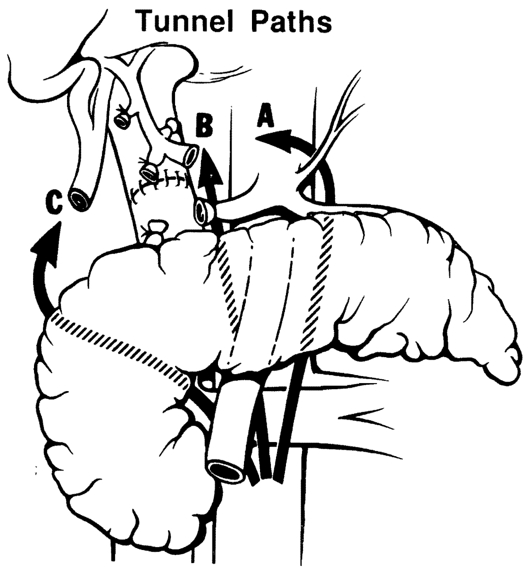

Fig 3. Three ways to make the retropancreatic tunnel for arterial graft use. A: Short tunnel. B: Intermediate tunnel. C: Long tunnel.

(Fig 4D). In 11 cases (5%), a CHA originating from the SMA was the only source of arterial blood supply to the liver (Fig 4E). In ten cases, a single CHA arose from the SMA; in one, both the RHA and the LHA arose from the SMA. Although most of the CHA arising from the SMA passed behind the pancreas and appeared in the hepaticoduodenal ligament from the right, 1 passed anterior to the common bile duct, and 1 ascended through the HDL from the left. Uncommon variations were found in 9 grafts (4.1%): a common trunk, 1 (0.5%) (Fig 4F-1); RHA from CA (2) (0.9%) (Fig 4F-2); RHA from AA (1) (0.5%) (Fig 4F-3); and LGA from AA (5) (2.3%) (Fig 4F-4), one of the latter giving an origin to a LHA.

Back Table Work

Creation of a common trunk using the foldover technique as previously described[5] was performed in 27 cases. In one case with the RHA from the AO, reconstruction was made anastomosing the aortic origin of the RHA to the SPA. There were five major arterial injuries involving anomalous arteries. Two grafts with RHA had a tear of the RHA at the origin of a small branch and were repaired by interrupted sutures. Three injuries, accidental transection of LHA (1) CHA with a LHA (1), and a RHA from the CA (1)

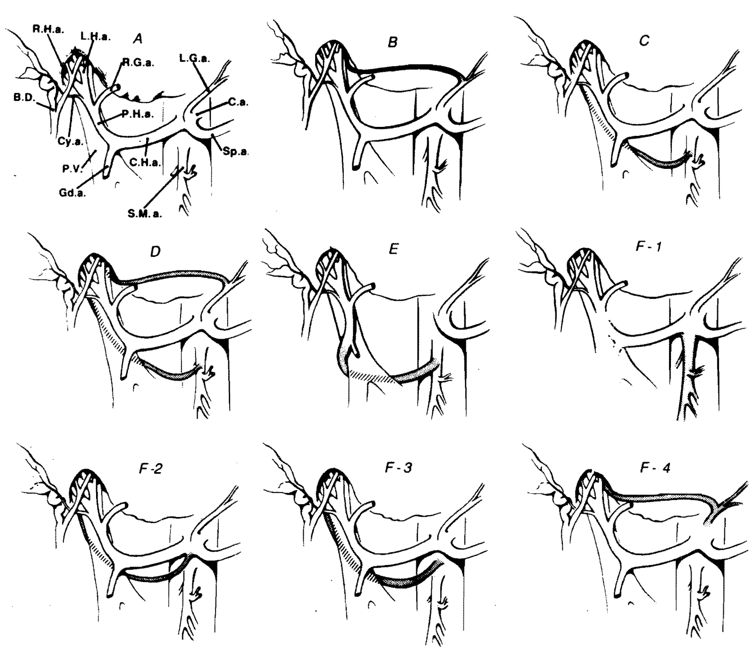

Fig 4. Anatomy of the donor hepatic artery.

occurred at the back table. The first two were reanastomosed, and the other was tied. There were no important complications after OLT_X in these cases.

Reconstruction of the Graft Artery

The method and frequency of arterial reconstruction undertaken in 228 hepatic allografts is shown in Table 1. An E-E was performed in 165 cases (72.4%), 113 adult OLT_X (80%) and 52 pediatric OLT_X (60%). Among 182 first OLT_X, an E-E was performed in 148 cases (81.3%), 101 adult (89%) and 47 pediatric (70%), but only 37% at $ReOLT_X$. The takeoff of the GDA was the most common location for the E-E at first OLT_X, 71% adult and 53% pediatric patients. After an initial E-E was performed, 13 cases had an inappropriate anastomosis or poor inflow. Reanastomosis to the proximal HA was done in six cases, and seven were reconstructed with a graft. The SPA of seven other cases was ligated to make the E-E at the CA or to obtain better inflow. Sixty-three OLT_X (27.6%) were reconstructed using AG, 28 adult and 35 pediatric patients. Seven of them were anastomosed with the AG placed at the previous OLT_X and one adult who had an hemangioendotheloid sarcoma was anastomosed by an E-E of the donor AA to an unusually large recipient HA. The incidence of graft use at first OLT_X and $ReOLT_X$ was 11.7% and 30%, respectively, in adults; in children, it was 53.8% and 75%, respectively. The reasons for this type of arterialization were small HA (26 cases, 8 adults and 18 children), poor inflow (13 cases, 11 adults and 2 children), intimal dissection (1 adult), and unsuitable recipient HA at $ReOLT_X$ (16, 5 adults and 11 children).

Hepatic Artery Complications

There were 25 complications in 23 patients (11%). One patient developed false aneurysms (HAA) after retransplantation for hepatic artery thrombosis (HAT), and the other had two episodes of HAT. Thrombosis occurred in 20 arteries (8.8%) in 19 patients, and 5 (2.2%) had HAA. There was no correlation of HAT with the original recipient disease except for biliary atresia, possibly because this is the most frequent indication for OLT_X in small children. There was no increased incidence of HA complications in the presence of HA anomalies in the donor.

The number of HA complications in relation to reconstructive methods are shown in Table 2. There were 13 HATs in children (14.9%) and seven in adults (5%). This higher incidence in children is statistically significant ($P < .02$), regardless of type of reconstruction. HAT is a special problem in children aged <1 year and occurred in 35% of these cases. Adult HAT occurred in $ReOLT_X$ (15.4%) more frequently than in the first OLT (2.6%) ($P < .05$). Although no statistical differences were noted between the E-E and AG reconstructions, adult OLT_X using IA were not complicated by HAT (0 of 22), whereas there was a 5% incidence of HAT using the

Table 1. Method and Frequency of the Reconstruction of Hepatic Artery in OLT_X

	Adult		Pediatric		Total	
Reconstruction	First OLT_X	$ReOLT_X$	First OLT_X	$ReOLT_X$	First OLT_X	$ReOLT_X$
E-E anastomosis	101	12	47	5	148	17
Arterial graft	14	14	20	15	34	29
IA	13	9	5	5	18	14
AA	0	0	7	3	7	3
TA	0	0	8	5	8	5
E-E*	1	5	0	2	1	7

OLT, orthotopic liver transplantation; IA, iliac artery; AA, abdominal aorta; TA, thoracic aorta.

*E-E, end-to-end anastomosis with the arterial graft placed at previous OLT_X.

Table 2. Number of Hepatic Arterial Complications at Respective Reconstructive Method

	Adult		Pediatric		Total	
Reconstruction	HAT	HAA	HAT	HAA	HAT	HAA
E-E anastomosis	6	1	7	0	13	1
Arterial graft	1	1	6	3	7	4
IA	0	1	1	0	1	1
AA	0	0	2	1	2	1
TA	0	0	3	2	3	2
E-E*	1	0	0	0	1	0

HAT, hepatic artery thrombosis; HAA, false aneurysm of the hepatic artery; other abbreviations as in Table 1.

*E-E, end-to-end anastomosis with the arterial graft placed at previous OLT_X.

E-E. The incidence of HAT with an E-E in children with 13.5% (7 of 52) as compared with 17.1% (6 of 35) with AG. There was no difference in HAT among children with AG with respect to the type of graft used (ie, iliac artery, abdominal or thoracic aortic conduit). The overall mortality of HAT was 58% (11 of 19). Eight were salvaged by $ReOLT_X$ whereas three others are still awaiting retransplantation. HAT and moderate rejection occurred simultaneously in two patients.

HAA occurred in 3 children and 2 adults and involved 1 E-E and 4 AG. HAA should be considered a different entity from HAT since local or systemic infection preceded the HAA in all patients. Three patients died of rupture of the HAA, 1 died of overwhelming sepsis, and 1 was saved by ligation of the HAA.

DISCUSSION

The incidence of anatomical variations of the hepatic artery arising from donor hepatectomies performed by a single transplant center approximates that reported by autopsy[6] and by angiogram findings.[7] The normal pattern described in anatomical literature actually occurred in ~65% of the donors in the present study, therefore approximately one-third of a large group of donors had HA anomalies. Michels classified anomalous hepatic arteries into the "accessory" or the "replacing" type.[6] The accessory type is an artery that provides blood supply along with a main HA, whereas the replacing type artery is the only source of blood to a segment or lobe of the entire liver. About one-half of all LHA and RHA are thought to be replacing arteries. A typical replacing artery is a CHA from the SMA. Ligation of such an artery would result in complete deprivation of hepatic arterial blood flow.

Careful removal of vascular grafts from all donors cannot be overemphasized, since their availability becomes critical in the event of unexpected problems at the time of revascularization of the allograft.[8] The methods for HA reconstruction using arterial grafts have evolved over time. Originally, the proximal subdiaphragmatic aorta was the site of anastomosis of the AG. More recently, the aorta has been approached from the right side, exposing the infrarenal AA above the inferior mesenteric artery.[9] This approach may often be complicated by significant bleeding, especially in patients with portal hypertension. The newest method, now used exclusively at this institution, involves standard direct aortic exposure and creation of a retropancreatic tunnel. This approach allows the safest and simplest method of liver arterialization using a vascular graft.

The clinical features of HAT have been reported previously.[2] Although our initial study suggested that donor factors such as graft anomalies or the creation of a common trunk produced an increased incidence of HAT, no such relationship was found in this study. The following preexisting recipient factors must be considered causes of HAT. First, the incidence of HAT in small pediatric cases is high regardless of the type of reconstruction used, implying that the size of the HA and hemodynamics after arterialization are the most important predisposing factors. Second, many patients with primary biliary cirrhosis have delicate visceral arteries leading to intimal dissection even with the most careful vascular technique. Although no correlation could be identified, the recipient's primary disease must always be an important consideration. Third, a redundent HA may cause HAT by kinking.[10] The HA length should be tailored to produce the shortest HA of suitable diameter required to perform a perfect anastomosis. Fourth, uncontrolled rejection is yet another cause of HAT.[10] Frequent monitoring and control of the immunological reaction of the liver should minimize this potential complication factor. Finally, poor arterial inflow must be considered the most important factor. The inflow must be carefully assessed both clinically and perhaps by flowmeter and must be corrected by an AG if found to be inadequate. Perfect technique and good arterial flow to the hepatic graft are the mainstays of successful HA management.

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