Published in final edited form as:
Transplant Proc. 1989 June ; 21(3): 3511–3513.

Is Hepatic Artery Thrombosis After Liver Transplantation Really a Surgical Complication?

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The complication of hepatic arterial thrombosis (HAT) leads to an almost universally fatal outcome without retransplantation.[1-4] Although this problem occurs in both adults and children, its incidence is significantly higher in the pediatric population, where it remains one of the major causes of mortality and retransplantation.[1,3] Some of the known causes of HAT after orthotopic liver transplantation (OLT) are purely technical. These include clamp traction on the recipient hepatic artery (HA), faulty placement of anastomotic sutures, and the inappropriate angulation of the donor HA or anastomotic site.[2,5]

Certain non-technical factors have been reported to predispose to HAT after OLT.[1-3,6] In this analysis, these factors were investigated in 13 patients who sustained HAT either during or after OLT and who underwent an attempt at secondary arterial reconstruction.

Materials and Methods

During the 1-year period between January 1 and December 31, 1987, 323 patients underwent 389 OLT at the University of Pittsburgh Health Center. HAT developed in 37 of these grafts (9.5%) during or shortly after OLT. The incidence was 5.7% in adults (16/282 patients) and 19.6% in children (21/107 patients). An attempt at HAT reconstruction was made in 13 of these 37 patients (35.1%). This was performed during the primary transplantation procedure in 6 patients and 4.7 ± 3.5 days later (mean ± SD; range 1 to 12) in the other 7 patients. The patients were aged between 17 months and 58 years, with a mean age of 22.0 years; 7 were female (53.8%). Objective evaluation of the etiology and factors responsible for the development of HAT in these 13 patients was undertaken at the time of reconstruction. The evaluation included HA blood flow measurements using an electromagnetic flowmeter[10] and revision of the HA anastomosis with close inspection of the anastomotic site.

Immediately following surgery, all pediatric patients were given, intravenous low molecular weight dextran 5-10 mL/kg for 4 days, heparin 50 U/kg subcutaneously every 12 hours throughout the hospital stay (approximately 4 weeks), aspirin 20-40 mg/day by mouth or nasogastric tube for at least 3 months, and dipyridamole (Persantine) 12.5-25 mg/day by mouth for at least 3 months. The above anticoagulation or antiplatelet therapy was discontinued if the patient demonstrated any clinical or laboratory evidence of coagulopathy. All adult patients who underwent OLT for Budd-Chiari syndrome received anticoagulation with heparin 5,000 units subcutaneously, three times a day, followed by Coumadin in a dose that maintained the prothrombin time at around 18 seconds.

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Results

Table 1 lists the clinical data of the patients who developed HAT during the actual transplant procedure. Possible causes of HAT in these patients included one case of poor inflow related to an arterial anomaly in the recipient, one case of rotation of an aortohepatic interposition graft in the retropancreatic tunnel, one case of intimal dissection of the recipient common HA due to excessive traction, and one case of disseminated intravascular coagulation (DIC) of the donor. There were no obvious significant factors in two other cases.

Table 2 lists the clinical data of the patients who developed HAT following OLT. Possible etiologic factors included one case of poor inflow related to an arterial anomaly of the recipient, one case of a marked discrepancy in size between the donor celiac axis and a donor iliac artery which had been used as an aortohepatic interposition graft, one case involving an end-to-side anastomosis, one case involving kinking of the anastomosis; and one instance of an infected donor hepatic artery. Two other patients did not reveal any obvious causes.

The etiology of HAT among the 13 patients was thus ascribed to purely technical factors in 5 (38.5%), poor inflow related to an arterial anomaly of the recipient in 2 (15.4%), DIC of the donor in 1 (7.7%), infection of the donor HA in 1 (7.7%) and unknown causes in 4 patients (30.8%).

Discussion

Other authors have reported multiple causes of HAT, including poor technical performance, uncontrolled rejection, high postoperative hematocrit, and in cases of pediatric OLT, small caliber of the vessels with associated low flow (Table 3).[1-3,6,7,10]

Untreated rejection is associated with marked reduction of hepatic blood flow in dogs following OLT, presumably due to increased resistance in the hepatic vascular tree.[8] This would imply that uncontrolled rejection may be one of the pathogenic factors of HAT in humans.[2,9]

Tisone et al[6] recently reported a correlation between HAT after OLT and a high hematocrit. A significantly higher incidence of HAT (24%) was observed in patients whose immediate postoperative hematocrit exceeded 44%, compared with an incidence of 3% in patients with a lower hematocrit. Overtransfusion, dehydration, or the combination of both may contribute to the higher incidence of HAT among these patients.

The etiology of HAT in small children remains to be elucidated. Measurements of hepatic blood flow revealed an extremely high incidence of HAT in grafts with HA flows less than 60 mL/min (5 out of 6 patients, 83.3%).[10] Further analysis of graft hemodynamics as an etiologic factor is in progress.

In adults, if the HA blood flow is 200 mL/min or less, we pursue measures to increase the arterial inflow. These include the placement of an aortohepatic interposition arterial graft or ligation of the splenic artery.

A recent study evaluating the effects of different preservation fluids on liver allografts demonstrated a significantly reduced incidence of HAT in livers preserved in University of Wisconsin (UW) solution, as compared with those stored in Euro-Collins solution (4.6% vs 12.5%, $p < 0.05$).[11] This may indicate that intimal damage or an increase in HA resistance from cellular swelling during storage[12] is a contributory factor to the development of HAT.

The cases presented in this report offered a unique opportunity to examine the possible causes of intraoperative or postoperative HAT at the time of attempted reconstruction. Nontechnical

factors, although less frequent than technical failures, accounted for or played a contributory role in the development of HAT in 4 of 13 patients (30.8%).

Acknowledgments

Supported by Research Grants from the Veterans Administration and Project Grant No. AM 29961 from the National Institute of Health, Bethesda, MD.

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Table 1

Clinical Data of Patients Who Developed Hepatic Artery Thrombosis and Underwent Reconstruction During OLT

Case	Age/Sex	Liver Disease	HA Anomaly (D/R)	Initial HA Anastomosis (D/R)	Possible Cause of HAT
1	29/M	CAH	—/R, P&LHA	CA/CHA	Poor inflow
2	19/F	Wilson	R&PHA/R&PHA	CA/AHIG	Rotation of AHIG
3	23 mo/F	CBA	R&PHA/—	CA/CHA	Unknown
4	21/M	Cystic fibrosis	—/—	CA/AHIG	DIC in the donor
5	17 mo/M	CBA	—/—	CA/CHA	Unknown
6	56/M	CAH	—/—	CA/CHA	Intimal dissection

HA = hepatic artery; D/R = donor/recipient; CAH = chronic active hepatitis; CBA = congenital biliary atresia; RHA = right hepatic artery; PHA = proper hepatic artery; LAH = left hepatic artery; CHA = common hepatic artery; AHIG = aortohepatic interposition graft; DIC = disseminated intravascular coagulation.

Table 2

Clinical Data of Patients Who Developed Hepatic Artery Thrombosis and Underwent Reconstruction Following OLT

Case	Age/Sex	Liver Disease	HA Anomaly	Initial HA Anastomosis	POD#	Possible Cause
1	34/F	PBC	—/R&PHA	CA/RHA	12	Poor inflow
2	30 mo/F	CBA	—/—	CA/CHA	4	Unknown
3	17 mo/M	CBA	—/—	CA/CHA	1	Unknown
4	58/F	PBC	—/—	CA/AHIG	3	Stricture due to size discrepancy
5	18/M	FLF	—/—	CA/AHIG	4	End-to-side HA anastomosis
6	4/F	FLF	—/—	CA/CHA	5	Infection
7	38/F	PSC	R&PHA/—	Distal SMA/CHA (fold over)	4	Kinking of anastomosis

HA = hepatic artery; D/R = donor/recipient; PBC = primary biliary cirrhosis; RHA = right hepatic artery; PHA = proper hepatic artery; CA = celiac axis; CBA = congenital biliary atresia; CHA = common hepatic artery; AHIG = aortohepatic interposition graft; FLF = fulminant liver failure; PSC = primary sclerosing cholangitis; SMA = superior mesenteric artery.

Table 3
Possible Causes of Hepatic Artery Thrombosis Following OLT

Possible Cause	Surgical Factor	Medical Factor
Inflow problem	rHA intimal dissection	rHA anomalies, ?rHA hypoplasia (children)
Anastomosis	Inversion, stricture	
	Kinking	
Donor HA	Intimal dissection	
	Kinking, HA anomalies	
Parenchymal run-off		s/p HAR for HAT, rejection, preservation, ?ischemia, donor DIC

rHA = recipient hepatic artery; HAR = hepatic artery reconstruction; HAT = hepatic artery thrombosis; DIC = disseminated intravascular coagulation.