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THE USE OF PULMONARY ARTERY SEQUESTRATION AS AN HEPATIC ARTERIAL CONDUIT

A CASE OF UNUSUAL HEPATIC ARTERIAL SUPPLY[1]

J. P. is a 20-month-old Portuguese male patient who presented with a diagnosis of biliary atresia. Approximately 10 months previously the patient had undergone a laparotomy and cholecystectomy for unexplained jaundice. At the time of surgery the diagnosis of biliary atresia was established, and the patient was later transferred for liver transplantation evaluation.

The physical examination showed a deeply jaundiced infant male patient in no apparent distress. Systolic blood pressure was 94, heart rate 102, respiratory rate 28, temperature 35.9°C, and the weight was 8.9 kg. The sclera were icteric, neck supple, lung fields were clear bilaterally, heart was regular and without murmurs. Abdominal girth was 52 cm with obvious superficial venous collaterals. The liver and spleen were enlarged and easily palpable.

Laboratory studies included: Hct 32%, WBC 9.0, Plt 249K, total bilirubin 16.2, GGTP 194, SGOT 268, SGPT 93, and AP 856. Blood urea nitrogen, creatinine, amylase, and albumin were all within normal limits.

Ultrasonography revealed a cirrhotic liver with patent hepatic vessels and no bile-duct dilatation.

The patient was transplanted on 7/15/87 without complications. The arterial anastomosis was performed between the donor abdominal aorta and the celiac axis of the recipient (after ligation of the donor left gastric and splenic arteries). On the 1st postoperative day the patient had an ultrasound that showed no pulsation in the hepatic artery. Arterial thrombosis was confirmed by angiography.

The patient was retransplanted the following day (7/17/87) with a liver that was slightly smaller than ideal. During the harvesting of the liver, it was noted that the donor (a 12-month-old child) had significant pericardial and right pleural adhesions. During the final stages of harvesting the liver, a large artery was found entering the right lower lobe of the lung from the diaphragm, an obvious pulmonary sequestration. The artery was preserved and was later found to be emanating from the celiac axis. In addition, the liver had a triple arterial supply: an hepatic artery from the celiac, a left hepatic branch from the left gastric artery, and a right hepatic branch from the

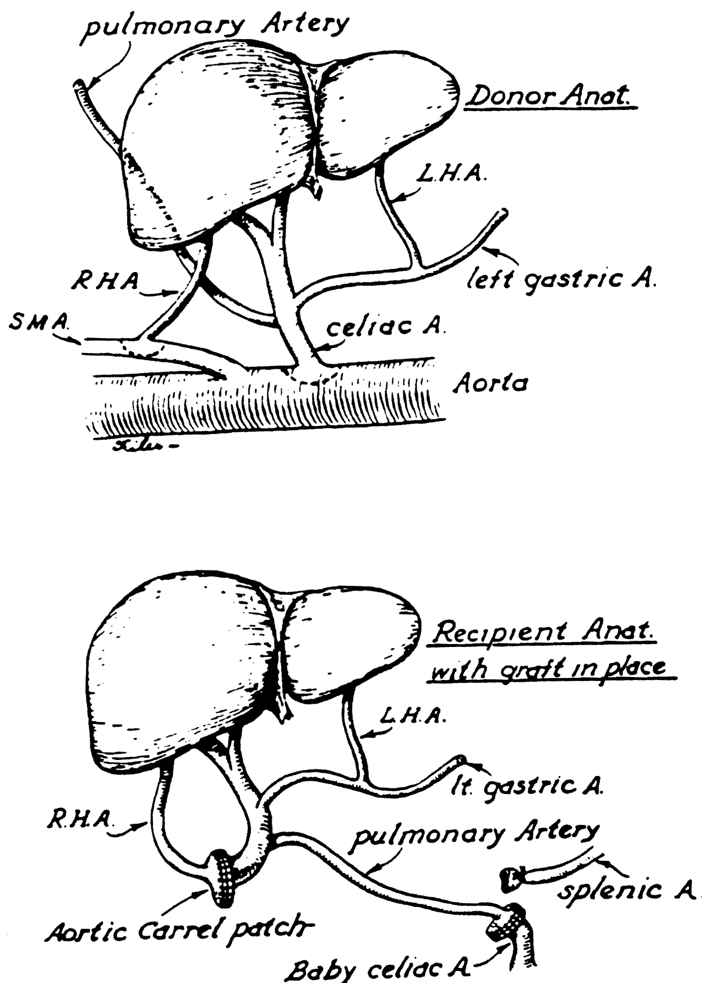

FIGURE 1. Donor anatomy (above) with final reconstruction (below). RHA: right hepatic artery; LHA: left hepatic artery; and SMA: superior mesenteric artery.

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superior mesenteric artery. A modification of the fold-over technique (*1*) was done, but because of the size of the liver there was not adequate length to comfortably anastomose this complex to the recipient celiac axis. For this reason, the pulmonary artery originating from the donor celiac axis was used to make the anastomosis (Fig. 1).

The patient recovered from this 2nd transplantation uneventfully and was discharged from the hospital on 8/23/87 with normal liver function. The patency of his arterial complex was established 4 times postoperatively by ultrasound. On his last out-patient visit, he was doing well with a bilirubin of 0.4, PT 11.7 sec, SGOT 19, SGPT 10, and a GTP 16.

The celiac axis as the origin of a pulmonary arterial sequestration is present 1% of the time (*2*). As symptoms of pulmonary sequestration almost always present early in life with complications leading to corrective surgery, this anomaly as a harvesting enigma will probably be found only in children. As was shown here arterial anomalies do not preclude the use of the hepatic graft and can often be used advantageously.

Donor organs should not be discarded secondary to vascular anomalies. A thorough understanding of reconstructing the hepatic arterial supply can be both organ- and life-saving.

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SELECTIVE DEPLETION OF KUPFFER CELLS IN MICE BY INTACT RICIN

Although the results of clinical liver transplantation have improved, rejection episodes occur in 35–75% of recipients (*1, 2*). While Kupffer cells, endothelial cells, and bile duct cells express the class II (Ia) antigens of the major histocompatability complex (*3, 4*), which are important in stimulating the immune response (*5*), hepatocytes do not express these antigens. Recent evidence has implicated transplanted donor Kupffer cells as an important mediator of hepatic allograft rejection (*6*). These liver macrophages may therefore act as the "passenger leukocyte" of hepatic transplantation. Selectively depleting these cells in donor hepatic tissue may therefore decrease donor liver immunogenicity.

Ribosomal inactivating proteins are toxins that are able to kill cells in a 1:1 ratio by enzymatically inhibiting the 60S subunit of the ribosome (*7, 8*). We have recently used one such toxin, the A-chain of ricin, to selectively deplete Kupffer cells in rats and mice (*9, 10*). The A-chain is a mannosylated glycoprotein targeted specifically to Kupffer cells presumably by the Kupffer cell (macrophage) mannose receptor. After 20 mg/kg ricin A-chain was given intraportally, Kupffer cells in mice were successfully reduced by 33% with no evidence of hepatic parenchymal damage. This effect persisted for at least 3 days with recovery evident at 1 week. Unfortunately, higher doses of ricin A-chain were not practical due to the development of nonspecific uptake and systemic toxicity. In rats, a similar reduction in Kupffer cells was observed after injection of 6 mg/kg ricin A-chain intravenously but only after simultaneous bilateral nephrectomy (*10*).

This toxicity prompted us to search for a more direct and specific toxin to Kupffer cells. In vitro studies have shown that intact ricin, a 65,000-dalton heterodimer made up of an A- and B-chain, is several times more toxic to Kupffer cells than to hepatocytes (*11, 12*). In vivo studies have also shown that labeled intact ricin injected intravenously into rats is preferentially incorporated by the nonparenchymal cells of the liver. Also, rat sinusoidal cells have been shown in vivo to be more sensitive to ricin than are the parenchymal cells (*13, 14*). This raised the possibility of using low doses of intact ricin as a toxin specific to Kupffer cells.

Ricin was purified from castor beans (*Ricinus communis*) by a protocol previously described (*15*). Briefly, a crude extract of ricin was bound to a 1.5×40-cm galactose-sepharose column in 10 mM Tris-HCl, pH 7.7. Purified ricin was then eluted with a linear D-galactose, 800 ml, 0–0.12 M gradient. Purity was confirmed by the presence of a single 65-kilodalton band on 9.0% SDS-polyacrylamide gel electrophoresis, and by toxicity to cultured rabbit alveolar macrophages. The LD50 (dose at which the mortality rate is 50%) of ricin is 2.7 μg/kg (*14*). Doses of 1–2.5 μg/kg were used in the present study.

Female Balb/c mice weighing 20 g (Sasco, Indianapolis, IN) were grouped into control and treated groups. Single intraportal injections of saline (n=4) or ricin at the doses described below (n=26) were administered 24 hr prior to assay of Kupffer cell numbers. Kupffer cell numbers were subsequently determined by phagocytosis of intravenously injected carbon particles. India ink (0.1 cc) (Higgins Ink, Faber-Castell Corp., Newark, NJ),