42 patients received cyclosporine. Our current one-year graft survival in all recipients of LRD allografts is 95%, cyclosporine being used in all except HLA-identical recipients.

Twenty-one patients (50%) currently have functioning allografts 1–14 years posttransplantation (mean: 5 years). Allograft failure occurred in eight patients within one year following transplantation. In two of these patients, arterial occlusion resulted in graft loss at 1 and 3 days, respectively. In the remaining six patients, rejection was responsible for graft loss. In three of these six patients, immunosuppression was less than optimum because of concurrent sepsis. A fourth patient was noncompliant with her immunosuppressive medications. Chronic rejection led to graft loss at 20 months in one patient and at 6, 7, and 14 years posttransplantation in three patients respectively.

In summary, no adverse affect upon patient survival was noted due to the presence of MRA. MRA, however, adversely affected graft survival in two cases. One allograft was lost in the early postoperative period due to infarction following repair of a completely transected superior polar vessel. It must be stressed that this complication has not occurred since 1976, and therefore, as more experience was gained, injury to these small vessels has been avoided. In the second patient, ligation of a superior polar vessel with resultant hypertension may have contributed to allograft loss 20 months following transplantation.

The presence of MRA and the additional time required for arterial reconstruction contributed to postoperative ATN in only one patient, who required a venous patch angioplasty prior to arterial reconstruction. In our recent experience, renovascular hypertension secondary to RAS occurred in 39 of 547 renal allograft recipients (7.1%) (2). In the present series, RAS was present in 5% of patients. In all cases RAS occurred proximal to the arterial reconstruction.

The most frequent urologic complication in the present series was distal ureteral fistula (in three patients). Interference with blood flow to the ureter may have occurred during dissection of the inferior polar artery. The presence of multiple renal arteries has previously been recognized as contributing to the formation of ureteral fistulae (3). Concurrent rejection in one patient may also have further affected blood flow to the ureter. No allografts were lost secondary to urologic complications.

In conclusion, the present study demonstrates that the use of donors with MRA is associated with increased risk to the allograft. Nonetheless, satisfactory patient and graft survival was achieved. Exclusion of donors based upon the presence of MRA would deny a large number of patients the advantages of an LRD renal allograft, 9% in our series, and place further demands upon the limited supply of cadaveric renal allografts.

ALLAN M. ROZA[1]
LEONARD J. PERLOFF
ALI NAJI
ROBERT A. GROSSMAN
CLYDE F. BARKER
Departments of Surgery and Medicine
University of Pennsylvania
Philadelphia, Pennsylvania 19104

[1] Address correspondence to: Allan M. Roza, M.D., Division of Transplantation, Froedtert Memorial Lutheran Hospital, 9200 West Wisconsin Avenue, Milwaukee, Wisconsin 53226.

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PORTAL VEIN THROMBOEMBOLISM OF LIVER ALLOGRAFTS FROM SPLENECTOMIZED DONORS[1]

Since first reported by Delatour (1) in 1895, thrombosis of the splenoportal venous system has been known as a serious complication of splenectomy. This complication primarily affects patients with congestive splenomegaly and myeloproliferative disorders (2, 3), and is supposedly rare after splenectomy for trauma or incidental injury during laparotomy (4, 5). We describe two patients who underwent splenectomy for trauma, in whom thromboembolism of the portal vein was identified later in the livers harvested for liver transplantation.

Case 1. A 13-year-old boy was involved in a traffic accident on July 1, 1987, and suffered from multiple trauma. The patient underwent splenectomy, in addition to other procedures. Because of irreversible head injury, the patient became a multiple organ donor, and the heart, liver and left kidney, were procured. The laboratory data before the organ donation were: SGOT 100 IU/L, SGPT 46 IU/L, total bilirubin 1.7 mg/dl, prothrombin time 13.7 sec, partial thromboplastin time 24.8 sec, leuko-

cyte count 12.5×10^3/mm³, hematocrit level 42.4%. The platelet count was not measured. At the time of organ procurement, the liver appeared grossly normal and soft on palpation, and the intestine showed no evidence of venous congestion or edema. A small amount of serosanguinous fluid was seen in the peritoneal cavity. In view of the extensive retroperitoneal hematoma, a rapid flush technique (6) was used: Briefly, the portal cannula was inserted into the portal vein through the inferior mesenteric vein; following cross-clamp of the supraceliac aorta, the intraabdominal organs were flushed with cold solution from the portal as well as terminal aortic cannulae. The allograft hepatectomy was uneventful with immediate blanching and fast cooling of the liver. The liver was then transplanted into a recipient with Laennec's cirrhosis. When the donor portal vein was transected prior to its anastomosis with the recipient portal vein, a fresh clot was found at the cut margin. The clot, which was cylindrical and measured 6.2×0.4×0.2 cm, was removed and a Fogarty catheter was passed into the allograft portal vein, which showed no evidence of residual thromboemboli. Following the portal vein anastomosis, the liver allograft was revascularized, and excellent reperfusion was observed. The

liver allograft functioned well and the recipient's postoperative recovery was uneventful except for an episode of rejection. An ultrasonograph performed on the eleventh postoperative day showed no evidence of portal vein thrombi. The recipient was discharged 48 days postoperatively with good allograft function.

Case 2. A 19-year-old male patient was involved in a motorcycle accident on December 27, 1987, in which he suffered from closed-head injury and blunt abdominal trauma. The patient underwent splenectomy for lacerated spleen. Because of irreversible brain damage, he became a multiple organ donor. The laboratory data prior to the organ donation were: SGOT 73 IU/L, SGPT 46 IU/L, total bilirubin 0.7 mg/dl, prothrombin time 12.8 sec, partial thromboplastin time 26.9 sec, leukocyte count $15.8 \times 10^3/mm^3$, hematocrit level 33.9%, and platelet count $146 \times 10^3/mm^3$. The liver and intestines appeared normal during organ recovery. The pancreas felt firm, but a biopsy of the pancreas showed no evidence of pancreatitis. No ascites was present. The portal cannula was inserted through the splenic vein after caudad mobilization of the pancreas. At the time of cannulation of the splenic vein, no clot was encountered. The supraceliac abdominal aorta was cross-clamped, and the intraabdominal organs were flushed with cold solution from the portal and terminal aortic cannulae. The liver blanched and cooled down fast, and hepatectomy was performed without difficulty. When the graft was brought back and a redundant portion of the donor portal vein was excised immediately before portal vein anastomosis, a semiorganized clot measuring $2.0 \times 0.8 \times 0.8$ cm was found lying loose in the donor portal vein. The clot was removed and the portal vein anastomosis was completed. The allograft perfused and functioned well. A Doppler ultrasonograph performed on the eighth postoperative day showed patent portal vein without any evidence of portal vein thromboemboli. The recipient was discharged 28 days postoperatively with good graft function.

Both donors were victims of multiple trauma in traffic accidents, for whom splenectomy was performed for blunt abdominal trauma. At the time of multiple organ donation, the liver and intestines showed no evidence of pathology, and liver allografts were harvested without technical difficulty. The intervals between splenectomy and multiple organ donation was 1 and 4 days, respectively. Fresh or semi-organized clots were identified lying loose in the portal vein of the liver allografts immediately before the portal vein anastomosis in the recipients.

It is unlikely that the donors had portal or superior mesenteric vein thrombosis before multiple organ donation, since the liver and intestines looked grossly normal, no bloody ascites or melena was observed, and posttransplant liver functions in the recipients of the livers were excellent without ischemic damage. Also, formation of a thrombus in the portal or superior mesenteric vein during liver harvesting in case 1 is unlikely, since the portal cannula was inserted immediately before cross-clamping of the aorta, without interfering with the superior mesenteric venous flow, and since 3 mg/kg of heparin was administered immediately after the portal cannulation.

Salter et al. (*4*) reported splenic vein thrombosis after splenectomy in 3 out of 7 patients (42.9%) on autopsy, and Broe et al. (*3*) described 2 patients in whom postmortem examination suggested the extension of thrombus from the splenic vein remnant to the portal vein. Since the splenic vein after the

splenectomy becomes a cul-de-sac with very low flow, it is not surprising for a thrombus to form in this remnant structure (*3*). Allograft hepatectomy is associated with significant manipulation of the splenoportal venous system. The size of the cylindrical thromboemboli in the allograft portal vein, $6.2 \times 0.4 \times 0.2$ cm in case 1 and $2.0 \times 0.8 \times 0.8$ cm in case 2, seems to correlate with the size of the residual splenic vein in case 1, and the size of the splenic vein remnant proximal to the distal splenic ligature placed for cannulation of the portal cannula through the splenic vein in case 2. Although not confirmatory, it is suggested that the thromboemboli in our donors were formed in the residual splenic vein, and dislodged into the portal vein during liver allograft harvesting.

Although portal vein thrombosis has been rare after liver transplantation in our experience (*7*), it can occur, and when it does, there is usually no good explanation for a thrombosis that apparently began in the liver allograft and extended downward. Emboli already present in the graft at the time of organ procurement could be responsible.

When liver allografts are harvested from splenectomized donors, manipulation of the remnant splenic vein or the pancreas should be minimized, and the portal vein of the graft should be examined immediately after allograft hepatectomy in the donor hospital, in order to avoid a disastrous portal vein thrombosis in the recipient.

KATSUHIKO YANAGA
ANDREI STIEBER
BABURAO KONERU
LUIS A. MIELES
ANDREAS G. TZAKIS
THOMAS E. STARZL[2]
Department of Surgery
University Health Center of Pittsburgh
University of Pittsburgh
Pittsburgh, Pennsylvania 15213

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