Liver Transplantation in Patients With Portal Vein Thrombosis and Central Portacaval Shunts

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The authors have analyzed the impact of pre-existing portal vein pathology on the outcome of orthotopic liver transplantation. The incidence was high in patients suffering from chronic active hepatitis, hypercoagulable states, trauma or previous dissection of the porta hepatis, and splenectomy. The existence of portal vein thrombosis (23 patients) or surgical central portosystemic shunt (10 patients) was documented by preoperative Doppler sonogram or angiography (26/33), or operative findings of occluded vein (7/33). Successful thrombectomy and dismantling of portacaval shunts were achieved in most cases (24/33). Only nine patients required the placement of an interposition vein graft to the superior mesenteric vein. The intraoperative course was characterized by increased blood loss and coagulopathy, significantly higher than in patients with a patent portal vein. When compared with all liver transplants, the immediate postoperative complication rate was higher for primary nonfunction (33% *versus* 8%), re-exploration for intraperitoneal bleeding and hematomas, and morbid infections. Rethrombosis rate of thrombectomized veins or vein graft was low (2/33). The mortality rate was 35% in the presence of portal vein thrombosis (PVT) and 30% for portacaval shuct (PCS), both significantly higher than the 12% for other orthotopic liver transplant (OLT) patients. These results are expected to improve with better patient selection, surgical experience, and anticipation of the complex postoperative course. The authors conclude that PVT or the presence of PCS are not contraindications to orthotopic liver transplantation.

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PRE-EXISTING PATHOLOGY OF the portal vein increases the surgical complexity of orthotopic liver transplantation (OLT) and may have a substantial impact on the perioperative morbidity and mortality rates.[1,2] Such abnormalities are defined as the preoperative or intraoperative findings of portal vein thrombosis (PVT) or the presence of central portosystemic shunts (portacaval shunt, PCS). This group of patients is considered to represent a higher surgical risk, and knowledge of such pathology during the evaluation process weighs on the decision of acceptance for OLT. In this study we describe evaluation, operative aspects, and the immediate and late postoperative complications of preoperative portal vein pathology. The purpose of this report is (1) to determine the impact of pre-existing portal vein pathology on the surgical procedure, (2) to identify specific preventable perioperative complications, (3) to describe reconstructive modalities for portal revascularization, and (4) based on our results, to determine whether this group of patients with end-stage liver disease are acceptable candidates for OLT.

Materials and Methods

The charts of 550 consecutive patients undergoing OLT at UCLA between 1986 and 1990 were examined. The patient population included 364 adults and 186 children, who underwent a total of 676 OLTs. Patency of the portal vein was determined in each case by ultrasound examination until 1986, and duplex sonogram since then. The findings of possible portal vein occlusion or the knowledge of previous portosystemic shunt were indications for selective celiac and superior mesenteric artery angiograms with plain and subtraction views of the venous phase. More recently magnetic resonance imaging or hemodynamic labeled ammonia nuclear scan were used in a few pediatric and adult patients. Information about the donor and recipient liver venous anatomy is detailed in operative fact sheets from both the donor and recipient operations and are available for all OLTs performed at UCLA. The method for portal vein reconstruction, including the exact anastomotic site between the recipient and the donor vein, successful performance of portal vein thrombectomy, and the placement of allogeneic iliac vein graft, was documented in each case. Postoperative follow-up included

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routine evaluation of portal vein patency, which was assessed in all patients by serial Doppler sonograms (duplex) studies and, if questionable, by selective venous phase angiography. Operative reports of redo cases and autopsy findings of early and late deaths were reviewed to determine the patency of the portal vein at the time of re-exploration or death. The overall intensive care unit (ICU) and hospital stay and complications directly related to the portal vein reconstruction were considered as important variables contributing to postoperative morbidity.

Operative Reconstruction of the Portal Vein

Techniques of recipient hepatectomy and grafting were employed as was previously described.[3] After dissection of the porta hepatis and assessment of the portal vein status, the patient was put on partial venous–venous bypass by cannulating the femoral and axillary veins.

Thrombosis of the portal vein was approached by dissection of its entire length to the confluence of the splenic and superior mesenteric veins (SMV). The portal vein then was incised transversely just inferior to its bifurcation, and thrombectomy was accomplished using endarterectomy spatula, Cannon strippers, or Fogarty balloon catheters. Successful removal of the thrombus, which was organized in most cases, allowed the insertion of a portal vein cannula and placement of the patient on full bypass (venous inflow through the portal and the iliac veins). In three cases, the portal cannula was not inserted because of fragility of the vein, or failure to detect intestinal congestion. Such patients were left on partial venous bypass; however, the thrombectomized vein was used as a conduit for portal blood inflow to the liver.

Failed thrombectomy or an absence of the portal vein were managed by placement of a donor iliac vein graft between the recipient SMV and donor portal vein. The vein graft was placed immediately after performance of the recipient hepatectomy and before grafting. During this period the patient was placed on full bypass using the inferior mesenteric vein (IMV) for portal inflow. Cannulas were never inserted into the vein graft.

Dismantling portacaval shunts was performed as previously described by our group.[2] The length of the recipient portal vein and its patency determined the need for interposition of an iliac vein graft. It was possible to insert a portal vein cannula and place the patient on full bypass in most of these patients.

Rethrombosis of a thrombectomized portal vein was managed by placement of iliac vein graft to the SMV.

TABLE 1. *Incidence of Pre-existing Portal Vein Pathology*

	Portal Vein Thrombosis	Portocaval Shunt
Adult patients (n = 28)		
Chronic active hepatitis	14	6
Primary biliary cirrhosis		2
Familial cholestasis	1	
Alcoholic cirrhosis	1	1
Budd–Chiari syndrome		1
Trauma	2	
Pediatric patients (n = 5)		
Biliary atresia	3	
α-1 antitrypsin deficiency	1	
Trauma	1	

TABLE 2. *Factors Associated With the Development of Portal Vein Thrombosis*

Factor	No.
Hematologic abnormalities (n = 3)	
Antithrombin III	1
Sickle cell	1
Polycythemia vera	1
Upper abdominal surgery (n = 14)	
Splenectomy	3
Trauma/iatrogenic/previous orthotopic liver transplantation	4
Kasai portoenterostomy	3
Cholecystectomy and bile duct exploration	4
Tumor thrombus (n = 2)	2

Results

Incidence of PVT and PCS

Pre-existing pathology of the portal vein was found over a wide spectrum of liver disease (Table 1). Of the 28 adult patients, the incidence of portal vein thrombosis or portacaval shunts was highest among those suffering from chronic active hepatitis (CAH, 20/28 patients, or 71%). This finding is different than the distribution of CAH, which encompasses 45% of all adult liver transplants. In contrast the portal vein was found to be patent in all cases of primary biliary cirrhosis and sclerosing cholangitis (22.5% of all adult liver transplants). In the pediatric population, the portal vein was thrombosed with cavernous transformation in three pediatric patients suffering from biliary atresia and in one child with α-1 antitrypsin deficiency.

A total of 23 patients were found to have an occluded or absent portal vein. Factors directly implicated in the development of PVT were found in 10 of 23 patients (Table 2). These patients were found to suffer from a hypercoagulable state (3/10), tumor thrombus (2/10), or have had previous surgery or trauma to the portal vein (5/10). In addition nine other patients had had upper abdominal operations that could have associated factors in the development of portal vein thrombosis. Three of these patients underwent splenectomy for symptomatic hypersplenism, the others underwent surgical exploration and extensive dissection around the porta hepatis (*e.g.*, Kasai

portoenterostomy, cholecystectomy with common bile duct exploration).

Ten patients underwent portacaval shunt procedures at various times before OLT. The shunt was done in patients with recurrent variceal bleeding, preserved synthetic liver function, and no other major complications related to their liver disease.

Indications for OLT

All the patients were found to suffer from progressive end-stage liver disease, with complications that could not be controlled by more conservative management. There was a higher incidence of profound encephalopathy in the thrombosed or shunted group (70% and 80%, respectively) when compared with other OLT candidates (40%). In addition severe portal hypertension was found in all patients with PVT, but not in the presence of patent PCS. The existence of hepatocellular carcinoma limited to the liver was known in three patients before surgery, and in two patients tumor involved the bifurcation of the right and left portal branches.

Patency of the Portal Vein

A routine sonogram or Doppler sonogram (duplex) test is part of the preoperative assessment for all OLT candidates in our program. Pre-existing portal vein pathology such as previous portacaval shunt, or occlusion as demonstrated by sonogram, mandates a selective visceral angiogram. Flow through the splenic vein and the SMV were documented on the venous phase of selective splenic and SMA injections. Studies were not done in three patients known to suffer from recent trauma and portal vein ligation.

Duplex scan suggested an occlusion of the portal vein in 13 patients; these findings were confirmed by a selective celiac and SMA angiogram in 13 of 13 cases (Fig. 1). The duplex scan failed to detect an occluded vein in seven patients who were found to have this pathology during exploration and hepatectomy. Considering the large number of patients who underwent duplex scan, the specificity of this examination is >93% (open portal vein was confirmed in 510/550 patients). False-negative results were high, however, because the duplex failed to detect the presence of thrombosed vein, which was first seen during hepatectomy in 7 of 20 patients. Recent data suggest that the use of color duplex may have the same or higher specificity, but lower rate of false-negative results.[4]

Angiography was found to be a valuable and accurate test for patients who underwent various types of PCS, and those with sonographic evidence of an occluded portal vein. The portal vein was found to be open, and the PCS was patent in 10 of 10 patients (Fig. 1). In addition angiography confirmed the duplex findings of occluded portal vein in 10 of 10 patients. Moreover selective angiography helps to exclude all false-positive duplex findings, in other words, when the PV was thought to be occluded by sonographic test but was found open by the angiogram (data not shown).

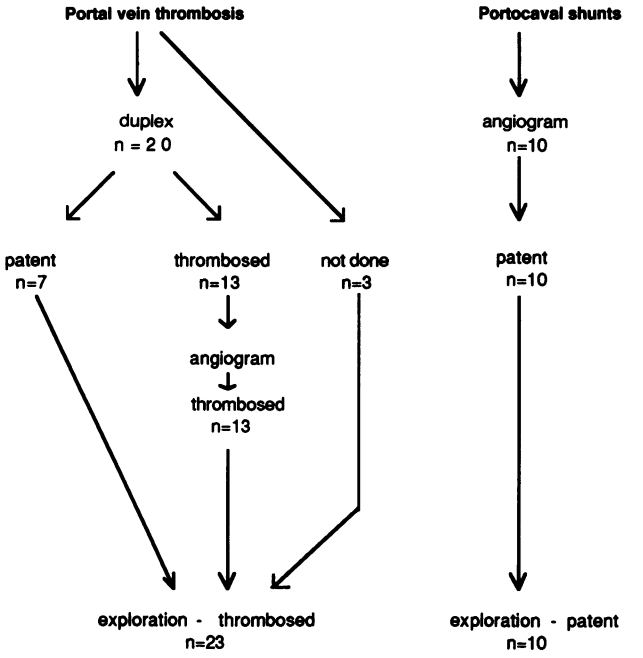

FIG. 1. Selective superior mesenteric artery angiography documented patent portal vein in the presence of portocaval shunt. Duplex scan was effective in the screening of most of the patients with portal vein thrombosis. However, occluded portal vein was found during exploration and hepatectomy in 7 of 23 cases.

Operative Management

The portal vein was successfully thrombectomized and preserved for anastomosis without the need for vein graft in 14 of 23 PVTs (Table 3). The coronary vein or large hepatoduodenal varix was used for portal inflow in two cases. Only seven patients needed the placement of donor iliac vein graft, six of seven were interposed to the SMV, and one of seven to the PV at the confluence of the splenic and the SMV.

The PCS was dismantled before the completion of the recipient hepatectomy in 10 of 10 patients (Table 3). In this group the entire length of the reconstructed vein was used as an inflow tract for portal blood perfusion of the liver graft.

Partial or full venous bypass was used in all the adult patients (Table 3). The full venous bypass was used in 17 of 33 patients, whereas partial bypass was used in 12 of 33 patients. Venous bypass was not used in four pediatric patients who were found to tolerate complete inferior vena cava (IVC) occlusion without significant changes in blood pressure or cardiac output.

TABLE 3. *Methods of Portal Vein Reconstruction*

Method	Portal Vein Thrombosis (n = 23)	Portocaval Shunt (n = 10)
Repair		
Dismantling with primary repair		10
Thrombectomy	14	
Vein graft		
to portal vein	1	
to superior mesenteric vein	6	
to coronary vein	2	
Venous bypass		
Full bypass		
Portal vein cannula	11	6
Inferior mesenteric vein cannula	3	
Partial bypass	7	4

TABLE 4. *Intraoperative Blood Loss and Urine Output*

	Portal Vein Thrombosis (n = 23)	Portocaval Shunt (n = 10)
Adult patients (n = 28)		
Estimated blood loss (blood volumes, mean + SD)	10.5 ± 10.2	12.4 ± 9.7
Urine output (ml/hr)	126	85
Pediatric patients (n = 5)		
Estimated blood loss (blood volumes, mean + SD)	9.1 ± 6.3	
Urine output (ml/kg/hr)	26	

Successful removal of the adherent clot and thrombus allowed the insertion of a portal vein cannula with the use of full venous–venous bypass in 11 of 14 patients with PVT. Flow rates through the thrombectomized vein exceeded 1000 cc/min. Patients who needed a portal vein graft were put on partial bypass (7/9) or full bypass using the IMV for portal blood outflow tract (3/9). Flow rates through the IMV were above 750 cc/min. It was elected to insert a portal cannula for full venous bypass in only 6 of 10 PCS cases. This was not done in the other four patients to minimize the trauma to an extremely delicate portal vein.

Fourteen patients required retransplantation at some point. The previously thrombectomized portal veins or portal vein grafts were successfully reused in all but two cases. Only one adult patient rethrombosed his portal vein, and portal blood flow to the new graft was established with the placement of interposition vein graft to SMV. The other patient, a child, clotted a vein graft to the SMV and died of septic complications before retransplantation. Venous–venous bypass was used in the retransplanted group with the same success rate as for the first transplant. Because all but one patient were found to have an open portal vein, the portal system was decompressed in the same fashion as the first OLT. The portal system of the patient with the thrombosed vein was decompressed through the IMV.

Intraoperative Complications

All patients maintained hemodynamic stability during their procedure. There was a significant increase, however, in the requirement of blood transfusion and the development of coagulopathy for transplanted patients with PCS and PVT (Table 4). A mean of 10.5 blood volumes were used for each adult patient. This is significantly higher than our average of blood loss of 3.2 blood volumes for all adult OLT cases. Correction of the coagulopathy required the continuous transfusion of fresh frozen plasma, as well as platelets and cryoprecipitate. These results were found to be similar in the pediatric patients. The mean blood transfusion was 9.1 blood volume, substantially higher then the average 3.3 blood volume in all pediatric OLT patients. Despite the excessive blood loss during procedure, most of the patients were kept with stable blood pressure. Urine output was >1 cc/kg/hr, and only four patients needed intraoperative or immediate postoperative dialysis (Table 4). There were no intraoperative deaths in this series.

Liver-related Postoperative Complications

Primary Nonfunction. The incidence of early (immediate) or delayed (3 to 6 days) primary nonfunction (PNF) of the liver graft was 35% for PVT (8/23) and 40% for PCS (4/10) (Table 5). These results are in contrast to the overall 8% incidence of PNF in our total experience with 676 liver transplants. Primary nonfunction was more common in patients with blood transfusion exceeding 60 units of packed red blood cells (PRBC). Reviewing the donor data and harvesting operative notes revealed three very unstable donors. These livers were used because of the urgent needs of the recipients.

Hepatic Artery Thrombosis. The incidence of hepatic artery thrombosis (HAT) was 9% (3/33) and occurred in two adult and one pediatric patient. These patients were out of the ICU with excellent graft function at the time

TABLE 5. *Postoperative Graft Complications*

	Portal Vein Thrombosis (n = 23)	Portocaval Shunt (n = 10)
Total retransplants (n = 15)		
Primary nonfunction	3	2
Delayed nonfunction (3–7 days)	4	2
Vascular complications (n = 4)		
Hepatic artery thrombosis	3	
Portal vein thrombosis	2	
Exploratory laparotomy (n = 14)		
Intra-abdominal bleeding	7	3
Bile leak	4	

of thrombosis. Hepatic artery thrombosis was attributed to hypotension after upper gastrointestinal bleeding in one pediatric patient; no specific cause was identified in the other two.

PVT. Rethrombosis of the portal vein occurred in 2 of 33 patients. In one case a thrombectomized vein was found to be occluded at the time of exploration for retransplantation. The clinical presentation was delayed PNF of the graft. The pathophysiology leading to rethrombosis can be explained, in retrospect, by poor venous blood flow through the thrombectomized vein during the first OLT (In this case the portal vein was not cannulated for venous bypass during the first OLT). The second patient had had an interposition vein graft to the SMV; the underlying pathology for occlusion of the graft was never found. The clinical presentation was progressive graft failure and severe sepsis.

Intra-abdominal Bleeding. Re-exploration for continuous intra-abdominal bleeding was required in 10 of 33 patients. The source of bleeding was not found in any of these patients, and was probably related to some degree of coagulopathy after multiple blood transfusions, as well as to poor synthetic function of the liver graft.

Bile Leak. Disrupted bile duct anastomosis was found in four patients, three of whom had HAT, and one case of portal vein rethrombosis. All three patients with HAT died of sepsis despite retransplantation.

Hospital Stay, Morbidity Rate, and Mortality Rate

Patients with pre-existing portal vein pathology were found to have prolonged ICU and hospital admission after OLT (Table 6). The survivors stayed an average of 11.5 days (PVT), and 16 days (PCS) in the ICU, with a total of 53.9 and 50 hospital days before discharge, respectively. This is in contrast to 6 ICU and 41.9 hospital days for all adult liver transplant patients.

Significant renal failure was seen in 4 of 22 survivors who needed prolonged postoperative dialysis. Kidney function returned to normal in all three cases, and compared favorably with other liver transplanted patients. Severe cytomegalovirus infection occurred in 2 of 22 survivors, which resolved after treatment with DHPG (ganciclovir).

The three-month mortality rates were 35% for the PVT and 30% for PCS patients. These are higher than the 12% 3-month mortality rate for all OLT at UCLA. The nonsurviving patients (11/33) stayed in the ICU for most of their hospital admissions. The cause of death was related to the development of uncontrolled fatal infections in 9 of 11 patients. The source of infection was related to peritonitis from bile leak (3/9), bowel perforation (1/9), pneumonia (3/9), and systemic infection from unknown source (2/9). In addition two patients died of rapidly progressive graft failure. These patients were comatose with evidence of irreversible brain damage, hemodynamically unstable and septic, and were not considered candidates for retransplantation.

Discussion

Patients with end-stage liver disease and documented thrombosed portal vein or prior portacaval shunt are considered high-risk patients, and are not uncommonly rejected as potential candidates, or referred to more experienced transplant centers. This approach to these patients is based on the complexity of portal vein reconstruction, and the clinical impression of increased morbidity and mortality rates in this group.[1,2] In this report we describe our results with OLT in the presence of pre-existing portal vein pathology.

The necessity for portal blood flow perfusion to the liver graft is well documented in early studies by Marchioro et al.[5] and Starzl et al.[6] Thus the presence of preexisting portal vein pathology and its potentially deleterious effects on the surgical procedure and graft survival has lead us to a policy of mandatory preoperative documentation of portal vein patency before OLT. The information was helpful to the medical and surgical team in the selection and preparation of the patient for a more complex procedure. The most common noninvasive reliable method is a duplex scan, which is accurate in identifying the vascular anatomy in 93% of the patients.[7,8] The small number of false-positive results were all eliminated by selective splanchnic angiography. The duplex scan test failed to detect PVT in a few patients found to have thrombosis during the hepatectomy. Other noninvasive tests to be considered are color duplex scan, magnetic resonance imaging, or nuclear imaging techniques.[9,10] We are studying the sensitivity of the color duplex scan in the detection of arterial and venous blood flow in the transplanted liver. Our preliminary results, as well as studies in peripheral vascular diseases, suggest that

TABLE 6. *Hospital and Intensive Care Unit Stay*

Duration of admission (mean + SD)	Portal Vein Thrombosis		Portocaval Shunt	
	Alive (n = 15)	Dead (n = 8)	Alive (n = 7)	Dead (n = 3)
Intensive care unit	11.5 (9.4)	41.4 (28)	16.0 (18.2)	24.4 (15.7)
Hospital	46.7 (25)	41.7 (27)	50.0 (21.2)	74.0 (45.6)

this test is more accurate in the detection of thrombi in the venous system.[4] Although selective celiac and mesenteric angiography was found to be an extremely reliable test, the use of this invasive and expensive procedure should be reserved for patients who are believed to have pre-existing portal vein pathology by duplex scan or a known surgical portosystemic shunt. In these patients it is important to determine the exact anatomy of the portal system by selective angiogram, because complete absence of a patent SMV or major mesenteric branches suitable to serve as a portal blood outflow tract are a contraindication to OLT.

The overall occurrence of PVT and PCS was found to be 6% (33/550 patients). To identify specific populations who are more prone to develop PVT, we have analyzed the incidence of this pathology in various types of end-stage liver diseases. A higher occurrence of PVT was found in patients suffering from chronic active hepatitis (CAH). Thrombosis of the portal vein was not seen in any patient with end-stage primary biliary cirrhosis or sclerosing cholangitis. This observation may relate to the impedance to blood flow through a small shrunken liver in patients with CAH. Thrombosis also was seen in patients with various hematologic disorders (*e.g.* polycythemia vera, Budd–Chiari, antithrombin III deficiency, etc.), as well as in patients who had a previous splenectomy or upper abdominal surgery involving dissection of the porta hepatis. Currently we document the patency of the portal vein by color duplex scan in these groups, or in equivocal cases we use angiography.

The operative reconstruction of the portal vein has been reported in several small series, as well as in a few case reports. It was shown that the occluded portal vein can be thrombectomized or bypassed.[11–13] The option of thrombectomy was exercised successfully in most of our patients, and was found to be safe, with a low re-thrombosis rate. Unlike previous reports, we were able to use the thrombectomized portal vein as a source of splanchnic outflow during venovenous bypass without the propagation of pulmonary embolus though the bypass system.[1] In the presence of surgical portacaval shunt, the shunt can be dismantled,[2] and the whole length of the vein can be reconstructed and used for successful portal blood revascularization of the graft. Once the shunt is taken down, it is safe to insert a portal cannula and place the patient on full venous–venous bypass.

When the native portal vein is not suitable for reconstruction, a donor iliac vein graft can be interposed between the recipient SMV and the donor portal vein.[14,15] The dissection in the colonic mesentery below the pancreas to locate the SMV is similar to that used for mesocaval shunts. Long-term follow-up in a few case reports found the grafts to stay patent. Using this procedure we encountered only one case of graft thrombosis. For the protection of the venous anastomosis, a portal cannula is not to be inserted through a vein graft to the SMV. In these cases, the portal system can be successfully decompressed through the IMV.[16]

As demonstrated in this study and other reports, the technical complexities of portal vein reconstruction can be solved. A different issue is whether these patients are acceptable candidates for OLT. Patients with PVT were rejected as OLT candidates in a few early series[17–19]; no specific indication is given for such exclusion. Because it is technically possible to reconstruct and restore portal blood supply to the liver graft, it is our opinion that this group of patients should be considered as OLT candidates. The increased risks should be understood and accepted, however, by the patients and the surgical team. Our results demonstrate that a pre-existing portal vein pathology is associated with increased perioperative complications and mortality rates. The intraoperative course is characterized by high blood loss, the need for multiple blood products, the development of coagulopathy, and increased rate of primary nonfunction of the new graft. This is not surprising, because it is expected that a difficult portal dissection in the presence of occluded portal vein and severe portal hypertension result in significant bleeding during hepatectomy. This is also true for patients with surgical portacaval shunt because the dissection of the shunt in scarred tissue planes often results in excessive bleeding. As a result, during the anhepatic phase, the patients develop severe acidosis and coagulopathy. The new liver graft is thus transplanted under less than optimal conditions in a compromised host, and the probabilities for failure are increased. We assume that this explains the relatively high rate of primary nonfunction of the graft, the prolonged postoperative coagulopathy, and the need for further explorations for evacuation of intra-abdominal hematomas. The possibility that sudden changes in the portal system or the prolonged period of diverted portal blood have some effect on the new graft can not be excluded.

The consequence of multiple explorations for intraperitoneal bleeding, bile leaks, or retransplantation in an immunocompromised host, and the prolonged ICU admission are the development of morbid infections and increased mortality rate.[20,21] The mortality rate in the presence of a thrombosed portal vein is 35%, and for portacaval shunt it is 30%. Collective review of small number of case reports show an early mortality rate of 15% to 50%.[11,14,15,22] These figures are significantly higher than the 86% 1-year survival for other OLT patients in our center. After a definite learning curve, however, our recent results during the past year are more encouraging. Anticipating a complicated course for patients with PVT or PCS allow better selection of the candidates. Patients with PVT or PCS are accepted as transplant candidates in the

absence of other significant organ system failure. Preoperative findings of tumor thrombus obstructing the portal vein is considered as contraindication for OLT. We now limit cold ischemic time to 8 hours for these difficult cases, in the hope of reducing the rate of PNF. Postoperatively the patency of the portal vein is followed by a routine duplex scan. Early exploration for thrombosed vein or artery, removal of thrombus, and re-do of the anastomosis may result in salvage of the graft. The current success rate in this group and the absence of any other alternative treatment justify the acceptance of patients with pre-existing portal vein pathology as OLT candidates.

Addendum

Since the submission of this article, the authors have operated on four more patients with portocaval shunts and three patients with portal vein thrombosis. The increased surgical experience with this pre-existing pathology of the portal vein, and the concerted effort to shorten ischemic time, resulted in no retransplants, less morbidity, and 100% patient survival. Our overall survival rate, to date, in patients with portal vein pathology is 72.5%.

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