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# Adult liver transplantation and abnormalities of splanchnic veins: experience in 53 patients

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**Abstract** The aim of this study was to analyze the influence of technical problems resulting from splanchnic venous anomalies on the outcome of orthotopic liver transplantation. From February 1984 until December 1995, 53 (16.3 %) of 326 adults underwent consecutive transplantations whilst having acquired anomalies of the splanchnic veins. These consisted of portal vein thrombosis ($n$ = 32, 9.8 %), thrombosis with inflammatory venous changes (phlebitis; $n$ = 6, 1.8 %) and alterations related to portal hypertension surgery ($n$ = 15, 4.6 %). Because of major changes in surgical technique, i.e., eversion instead of blind venous thrombectomy, *immediate* superior mesenteric vein approach in cases of extended thrombosis, and piggyback implantation with preservation instead of removal of the inferior vena cava, patients were divided into two groups: those who underwent transplantation during the period February 1984 to December 1990 (group 1) and those transplanted between January 1991 and December 1995 (group 2). Surgical procedures to overcome the anomalies consisted of venous thrombectomy ($n$ = 26), implantation of the donor portal vein at the splenomesenteric confluence ($n$ = 5) or onto a splenic ($n$ = 1) or ileal varix ($n$ = 1), interposition of a free iliac venous graft between recipient superior mesenteric vein and donor portal vein ($n$ = 9,) and interruption of surgical portosystemic shunt ($n$ = 13). All patients had a complete follow-up. The 1- and 5-year actuarial patient survival rates were similar in patients with ($n$ = 53) and without ($n$ = 273) splanchnic venous abnormalities (75.5 % vs 78.1 % and 64.3 % vs 66.9 %, respectively). Early ( < 3 months) post-transplant mortality was 24.5 % (13/53 patients). Mortality was highest in the portal vein thrombophlebitis group (5/6, 83.3 %), followed by the portal hypertension surgery group (5/15, 33.3 %) and the portal vein thrombosis group (3/32, 9.4 %). Technical modifications significantly reduced mortality in group 2 (10.3 %, 3/29 vs 41.7 %, 10/24 patients in group 1; $P < 0.05$) as well as the need for re-exploration for bleeding (13.8 %, 4/29 patients in group 2 vs 15/24, 62.5 % in group 1; $P < 0.01$). Mortality directly related to bleeding was also significantly lowered (1/29, 3.4 % in group 2 vs 9/24, 37.5 % in group 1; $P < 0.01$). We conclude that liver transplantation can be safely performed in the presence of splanchnic vein thrombosis and previous portal hypertension surgery.

**Key words** Liver transplantation, Splanchnic vein thrombosis · Thrombosis, splanchnic vein

**Fig. 1** Eversion venous thrombectomy technique. The carotid endarterectomy dissector separates the thrombus from the vascular wall. This maneuver is done while the surgeon occludes the splenomesenteric confluence with the index finger and the first assistant progressively everts the venous wall. One can see the extension of the thrombus into the splenic vein

## Introduction

Liver transplantation is an accepted therapy for end-stage liver diseases, even when significant local and systemic risk factors exist. Abnormalities of portal and/or splenic and superior mesenteric veins (SMV) were initially considered absolute contraindications [29]. Improvements in surgical technique, however, now permit liver transplantation in these situations. The aim of this study was to evaluate, in a single center experience, the impact of surgical technical modifications on the outcome of liver transplantation performed in patients presenting splanchnic vein abnormalities.

## Materials and methods

During the period February 1984 to December 1995, 53 of 326 adults (16.3 %) consecutively received a primary liver transplant in the presence of acquired abnormalities of the splanchnic venous system. There were 34 males and 19 females with a median age of 46 years (range 18–68.5 years). Indications for liver transplantation were: posthepatitic cirrhosis ($n = 36$, including 4 who also had a hepatocelllular carcinoma), alcoholic cirrhosis ($n = 5$), autoimmune cirrhosis ($n = 2$), primary biliary cirrhosis ($n = 2$), primary sclerosing cholangitis ($n = 2$), fulminant hepatic failure ($n = 2$), Wilson's disease ($n = 1$), Budd-Chiari syndrome ($n = 1$), hypervitaminosis A ($n = 1$), and cholangiocarcinoma ($n = 1$). None of the cancer patients had tumour thrombus in the portal vein. Five cirrhotic patients (10 %) were classified Child Pugh A, 16 (32 %) class B, and 29 (58 %) class C.

The splanchnic venous abnormalities consisted of portal vein thrombosis (PVT; 32/53, 60.4 %), inflammatory changes of the portal vein (PVT-itis; 6/53, 11.3 %), and changes due to previous portal hypertension surgery (PHS; 15/53, 28.3 %). Three patients had a thrombosed portocaval shunt and two had a PVT following splenorenal shunting. These two patients were considered part of the PVT group because of the main repercussion of thrombosis on allograft implantation. Twenty-seven of the 38 thromboses were total. The portal thrombus extended four times into the SMV, three times into the splenic vein, and four times into both the superior mesenteric and splenic veins.

Prior to 1991, assessment of liver transplant candidates included both Doppler ultrasonography and angiography. From 1991 onwards, angiography was only performed when ultrasound, performed by the same operator (T. P.), was inconclusive. Abdominal CT scan and/or magnetic resonance imaging were performed in order to accurately delineate the extent of the thrombosis and to detect possible inflammatory changes in the splanchnic veins.

In cases of PVT, the surgical technique used has to depend on the extent of thrombus and the quality of the vessel wall. If the thrombosis is limited to the portal trunk or is present in a vessel that still has an adequate wall, a hilar approach is preferred. This approach includes dissection down to the splenomesenteric confluence in order to facilitate thrombectomy. If the thrombus extends to the splenomesenteric confluence, if the portal vein is reduced to a fibrotic vessel remnant, or if inflammatory portal vein changes are present, an infracolic approach is performed.

Using the hilar approach, the portal vein is transected flush with the liver parenchyma once the liver is ready to be removed. There is no interruption of the collateral venous circulation, apart from very pronounced left gastric varices; their ligation is necessary to optimize portal perfusion.

Thrombectomy is best done under complete visual control. Using a carotid endarterectomy dissector, it is relatively easy to find the cleavage plane between thrombus and intima [8]. The thrombus is progressively freed by everting the venous wall, whilst the left index finger of the surgeon occludes the splenomesenteric confluence from behind (Fig. 1). In contrast to blind thrombectomy, in which thrombotic material is grasped and pulled out, this maneuver allows complete thrombectomy under direct vision without major blood loss, with minimal risk of tearing out the vessel wall, and with inspection of the intima of the thrombectomized vein.

**Table 1** Liver transplantation and splanchnic venous abnormalities: technical adjustments († patient deaths)

| Venous modification | *n* | Thrombectomy | Superior mesenteric vein implantation | Confluence dissection | Other | Early mortality (< 3 months) |
|---|---|---|---|---|---|---|
| Portal vein thrombosis | 32 | Blind 4<br>Eversion 18[a] (2 †[c]) | 7 | 2 (1 †) | Splenic varix implantation 1 | 3 |
| Portal vein thrombophlebitis | 6 | Blind 1 (1 †)<br>Eversion 1 | 1 (1 †) | 2 (2 †) | Ileal varix implantation 1 (1 †) | 5 |
| Portal hypertension surgery | 15 | | | | | |
| Portocaval | 13 | Blind 2[b] (1 †) | 1[b] (1 †[d]) | | Shunt division 13 | 4 |
| Splenorenal | 1 | | | 1 with banding of shunt (1 †) | (2 †) | 1 |
| Mesocaval | 1 | | | | | |
| Total | 53 | Blind 7 (2 †)<br>Eversion 19 (2 †) | 9 (2 †) | 5 (4 †) | 15 (3 †) | 13 |

[a] Two patients also had splenorenal shunt
[b] Patient also had portal vein thrombosis
[c] Patient died due to graft dysfunction related to steatosis and procurement trauma
[d] Patient had phlebitis

When inflammatory (peri)vascular changes are present, especially extending to the mesenteric and splenic veins, thrombectomy is too dangerous due to the high risk of tearing the vein wall. In such cases, the SMV is prepared using an infracolic approach to allow interposition of a free iliac vein homograft between donor portal vein and recipient SMV. This vein graft is placed in a prepancreatic and retrogastric position.

Surgical portocaval shunts are left intact until the end of the hepatectomy. End-to-side portocaval and splenorenal shunts indeed serve as partial venovenous bypass throughout the procedure. A distal splenorenal shunt can be left intact unless intraoperative electromagnetic measurement reveals inadequate, i.e., no or low flow, portal venous allograft perfusion. In such cases, the shunt should be closed either surgically or using (peritransplant) interventional radiology [8, 11].

When performing piggyback liver transplantation the liver is freed from the inferior vena cava without dividing the portal vein [3, 18, 29]. This technique enables the surgeon to prepare the modified venous revascularization site just before removal of the diseased liver.

Different technical adaptations were needed in order to deal with the different venous modifications (Table 1). PVT was managed by thrombectomy (18 and 4 patients, respectively, had eversion and blind thrombectomy), by dissection of the splenomesenteric junction ($n = 2$), by use of venous homograft to the SMV ($n = 6$), and by direct implantation of donor portal vein onto recipient SMV ($n = 1$) or splenic varix ($n = 1$). Portal vein thrombosis with phlebitis (PVT-itis) was handled by implantation of the donor portal vein at the splenomesenteric junction ($n = 2$), by implantation of a venous graft onto a phlebitic SMV ($n = 1$) or ileal varix ($n = 1$), and by blind ($n = 1$) and eversion ($n = 1$) thrombectomy.

Ten patients had a patent and three a thrombosed portocaval shunt. All portocaval shunts were divided, in one case by stapling and in the rest by suturing. Blind thrombectomy ($n = 2$) and venous grafting ($n = 1$) were necessary because of shunt occlusion. One partially thrombosed mesocaval shunt was left in place. Three patients had a distal splenorenal shunt, two of whom also had PVT. This shunt was left intact twice and banded once in order to improve portal perfusion. Venous grafting to the SMV was necessary once.

After 1991, portal vein flow was checked by intraoperative electromagnetic measurement. None of the 53 patients received intraoperative aprotinin or post-transplant anticoagulation therapy. Because of important modifications in surgical technique, namely, piggyback implantation, eversion thrombectomy, and systematic use of homograft interposition between donor portal vein and recipient SMV in cases of extended venous thrombosis, patients were divided for analysis into two groups: those transplanted between February 1984 and December 1990 (group 1, $n = 24$) and those transplanted between January 1991 and December 1994 (group 2, $n = 29$). In group 2, all patients had classical liver implantation, replacing the recipient's inferior vena cava and using venovenous bypass ($n = 20$). In group 2, all but two patients had a piggyback implantation with preservation of their vena cava; bypass was used only six times. Piggyback liver transplantation was not possible in the case of vena cava thrombosis due to Budd-Chiari syndrome and in the case of pronounced narrowing of the infrahepatic IVC following portocaval shunt dismantling. When used, venovenous bypass consisted of portofemoroaxillary (n = 8), inferior mesentericofemoroaxillary ($n = 10$), and femoroaxillary (n = 8) cannulation.

Intraoperative complications, intraoperative blood product use (including autotransfusion), morbidity, and mortality were analyzed for each group with reference to the different types of splanchnic venous abnormalities encountered. One unit of red blood cells corresponds to 400 ml. All patients had three monthly Doppler ultrasound examinations during the 1st year of follow-up; afterwards they had yearly examinations.

Early morbidity and mortality were defined, in accordance with criteria of the European Liver Transplant Registry, as events occuring within 3 months post-transplantation. All patients were followed-up until death or for a period of at least 6 months.

Actuarial survival curves were calculated following Kaplan-Meier. Comparison between proportions was performed with the $\chi^2$ test, the exact test being used when appropriate.

## Results

### Portal vein thrombosis

Eversion thrombectomy was successful in 18 patients; all were treated after 1991. The thrombus was completely removed in all but one patient. Only two patients (11 %) needed relaparotomy for bleeding related to the surgical technique. One patient died on day 28 of multiorgan failure; he had been regrafted because of graft dysfunction caused by both steatosis and severe procurement trauma. A second patient died on day 62 because of graft dysfunction caused by severe steatosis, and a third patient was successfully regrafted because of primary nonfunction, again related to steatosis. All three patients had uneventful thrombectomy; at regrafting, portal flow was normal. In contrast, all four blind thrombectomies were complicated by severe bleeding. Retransplantation was necessary once because of graft dysfunction; this patient eventually died on day 199 because of sepsis.

Both splenomesenteric confluence dissections with subsequent anastomoses were complicated by severe bleeding. One patient died; he also had portal vein rethrombosis. All seven implantations on the recipient SMV were successful. Implantation of donor portal vein on a splenic varix was followed by successful retransplantation, which was necessary because of graft dysfunction related to bleeding.

### Portal vein thrombosis and phlebitis

Five of six patients (83.3 %) with this condition died. Two confluence dissections, one iliac venous interposition, and one blind thrombectomy were all complicated by lethal perioperative bleeding. Implantation of donor portal vein onto an ileocolic varix resulted in bleeding and inadequate allograft portal perfusion; this patient eventually died of hemodynamic failure. Blind thrombectomy was followed by portal vein rethrombosis. Three of these patients, on preoperative CT scan, presented inflammatory changes of the perivenous fat extending into the infrapancreatic region (Fig. 2).

The only survivor in this group had a successful, albeit difficult, eversion thrombectomy.

### Portosystemic shunt

Banding of a distal splenorenal shunt caused lethal intraoperative bleeding and a pulmonary embolism. Eight of thirteen portocaval shunt divisions (61.5 %) were complicated by bleeding; two patients developed PVT. Four patients died and one had to be retransplanted because of early graft nonfunction. One patient needed re-

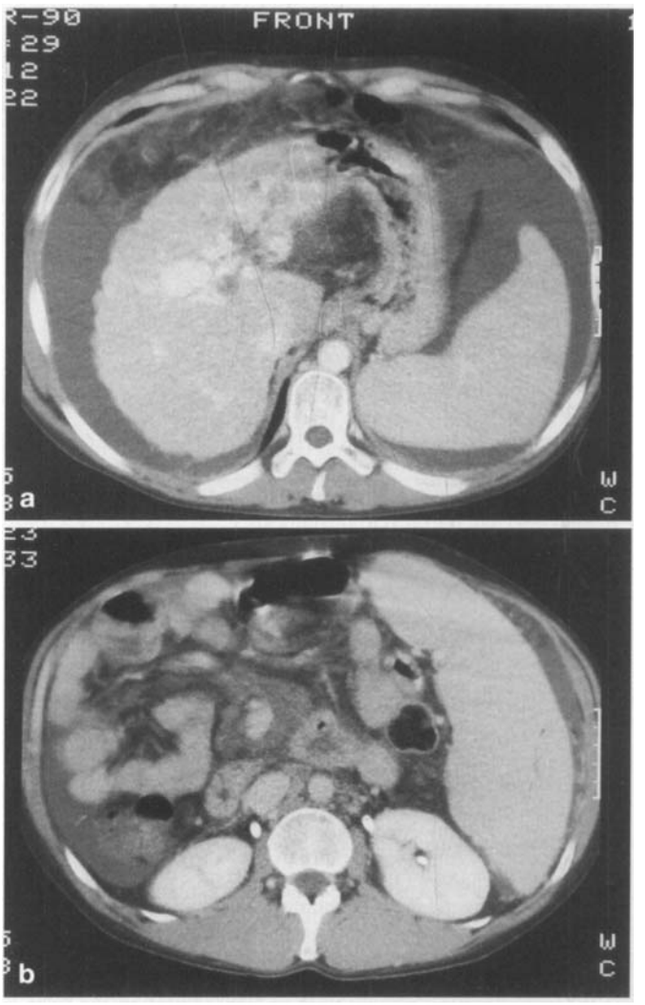


**Fig. 2a, b** Abdominal CT scan showing extensive perivenous fat inflammatory changes reaching the infrapancreatic level. At transplantation, dissection of the phlebitic splanchnic veins, including the superior mesenteric vein, was responsible for uncontrollable bleeding

grafting because of graft nonfunction following uneventful shunt division. Two of the three shunt desobstructions were complicated by lethal bleeding; one of these patients also had severe phlebitis at transplantation.

The 1- and 5-year patient survival rates of patients with and without splanchnic vein abnormalities were similar (75.5 % vs 78.1 % and 64.3 % vs 66.9 %, respectively). Thirteen patients (24.5 %) died during the early post-transplant period because of bleeding ($n = 8$), allograft failure precipitated by major bleeding ($n = 2$), pulmonary embolism and bleeding due to banding of splenorenal shunt ($n = 1$), and allograft dysfunction related to severe steatosis ($n = 2$). Early mortality was

**Table 2** Liver transplantation and splanchnic venous abnormalities: early mortality – relation to time period of grafting

| | 1984–1990 $n = 24/171$ (14 %) | 1991–1995 $n = 29/155$ (21.3 %) | Total $n = 53/326$ (16.3 %) | $P$ |
|---|---|---|---|---|
| Portal vein thrombosis | 1/ 9 (11.1 %) | 2[a]/23 (8.7 %) | 3/32 (9.4 %) | NS |
| Portal vein thrombophlebitis | 4/ 4 (100 %) | 1/2 | 5/6 (83.3 %) | NS |
| Portal hypertension surgery | 5/11 (45.5 %) | 0/4 | 5/15 (33.3 %) | NS |
| | 10/24 (41.7 %) | 3/29 (10.3 %) | 13/53 (24.5 %) | < 0.05 |

[a] Mortality due to allograft steatosis and procurement trauma

**Table 3** Liver transplantation and splanchnic venous abnormalities: intraoperative blood product use – relation to time period of grafting

| | 1984–1990 ($n = 24$) | 1991–1995 ($n = 29$) | $P$ |
|---|---|---|---|
| Use of red blood cells (ml),[a] including auto-transfusion | 15 118 ± 16 312 | 3703 ± 5634 | < 0.001 |
| Use of fresh frozen plasma (ml)[a] | 11 109 ± 20 846 | 2996 ± 4010 | < 0.05 |
| Reoperation for bleeding | 15 (62.5 %) | 4 (13.8 %) | < 0.01 |
| Mortality related to bleeding | 9 (37.5 %) | 1 (3.4 %) | < 0.01 |
| Supplementary graft loss related to bleeding | 3 (12.5 %) | 0 | NS |

[a] Mean values

highest in the phlebitic group (5/6 patients, 83.3 %), followed by the PHS group (5/15, 33.3 %) and the PVT group (3/32, 9.4 %; Table 2). The mortality of liver transplantation in the presence of splanchnic venous modifications was significantly lower in group 2 (3/29 patients, 10.3 % vs 10/24 patients, 41.7 % in group 1; $P < 0.05$)].

Thirty of the 53 patients (56.6 %) had severe early postoperative complications; these were always related to perioperative bleeding. Morbidity consisted of abdominal bleeding necessitating relaparotomy ($n = 19$), gastrointestinal bleeding ($n = 8$), portal vein thrombosis ($n = 4$), sepsis ($n = 3$), biliary tract complications ($n = 3$), early graft nonfunction due to severe perioperative bleeding ($n = 3$), and colonic perforation ($n = 1$). Portal vein thrombosis was, in all four cases, diagnosed by palpation. Morbidity was highest in the PVT-itis group (6/6, 100 %), followed by the PHS group (12/15, 80 %) and the PVT group (12/32, 40.6 %).

One (2.5 %) of the 40 long-term ( > 3 months) survivors developed a PVT 4 years after transplantation using venous graft between portal vein and SMV.

Early mortality and morbidity correlated well with the intraoperative blood product requirements during the different study periods (Table 3). Nine of 24 patients (37.5 %) died from perioperative bleeding during the first period, whereas only one patient (3.4 %) died of this complication during the second period ($P < 0.01$). This patient, moreover, had poor graft function following insufficient portal vein perfusion. The number of surgical reinterventions for intra-abdominal bleeding was also significantly higher in group 1 than in group 2 (15/24 patients, 62.5 % vs 4/29, 13.8 %, $P < 0.01$).

Fourteen (58.3 %) of 24 group 1 patients needed more than 10 l of red blood cells during surgery, whereas only 4 (13.8 %) of 29 group 2 patients had such a transfusion requirement ($P < 0.01$). These latter patients had Budd-Chiari syndrome with vena cava and PVT, allograft procurement trauma and dysfunction due to steatosis, and severe coagulation disturbances due to a dysfunctioning split-liver graft.

## Discussion

Abnormalities of the portal vein and/or its tributaries were initially considered a contraindication to liver transplantation [29]. As the incidence of PVT reaches 15 % in patients with chronic liver disease [23, 26] and ranges from 6 % to 21 % in surgically treated cirrhotics [4, 26], several technical modifications had to be developed in order to allow allograft implantation [28–30, 33]. Despite the introduction of technical refinements, the operative mortality rates reported since 1990 remain high (range 9.1 %–42 %) in the presence of splanchnic thrombosis [7, 10, 16, 22, 29] (Table 4) and previous portosystemic shunting (range 7 %–33 %) [1, 5, 17, 21, 26, 32] (Table 5). The prerequisites for successful liver transplantation in such cases are precise preoperative evaluation of the splanchnic veins and/or portosystemic shunt anatomy, as well as a standardized surgical approach.

The most used screening technique for imaging the portal vein is Doppler ultrasound [13, 33]. In cases of previous portal hypertension surgery and when there is pronounced portosystemic collateral formation, selective angiography with or without magnetic resonance imaging or CT angiography is required [6, 12, 20]. The latter examinations have the advantage that anatomic interrelationships are easier to judge, that less contrast medium is required, that the portal vein can be visual-

**Table 4** Liver transplantation and splanchnic vein thrombosis: recent literature survey

| Author | Center | Year | Number of patients | Perioperative mortality | One-year survival |
|---|---|---|---|---|---|
| Shaked et al. [26] | (Los Angeles) | 1991 | 23/550 (4.2 %) | 35 % | 65 % |
| Stieber et al. [29] | (Pittsburgh) | 1991 | 34/1585 (2.1 %) | 33 % | 67 % |
| Langnas et al. [16] | (Omaha) | 1992 | 16/495 (3.2 %) | 19 % | 81 % |
| Cherqui et al. [7] | (Paris) | 1993 | 11/69 (15.9 %) | 9.1 % | 73 % |
| Moreno Gonzalez et al. [22] | (Madrid) | 1993 | 14/195 (7.2 %) | 35.7 % | 57.2 % |
| Davidson et al. [10] | (London) | 1994 | 14/132 (10.6 %) | 42 % | 58 % |
| UCL | (Brussels) | 1996 | 38/326 (11.7 %) | 26.3 % | 73.7 % |

**Table 5** Liver transplantation and portosystemic shunting: recent literature survey

| Author | Center | Year | Number of patients | Perioperative mortality | One-year survival |
|---|---|---|---|---|---|
| Mazzaferro et al. [21] | (Pittsburgh) | 1990 | 58/1445 (4 %) | 33 % | 67 % |
| Aboujaoude et al. [1] | (London–Ontario) | 1991 | 27/174 (15 %) | 7 % | 76 % |
| Boillot et al. [5] | (Paris) | 1991 | 18/220 (8.1 %) | 33 % | 67 % |
| Shaked et al. [26] | (Los Angeles) | 1991 | 10/550 (1.8 %) | 30 % | 70 % |
| Turrion et al. [30] | (Dallas) | 1991 | 21/300 (7 %) | Not available | 85 % |
| UCL | (Brussels) | 1996 | 15/326 (4.6 %) | 33.3 % | 66.7 % |

ized in the presence of reversed flow, and that phlebitis or perivascular inflammatory changes may be accurately recognized [6, 13, 34]. The diagnosis of perivenous inflammation, however, remains difficult and usually relies on indirect signs, such as fatty perivascular infiltration. Angiography and magnetic resonance imaging should both be done in order to obtain the most information about the modified vessel status. If extensive perivenous or venous inflammatory changes reaching the infrapancreatic region are diagnosed, we feel transplantation should not be performed because of the extremely high risks of uncontrollable bleeding. If diffuse thrombosis of the splanchnic veins is diagnosed, combined liver-intestinal transplantation remains the only therapeutic option [2].

Some abnormalities can be avoided by appropriate choice and planning of previous hepatobiliary or portal hypertension surgery [29]. If portal decompression is needed, mesocaval H-graft and distal splenorenal shunts are preferred, as there is no liver hilum dissection [5, 17, 21, 27, 29]. A distal splenorenal shunt usually does not have to be ligated [11, 16, 22, 27]. Laterolateral portocaval shunts may nowadays be replaced by the attractive alternative of transjugular portosystemic shunting, although this technique may also be responsible for other technical difficulties, such as stent dislocation into the suprahepatic vena cava, inflammatory changes of the venous vessels, or even development of portal vein aneurysm [19].

The increased complexity of the surgical procedure in cases of splanchnic vein abnormalities is responsible for enhanced morbidity and mortality due to compromised allograft venous inflow (once in these series), portal vein stenosis or rethrombosis (four times in these series), and perioperative bleeding with the associated risks of infection and graft nonfunction (three times in these series) [5, 10, 16, 22, 26, 29, 30].

It is important that the donor and recipient operations are timed to keep cold and warm ischemia times to a minimum [21, 28]. The key is to decide on the method of portal vein reconstruction before starting the implantation of the graft. The hilar or supracolic approach to the recipient portal vein or splenomesenteric junction is preferred when thrombosis or phlebitis does not extend to the splenomesenteric confluence and when the venous wall is of good quality. Obstruction of the venous inflow tract must be relieved, preferably by eversion thrombectomy [8]. If the thrombosis extents to the SMV or if the portal vein has been reduced to a fibrotic vessel remnant, an infracolic approach must be taken using an iliac venous homograft to join donor portal vein and inframesocolic SMV [28, 31]. Extra portal vein length can be obtained during organ procurement by en bloc removal of liver and pancreas, allowing retention of the portomesenteric venous axis [32]. In some exceptional cases, anastomosis between donor PV and recipient splanchnic system is impossible. Intraoperative venography through ileocolic or inferior mesenteric veins may be helpful in order to properly assess the venous anatomy [16, 30]. In such cases, successful restoration of portal allograft perfusion has been obtained by anastomosing donor portal vein to left gastric vein [9, 29], hepatoduodenal [27], bile duct [7, 14), gastroepiploic [10], and ileocolonic varices [16] and even by (partial) arterialization of the portal vein [25, 30].

Portal allograft perfusion should be assessed by electromagnetic measurements. If inadequate, venous collaterals or a surgical distal splenorenal shunt should be interrupted [11, 16].

The safety of the transplant procedure can be further improved in these patients by combining the abovementioned approaches with the piggyback implantation technique [18, 29]. This technique allows one to dissect the recipient liver from its own inferior vena cava without interrupting the portal vein or dividing any portosystemic surgical shunt. What is potentially the most dangerous part of the recipient procedure, i.e., taking care of the thrombosed portal vein or interruption of portosystemic shunt, can be done immediately before completion of hepatectomy, a stage at which control of bleeding is usually easier to perform.

In conclusion this review confirms that patients with abnormalities of the splanchnic venous system require detailed preoperative investigation to exactly delineate the extent and the nature of the anomalies and that a standardized surgical approach is especially valuable with regard to perioperative blood product use. By employing such a strategy, mortality and morbidity can be significantly reduced in these patients.

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