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Vascular complications after orthotopic liver transplantation

M.S. Novruzbekov, O.D. Olisov

N.V. Sklifosovsky Research Institute for Emergency Medicine, Moscow, Russia

Correspondence to: Murad S. Novruzbekov, Cand. Med. Sci., Head of the Scientific Department for Liver Transplantation at N.V. Sklifosovsky Research Institute for Emergency Medicine, Moscow, Russia, e-mail: N.M.S@bk.ru

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Vascular complications (VCs) comprise one of the most serious problems in liver transplantation. This is the next reason of graft loss after primary graft non-function, with the majority of complications being arterial by etiology. VCs significantly decrease the graft and patient survival; contribute to the incidence of retransplantation. This review focuses on the epidemiology, etiology, diagnostics, and treatment of VCs.

Keywords: liver transplantation; vascular complications, arterial thrombosis, portal vein thrombosis, hepatic vein thrombosis.

An impaired blood supply remains one of the most serious complications following orthotopic liver transplantation (OLT) [1, 2]. Severe and sometimes irreversible ischemic damage of the graft induces massive necrosis of hepatocytes and biliary epithelium and serves as a trigger in the development of multiple organ failure and uncontrollable sepsis. In such a situation, the only hope for saving a patient's life is an urgent retransplantation, its probability being, in turn, significantly restricted by an acute shortage of donor organs, and a severe patient's condition [1, 3].

Therefore, the prevention of vascular complications, their timely diagnosis, and optimal treatment tactics aimed at eliminating the injury caused by vascular problems, acquire a great importance for improving the early and long-term OLT outcomes.

Hepatic artery thrombosis

Hepatic artery thrombosis (HAT) can be referred to the most widespread and most threatening of post-OLT vascular complications, namely, the arterial complications (with HAT occupying a leading place) which constitute 64-82% of all post-transplantation vascular complications [4].

In the period of an OLT technique implementation, the rate of arterial thrombosis was about 12% among adult recipients, and about 42% among children. The evolution of the surgical techniques brought about a significant improvement in the outcomes of arterial reconstructions in OLT; nowadays, the median of HAT incidence makes 4.4%. In pediatric practice, HAT complicates about 8.3% of liver transplants [1, 2]. The problem of arterial complications after OLT has been one of the most discussed in hepatobiliary surgical community; recent discussions have often revealed the ambiguity, and sometimes conflicting opinions and views on this issue [1].

Technical difficulties and errors in surgical technique have been considered the main cause of HAT for a number of years. Despite the fact that the technical factors remain dominating in the pathogenesis of hepatic artery thrombosis, the problem of arterial complications after OLT has been qualified as a multifactor problem that can hardly be explained solely by the technical aspects of forming an arterial anatomizes [1, 5-7].

Technical (surgical) causes of hepatic artery thrombosis

The HAT causes that should be referred to technical ones may include the tactical and technical errors made during the donor phase of surgery, namely an excessive hepatic artery dissection, the damage to its wall, an intimal tear, perivascular hematomas that contribute to HAT formation [2].

A variation of hepatic arterial anatomy in a donor may be a risk factor of the arterial thrombosis development. As early as at the stage of donor organ retrieval, the errors in identifying the graft arterial blood supply could result in unintentional damage to the hepatic artery and its branches [8]. Furthermore, the presence of several independent sources of arterial blood supply usually requires complex arterial reconstructions at "back-table" step, which is also one of the risk factors for arterial thrombosis [1, 2, 5, 9]. Thus, T. Soliman et al. (2003), P. Duffy et al. (2009), P. Warner et al. (2011) and Y. Yang et al. (2014) in their studies showed that the existing variation anatomy and making complex arterial reconstructions might be associated with a significantly higher percentage of thrombosis cases as compared to implantations performed in conditions of a standard arterial anatomy [5, 10-12]. In contrast to their opinion, M. Silva et al. (2006), E. Melada (2005), B. Seket (2009), and W. Andraus (2013) do not refer the variation anatomy and complex arterial reconstructions to HAT risk factors [6, 13-15]. So we have to admit that no consensus has been achieved yet on the role of non-standard arterial anatomy in the HAT genesis [16].

The condition of the arterial wall should also be referred to the technical factors. The shortage of donor organs becoming more acute with every year passing dictates the need to increase the donor pool by using organs from expanded criteria donors. Quite often, this category is

represented by the donors over 60 years old having disseminated atherosclerosis of visceral vessels, including those in the vascular pool of the celiac trunk and the superior mesenteric artery. The risk of developing arterial complications in this population is naturally higher due to the threat of intimal detachment or embolism caused by atherosclerotic plaque. If all the above mentioned factors were added to a non-standard arterial anatomy and the need for the arterial trunk prosthetics with the donor conduits that might also have been affected by atherosclerosis, one could assume that the probability of the hepatic artery thrombosis would increase many-fold [1, 17, 18].

Not only the quality of anastomosed vessels but also their diameter may affect the arterial reconstruction outcome. The artery diameter less than 3 mm represents a real risk of HAT [19]. The use of magnifying optics and microsurgical techniques does not warrant the HAT avoidance in case of anastomosing a small-diameter artery or the artery substantially different in size. This implication has been particularly relevant in liver fragment transplantation (in living-related and split-liver transplantation) where the anastomosed arteries are significantly smaller by diameter than in whole organ transplants [1, 2, 17, 20-22]. We should note that the negative impact of liver fragment transplantation on HAT incidence has not been unanimously admitted by all authors [5, 6].

Arterial competence greatly depends on the choice of arterial reconstruction technique. Arterial end-to-end anastomoses are susceptible to thrombosis more frequently than the arterial anastomosis performed using the aortic Carrel patch [1, 23].

The technical difficulties while forming arterial anastomosis may be predetermined by the quality of the donor hepatic artery segment, and also

by the condition of the recipient's own arteries. A persistent arterial spasm, an inadequate diameter, atherosclerotic lesion of the native hepatic artery and inadequate arterial blood flow, a history of endovascular interventions (hepatic artery embolization) or preexisting thrombosis may require using alternative arterial reconstruction techniques. In such situations, the most common solution is to perform the anastomosis directly between the recipient's aorta and the donor's celiac trunk using a vascular allograft constructed of donor's iliac vessels, as a rule. [17, 24, 25]. However, many authors believe that such anastomoses are associated with a statistically significant higher HAT rate compared to the traditional arterial reconstruction technique [1, 2, 6, 10, 17]. Direct aortic anastomoses have such inherent undesirable phases as a broad proximal or distal aortic dissection (depending on the level of the intended anastomosis: either epigastric, or infrarenal one), and clamping of the aorta (which is undesirable in the situation of unstable hemodynamics). Previous surgical interventions, adhesions, and obesity complicate the exposure of the corresponding aortic wall part. According to W. Vanderlan et al. (2008), in case of a poor condition of the recipient's native hepatic artery, the anastomosis with the splenic artery should be preferred; S. Dookmak (2015) recommends using the recipient's celiac trunk in such cases [18, 25].

Concluding the section on HAT surgical causes, we can not ignore one of the most important aspects of this problem, namely, the value of a personal surgical experience. It is obviously after all, the more frequently the surgeons perform arterial reconstructions, the better will be their outcomes. The centers where liver transplantation has been routinely performed for many years, demonstrate a significant reduction in the HAT number with gaining the experience. In the clinics with fewer that 30 operations

performed annually, the HAT rates are significantly higher than in the clinics with a more extensive transplant activity [1, 2, 12, 26].

Other (non-surgical) causes of hepatic artery thrombosis

The OLT performed disregarding the anthropometric parameters of both the donor, and the recipient entails the risk of the graft perfusion impairments followed by HAT occurrence. The greatest risk of HAT is observed in large-size liver transplantation where the surgical wound closure is accompanied by the graft mechanical compression, impaired perfusion, and HAT development. The recipient-to-donor weight ratio exceeding 1.15-1.25 is believed to be a HAT risk factor [5, 7, 27]. The recipient selection considering the donor's anthropometric measurements (weight, height; chest and waist circumference) would reduce the risk of HAT.

A majority of authors have regarded a prolonged thermal ischemia of the graft to be an immediate risk of its arterial insufficiency [1]. According to P. Warner et al. (2011), every extra 10 minutes between the venous and arterial reperfusion increased the risk of HAT by 27% [10]. Also, the data obtained by Y. Yang (2014) showed that arterial reconstruction phase lasting for more than 80 minutes predisposed to HAT [5].

A massive blood loss and the transfusion of blood components have been considered as preconditions for the post-OLT arterial thrombosis development: the HAT rates significantly increase in the cases of transfusing 7 or more doses of packed RBCs and over 6 doses of fresh frozen plasma [1, 28]

The recipient hypercoagulation status, smoking, obesity, history of diabetes, alpha-1 antitrypsin deficiency predispose to thrombosis and can provoke HAT in the postoperative period [1, 2, 28, 29].

Non-surgical causes of HAT include also the following: the immunologic conflict arising in transplantation of AV0-incompatible liver, cytomegalovirus infection in organ transplantation from a seropositive donor to a seronegative recipient, more than one episode of acute cellular rejection occurring for 7 days, and significant age differences in the donor/recipient pair (a young recipient and an aged donor) [7, 29].

Hepatic artery thrombosis classification

HAT has been classified depending on the time of its occurrence. In general, early and late arterial thrombosis shall be distinguished [1]. The need for such differentiation is dictated by the differences in the clinical presentation, the course, prognosis, and the choice of treatment tactics. The majority of authors recognize that early HATs are significantly more aggressive and have a worse prognosis compared to late HAT [5, 19, 20]. This statement is logically based on the fact that the transplanted liver in the early postoperative period has no arterial blood supply sources other than the hepatic artery main trunk, as arterial collaterals have not been formed yet [1]. In the situation with the late HAT, the collateral blood flow is significantly more likely to take place, mainly through diaphragmatic arterial branches. Meanwhile, we must recognize that there has been no consensus reached as to what time interval could be a measure of early or late HAT [2, 4]. M. Mourad et al. (2014) suggested that HAT diagnosed within the first 3 weeks after transplantation should be classified as an early HAT [30]. J. Bekker et al. (2009) defined early HAT as the thrombosis occurred within 2 months after transplantation [1]. O.Abbasoglu et al. (1998) and J. Rennert et al. (2012) classified HATs as early and late ones, the latter developing at expiry of 3 months after transplantation [31, 32]. Most authors consider the

threshold to distinguish between the early and late HAT to be at 1 month after the OLT [2, 6, 10, 12, 19, 28].

HAT diagnosis: laboratory, clinical, and instrumental tools

The clinical-and-laboratory presentation of the graft arterial insufficiency ranges from a moderate dysfunction to a fulminant hepatic failure [4]. In the majority of cases, the HAT the laboratory pattern manifests itself with an abrupt rise in the levels of cytolytic enzymes, bilirubin, and with a persistent increase in international normalized ratio. Subsequently, if untreated, or treated ineffectively, it displays deteriorating signs of progressive liver failure, lactic acidosis, and the laboratory evidence of sepsis. Abnormal laboratory findings and clinical pattern correlate to the extent of graft necrotic lesion [2, 19, 28, 33, 34].

The clinical presentation of early HAT is logically consistent with laboratory abnormalities: the severity of patient's condition is determined by the liver failure and sepsis refractory to therapy, which have been caused by massive necrosis of hepatocytes and bile ducts. The cessation or significant compromise of the blood flow in the graft often hide themselves under the guise of biliary complications, so any biliary complication should be carefully examined for the presence of concomitant arterial problems. HAT is typically characterized by multiple non-anastomotic strictures of bile ducts, recurrent pyogenic cholangitis, and the biliary sepsis as a consequence. As already noted, the most dramatic scenario develops in early HAT, while the clinical and instrumental pattern of late HAT may be less obvious, and at times absent [2, 6, 19, 26, 33].

Instrumental techniques used to diagnose arterial complications after OLT include both direct and indirect visualization of arterial blood flow.

Doppler ultrasonography (DUS) has been the most commonly used technique to assess the graft hemodynamics. The majority of authors consider DUS to be the first-line diagnostic tool for post-transplant vascular complications [2, 4-6, 28]. The main parameters investigated with ultrasonography include: the detectable flow signal from the hepatic artery, the peak systolic velocity, and the hepatic artery resistance index. The values being considered normal in transplant patients make about 103 cm/s for arterial blood flow velocity and range between 0.55-0.85 for the resistance index [35, 36]. However, one should remember that the parameters can substantially differ from normal in the first few days after the OLT that can be attributed to graft edema, impaired central hemodynamics, and a peripheral arterial spasm. In order to avoid a fatal error, it is mandatory to monitor arterial blood flow at Doppler ultrasound both at the level of the hepatic artery, and also in intrahepatic branches, as far as the signal from the gastroduodenal artery in a number of cases can be mistaken for the hepatic artery blood flow [4]. According to R. Sanyal (2014), DUS enables HAT suspicion in 92% of cases. DUS has undeniable advantages, being a noninvasive investigation, available by cost, and can be repeated for 24 hours [35].

A routine Doppler ultrasonography is a factor that considerably determines the success of a timely diagnosis, early thrombectomy, and reconstructive surgery on the hepatic artery [4]. The highest percentage of HAT diagnosed at early stages (median within 6.9 days) was observed in the centers where ultrasonography was routinely used in the first postoperative week [1]. The need for routine DUS seems undoubted, but not all transplantation centers use this method in the daily assessment of the graft;

and some clinics perform ultrasonography only when suspicious of vascular complications [1, 6, 10].

Data obtained at ultrasonography, as a rule, require an immediate confirmation by direct or indirect angiography. Despite the direct angiography remains the "gold standard" for the post-transplant HAT diagnosis, the multislice spiral computed tomography (MSCT) has become a more widely used; MSCT findings are comparable to angiography data [1, 2, 22, 36-38].

Treatment of hepatic artery thrombosis

Cessation of arterial blood flow in the liver graft can be characterized as a critical condition that requires immediate measures to be taken to cope with it. In general, there are only two possible ways out of the crisis situation. The first one is an emergency revascularization of the graft consisting of thrombectomy and(or) the arterial anastomosis reconstruction. The success of using this treatment technique is greatly depends on a timely made HAT diagnosis, the distal (intrahepatic) arterial bed condition, and histology findings in the graft. Ultimately, it is the time factor that determines the graft revascularization efficacy [6]. Thus, according to J.Bekker et al. (2009), the highest success rate of repeated interventions on the hepatic artery was observed in the clinics routinely using Doppler ultrasonography and therefore timely detecting HAT [1]. However, even with such a concept of active postoperative screening, a timely diagnosed HAT, and a seemingly timely surgical treatment, the revascularization success rate has not exceeded 66%.

The graft revascularization may be supplemented with endovascular techniques, specifically, the intra-arterial thrombolytic therapy, especially in

the cases where the patency of intrahepatic arterial bed is extremely doubtful. [39]. However, one should mention that endovascular interventions are usually low-effective in thromboses caused by technical factors. Moreover, the thrombolytic therapy is accompanied by hemorrhagic complications in 20% of cases, and may result in death in some cases [39]. According to Y. Yang et al. (2008), endovascular interventions were more demanded for hepatic artery stenoses, while retransplantation remained the best technique for HAT [40].

Retransplantation is the other option to solve the HAT problem. Retransplantation is indicated in 53% of patients with HAT and in 30% of patients undergoing thrombectomy and reconstructive intervention on the hepatic artery. Some authors regard retransplantation as the most efficient treatment indicated for a great number of HAT patients [19, 26, 41, 42]. According to R. Bussutill and G. Klintman (2014), in the USA, a thrombosed arterial anastomosis early after OLTs makes an absolute indication for listing the patient as urgent for liver retransplantation [19]. The situation is dramatic because retransplantation is necessary to be performed in the shortest possible time that is very difficult given the current acute shortage of donor organs. Besides the availability of donor graft, the key prerequisites for retransplantation include its individual patient tolerability, and control of sepsis which risk is extremely high and persisting [6, 41]. All of the above explains an extremely unfavorable prognosis in HAT: a graft loss being reported in 53% of cases, an average mortality making 33%, reaching 80% in some reports [1]. Prognosis in late thrombosis has been somewhat more optimistic as the developed collateral blood flow can partially compensate for the existing arterial insufficiency. In this situation, a clinician obtains a certain time reserve, which allows stabilizing

a patient's condition and placing the patient on the list for retransplantation [1, 2].

The methodology of a portal vein arterialization used by F. Melandro (2013) and P. Bhangui (2014) is worthwhile to note [3, 43]. The authors emphasized that the technique should not be used as an alternative to retransplantation, but could be a palliative measure giving the chance to gain valuable time and prepare the patient for retransplantation.

Portal vein thrombosis (PVT) is a common complication of liver cirrhosis [44-46]. Until recently, PVT has been considered as an absolute contraindication to OLT [4, 44, 47]. In 1985, a working group from the University of Pittsburgh made a series of successful transplants in cirrhotic patients with PVT [48]. Those successful interventions initiated the evolution of views on the problem of PVT in candidates for OLT. Now PVT is not considered an absolute contraindication to transplantation any more. However, the preexisting PVT is regarded by most experts as a risk factor of severe complications in the intra- and postoperative periods [49-51].

PVT rates in the population of patients suffering from liver cirrhosis make about 10-15%, reaching 40% in some studies [45-47, 52]. Most PVT cases are diagnosed at patient's examinations and evaluations while on the waiting list. However, 12%-60% of PVT cases are identified intraoperatively [45, 49]. The post-OLT PVT rate is about 1-2% in the patients without pre-existing PVT. In the patients undergoing transplantation with a preexisting confirmed PVT, the rethrombosis complicates about 9% of OLTs, ranging from 5 to 30% [4, 19, 46].

Risk factors for PVT include: the presence of pre-existing thrombosis, portocaval anastomosis, previous splenectomy, a small diameter (less than 5 mm), sclerosis or hypoplasia of the portal vein, a donor/recipient difference

between the anastomosed parts of the portal vein segments, the liver fragment transplantation (living-related or split-liver transplantation) and the use of venous conduits in PVT, a reduced portal blood flow velocity lower 15 cm/s, an impaired venous outflow in caval-caval anastomosis, hepatocellular carcinoma, Budd-Chiari syndrome, Class C Child-Pugh liver cirrhosis and a high MELD score, and a cirrhosis etiology (alcoholic, autoimmune, cryptogenic) [12, 20, 28, 46, 47, 49, 50, 53].

The PVT clinical presentation, particularly in acute portal vein occlusion, demonstrates a graft dysfunction, its severity being determined by the extent of the ischemic lesion. The PVT pathognomonic signs appear as a persistent or progressive portal hypertension, encephalopathy, refractory ascites, esophageal and gastric variceal haemorrhage. Laboratory abnormalities in acute PVT are manifested in enzymemia with its level varying from mild to critical [4, 19, 28, 33, 34, 46].

The morphologic pattern typical for partial PVT of the transplanted liver graft includes hepatocyte degeneration extending from periportal to the central zone. The morphological pattern in complete PVT in the early postoperative period is consistent with massive coagulative necrosis [19, 33].

The most widely used tools to diagnose PVT are Doppler ultrasonography (DUS) and intravenous bolus contrast-enhanced MSCT. Both methods are informative, non-invasive, and mutually complementing one another [28, 36, 37, 45, 49, 54]. DUS findings in PVT are characterized by the absence of Doppler signal from the main portal vein and its intrahepatic branches, by an enhanced echo signal from intrahepatic arteries, and by the appearance of clear-cut venous collaterals in periportal space. One should emphasize an increasing role of ultrasonography in occlusive

PVT; however, a partial PVT can hardly be detected by ultrasonography. DUS sensitivity makes 48% for PVT Grade I, 82% for PVT Grade II, 100% for PVT Grade III and IV, each [49].

The problem of pre-existing thrombosis dictates the necessity of thorough planning the surgery, choosing an appropriate technique of portal blood flow recovery, minimizing intra and post-operative risks. It is necessary to properly assess the venous obstruction degree, its proximal extent and, based on the findings, to plan further actions [49]. Among existing PVT classifications, the one proposed by M. Yerdel et al. has been the most commonly used and practically relevant (particularly for liver transplantation) [45, 46, 52, 55, 56].

Yerdel Classification for portal vein thrombosis (2000) [57]:

• Grade I: occlusion of less than 50% of main portal vein lumen with no or minimal obstruction of the superior mesenteric vein.

• Grade II: greater than 50% occlusion of the main portal vein lumen including its total obstruction.

• Grade III: complete occlusion of the main portal trunk and proximal superior mesenteric vein.

• Grade IV: complete occlusion of the main portal trunk and the superior mesenteric vein.

At venous implantation stage, the surgical approach for thrombosis Grade I-II implies thrombectomy with subsequent portal-portal vein anastomosis. Short- and long-term OLT outcomes in thrombosis of the above extent are generally comparable to transplant outcomes in recipients without preexisting thrombosis [44, 45, 47, 49, 50].

Grade III thrombosis is characterized by greater extent that considerably restricts using thrombectomy. The most commonly used interventions considered for this type of occlusion should be a so called "jump-graft shunt". A donor iliac vein segment is typically used as the shunt. A proximal venous anastomosis is formed with the initial portion of the superior mesenteric vein or, if this is not possible anatomically or otherwise, with one of the venous collaterals. The pericholedochal venous branches and the gastric coronary vein are the ones most commonly used for this purpose [45, 49].

The greatest difficulties with portal revascularization arise in patients with Grade IV portal blood flow occlusion. Decompensated portal hypertension contributes to the severity of somatic status and creates difficulties for restoring a portal blood flow in the graft. The stage of venous implantation to be implemented in PVT Grade IV may have the following options [44-46, 49]:

1. *Anastomosis with splanchnic branches of the portal system.* Theoretically, any venous collateral of 2 cm or more in diameter can be used for anastomosis. The gastric coronary vein, pericholedochal branches, or the middle colic vein are the most commonly used for this purpose.

2. *Arterialization of the portal blood flow* is a technique comprising the formation of arterio- or aorto-portal vein anastomosis. Portal vein is anastomosed to the hepatic or gastroduodenal artery or to the aorta, using an arterial (iliac) conduit. The technique disadvantages include a right ventricle failure, portal vein rethrombosis, graft fibrosis, aneurysmal expansion of the portal vein and its branches.

3. *Porto-renal anastomosis* is indicated for the cases of compromised venous blood flow in the visceral vessels; the one most commonly used for

anastomosis is the left renal vein. This method does not eliminate portal hypertension, so the postoperative course is often complicated by edematous-ascitic syndrome and bleeding from esophageal varices. Moreover, this surgical intervention is characterized by a high frequency of postoperative renal failure.

4. *Portocaval transposition* is the technique applied in rare cases of extended visceral portal vein thrombosis. The blood flow from the inferior vena cava (IVC) is redirected into the graft, the anastomosis can be performed either end-to-end or end-to-side. This technique has been associated with high mortality from septic complications (about 33%), and with postoperative renal failure.

5. *Multivisceral transplantation* is a last resort in the patients with PVT Grade IV.

Despite the obvious technological advances in the field of improving OLT surgical techniques, the thrombosis problem can not be regarded as solved. In comparison with OLT outcomes in the patients without PVT, liver transplantations in the patients with occluded portal blood flow are associated with a grater (approximately 2-fold) intraoperative blood loss, prolonged cold ischemia times, higher rates of early postoperative mortality, lower 1- and 5-year recipient and graft survival rates. The more extended is the thrombosis, the poorer are the short- and long-term OLT outcomes. The portal vein rethrombosis rates after OLT also correlate with the severity of portal vain occlusion [50, 51, 54, 58-61].

The tactics of PVT treatment in transplant patients should be chosen on the basis of obtained clinical and laboratory findings and instrumental test results [4]. Incomplete thrombosis with intact graft function may be treated either conservatively or using endovascular techniques or their combination

[12, 28, 62]. In case of complete occlusion of the portal blood flow in the early postoperative period, it is necessary to attempt thrombectomy. If thrombectomy is deemed impossible or ineffective, then the urgent retransplantation is the only possible treatment [4, 12, 19, 28]. P.A. Clavien and J. Torter (2012) regarded the portal thrombosis with the clinical course of a fulminant hepatic failure as an indication to retransplantation. The authors recommended performing an urgent liver graft biopsy at relaparotomy and thrombectomy; the finding of massive necrosis (> 50%) at biopsy should be the indication for retransplantation [63].

An impaired venous outflow from the liver graft presented as stenosis and thrombosis of the hepatic veins is a rare, but severe complication.

A traditional hepatectomy technique proposed by T. Starzl to be used in a recipient involves the procedure of veno-venous bypass grafting (VVB), a complete cessation of blood flow in the IVC system, and the resection of its retrohepatic part [64, 65]. Severe hemodynamic impairments developing during the anhepatic phase and caused by a decreased venous return to the heart, the risk and technical problems arising during retrocaval space dissection, economic costs, and the complications associated with VVB were the reasons to seek alternative hepatectomy techniques. New methods for a recipient's liver removal were proposed by R. Calne and subsequently popularized by A. Tzakis; hepatectomy was performed preserving the retrohepatic IVC with separate ligation of short and main hepatic veins. The caval implantation was performed in the end-to-side fashion: the suprarenal segment of donor's IVC was implanted to the common orifice formed of the unified left and middle, or all three hepatic veins. The infra-hepatic IVC

segment of donor's liver was ligated or sutured. That hepatectomy technique was termed "piggyback"; and currently it is being widely used by many transplant centers [66, 67].

H. Bismuth, and later on, J. Belghiti modified the technique by proposing to perform the caval implantation in the side-to-side manner with longitudinal dissection of the IVC anterior wall [68]. The surgery was performed by means of partial squeezing of the IVC wall laterally using the Satinsky clamp. Currently, liver transplantation is performed in most cases with the IVC preservation [69]. However, despite the obvious advantages of the "piggyback" technique, the risk of complications arising from this type of caval implantation always exists.

A higher postoperative incidence of hepatic vein stenosis and thrombosis is typical for IVC preservation technique compared to the traditional technique, and ranges within 0.8-10%, depending on its modification [70]. The median venous thrombosis rate makes 4.6% when using the "piggyback" technique up to Tzakis, and 1.4% with the modified technique by Belghiti. The highest complication rates in relation to an impaired venous outflow ranging 3.9-16.6 % have been reported in liver fragment transplantation [71].

The causes of the impaired venous outflow in the hepatic vein system are associated, as a rule, with technical factors that include the following [69, 70, 72, 73]:

- misfit of anastomosed IVC parts by diameter and the specific anatomy of hepatic veins;

- IVS anastomosis compression due to the increased size of the hepatic graft and(or) enlarged segment I of donor liver;

- kinking hepatic veins, the cava-caval anastomosis rotation that can be observed in excess length of IVC and the hepatic veins, and also in liver fragment transplantation (split or related transplantation);

- the proximity of sutures to the inflow orifice of hepatic veins. This situation may arise either directly due to surgical errors or due to the peculiar donor liver explantation where the suprahepatic IVC segment appeared unduly shortened. This may occur at simultaneous retrieval of the donor heart or the cardiopulmonary complex when a significant portion of suprahepatic IVC segment can be removed as a part of the harvested cardiac complex.

Stenosis and thrombosis of hepatic veins usually manifest themselves in the early postoperative period, but their occurrence is possible in the long-term period of OLT [73, 74]. The clinical syndrome that develops in terms of an impaired venous outflow is consistent with the Budd-Chiari syndrome, and characterized by the graft dysfunction, a resistant-to-therapy edematous-ascitic syndrome (recurrent ascites, edema of lower extremities), an impaired kidney function. The clinical symptom severity depends on the degree of hepatic vein stenosis [69, 72, 73, 75, 76]. One should remember that a compromised venous outflow creates preconditions for afferent blood flow impairments in the graft.

The diagnosis of blood flow disorders in the hepatic venous pool shall be made on the basis of using indirect and direct imaging techniques. The US and DUS typical signs of hepatic vein stenosis include a turbulent pattern of blood flow with monophasic waveform. Hepatic vein thrombosis is characterized by echogenic masses present at B-mode exam and by a lacking color signal at duplex scanning. Despite the fact that the MSCT and MRI are increasingly used as noninvasive and highly informative methods

for assessing the efferent venous system of the liver graft, the direct vena cavagraphy still remains the golden standard in diagnosing this kind of complications [72, 76]. The vena cavagraphy evaluates the venous pressure gradient in the anastomosis zone and in the segment between the suprahepatic IVC and the right atrium. It is considered that the values exceeding 5 mm Hg for transanastomotic space, and the gradient value over 10 mm Hg in the area between the suprarenal IVC and the right atrium are considered indicative of an impaired efferent blood flow in the liver graft [72, 77].

A delayed diagnosis of venous outflow impairments in the graft is fraught with a persistent graft dysfunction or the graft loss. The treatment of the impaired hepatic venous blood flow depends on the degree of stenosis, the timing of its occurrence, and specific structural anatomy of hepatic veins. In situations where the compromised venous outflow has been diagnosed intraoperatively or within the first several days after surgery, the optimal correction method will be a repeated surgical intervention and the anastomosis exploration/reconstruction [73]. In case of later diagnosis, the treatment is advisably to start with applying endovascular techniques, namely, the balloon angioplasty of the hepatic veins and the stent placement. We should note that the balloon angioplasty alone is not always capable to resolve the stricture; such cases may require several sessions followed by the placement of hepatic vein stents [69]. Furthermore, it is important to note that the balloon angioplasty used in the early postoperative period may traumatize the anastomosis zone and give rise to hemorrhagic complications [78]. Balloon angioplasty is also ineffective for venous outflow impairments associated with tortuosity of hepatic veins, and the treatment of choice for such cases would be the intervention for stent placement [72, 78]. If all the

above methods appear ineffective, and also in the cases of severe and progressive graft dysfunction, retransplantation remains the only alternative.

The prognosis in post-OLT hepatic vein stenosis/thrombosis is unfavourable: a 1-year graft survival in patients with impaired hepatic venous outflow makes 50%. According to reports by U. Settmacher (2000), M. Ceson (2005), and P. Parilla (1999), the need for retransplantation in patients experiencing that complication made as much as 30%, 40%, and 80%, respectively [69, 79, 80] .

Conclusion

A high percentage of septic complications and mortality associated with a compromised blood flow once prompted to characterize HAT as an "Achilles heel" of liver transplantation; though traditionally this term has been applicable to post-transplant biliary complications.

Vascular complications invariably accompany OLT, and despite their relatively rare occurrence, their consequences are truly disastrous. They also appear the next most common cause of the graft loss after the primary graft non-function, wherein the blood flow impairments play the leading role in the structure of the post-transplant vascular problems. The risk of vascular complications should be considered at all stages of a transplantation procedure: from the removal and preservation of donor organ till its extracorporeal management and further implantation. The improvements of surgical technique, a constant gain of surgical experience, delicate graft handling, strict adherence to the cold preservation regimen, reduction in the cold and warm ischemia time, the correct selection of the recipient, while taking into account all the risk factors in medical history, contribute to reducing the incidence of vascular complications.

Timely diagnosis of vascular complications is the factor that largely determines the efficacy of their treatment. In this regard, we would like to repeat that, in our view, the laboratory monitoring and ultrasonography are the diagnostic methods that should be applied to daily, at least for the first post-transplant week, particularly in the patients compromised by vascular complications. Economic motivation must not restrict the proposed screening model, as the delayed diagnosis of HAT or PVT would bring incomparably more damage, not mentioning a direct threat to patient's life.

We have to admit that given the recent advances in surgery, anesthesiology, intensive care, interventional radiology, and antimicrobial therapy, nevertheless, retransplantation remains the most demanded treatment of HAT, PVT and hepatic vein thrombosis consequences. One should remember that in acute HAT or PVT and no prospects with any treatment other than retransplantation, the time margin is very small and usually amounts to several days. Therefore, along with the timely determined indications for retransplantation, the success of HAT and PVT treatment is greatly depends on the level of the existing organ donation system.

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