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THE ROLE OF ENDOVASCULAR AND ENDOBILIARY METHODS IN THE TREATMENT OF POST-LIVER TRANSPLANT COMPLICATIONS

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Liver transplantation is the treatment of choice for patients with end-stage liver disease or acute liver failure. However, vascular complications, such as hepatic artery stenosis and/or thrombosis, graft portal vein stenosis and biliodigestive strictures following liver transplantation are still common despite improvements and innovations in surgical techniques. These complications can lead to graft damage or even death, and they are caused by many factors. Although minimally invasive interventional radiology is an optional treatment for such post-liver transplant complications, there is little research on this method of treatment.

Keywords: liver transplantation, endovascular treatment, endobiliary treatment, hepatic artery stenosis, hepatic artery thrombosis, portal vein stenosis, biliodigestive strictures.

INTRODUCTION

The American Association for the Study of Liver Diseases and the American Society of Transplantation suggest that screening and detection of post-liver transplant surgical complications be performed regularly and routinely [1].

Surgical complications such as hepatic artery stenosis and thrombosis, portal vein stenosis and biliary strictures are optimally diagnosed and treated at the transplant center. However, there is no consensus on the monitoring of these complications and their treatment tactics.

HISTORICAL ASPECTS OF THE DEVELOPMENT OF MINIMALLY INVASIVE TREATMENT METHODS

The development of catheter technology dates back to ancient times. The ancient Egyptians, 3,000 BC, were the first in the world to perform bladder catheterization using special tubes. Erasistratus was the first to use the word "καθετηρ" (catheter) around 300 BC. to describe this instrument. Classic S-shaped catheters date from this period, and some of them were discovered during excavations of the house of a surgeon in Pompeii, who was buried by a volcanic eruption in 79 AD [2].

In 1711, Dutch physiologist Stephen Hales performed the first catheterization of the heart chambers in a horse using brass and glass tubes [3].

A very important discovery for further development of medicine was made by Wilhelm Roentgen, who in 1895, discovered radiation, which he called X-rays [4]. By 1896, Vladimir Bekhterev had predicted the discovery of angiography: "...*Since it became known that some solutions do not transmit X-rays, brain vessels can be filled with them and photographed in situ*" [5].

The year of emergence of interventional radiology can be considered 1929. Werner Forssmann, a 25-year-old resident surgeon at Auguste Viktoria Home Red Cross Hospital in Eberswalde, Germany, in an experiment, for the first time in the world, proved the safety of inserting catheters into the human heart when he performed a catheter through the ulnar vein into the right atrial cavity [6].

In 1953, Swedish doctor, Sven Seldinger, was the first to use a technique for obtaining puncture access to the Seldinger's artery, which laid the foundations of modern interventional radiology [7].

In 1958, Mason Sones, a pediatric cardiologist at the Cleveland Clinic (USA), was the first to record X-ray contrast images of coronary arteries on film during aortography [8]. The first selective coronary angiography in the USSR was performed in 1971 by Yu.S. Petrosyan and L.S. Zingerman at Bakulev Institute of Cardiovascular Surgery under the USSR Academy of Medical Sciences [9].

In 1977, Andreas Gruentzig and Richard Myler at St. Mary's in San Francisco (USA) performed the first coronary balloon angioplasty in humans [10]. The first coronary balloon angioplasty procedure in the USSR was performed by Rabkin and Abugov at the All-Union Scientific Center of Surgery in 1982 [11].

The end of the 20th century was the heyday of endovascular surgery, whose methods have been applied in other fields of medicine.

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Bjerkvik *et al.* [12] in 1989 described a case of percutaneous graft revascularization by transcatheter fibrinolysis in one patient with hepatic artery thrombosis (HAT). However, balloon angioplasty (BA) was required in this case. The role of fibrinolysis in the treatment of early HAT remains a subject of dispute. Hidalgo *et al.* [13] in 1995 achieved good outcomes using urokinase for local fibrinolysis in two patients with early HAT; however, after fibrinolysis, both patients also required additional BA.

Angioplasty and portal vein stenting were described for the first time in 1990 by Olcott *et al.* [14] at the University of California, San Francisco, USA. Raby *et al.* [15] was the first to propose portal angioplasty in children. In subsequent years, this technique has become the treatment of choice for post-transplant portal stenosis with good outcomes and low rate of postoperative complications.

In 2001, Schwarzenberg et al. from the University of Minnesota, USA, reported positive outcomes in the relief of biliary stricture [16]. Balloon dilatation of the anastomotic stenosis and installation of external-internal drainage were performed in 6 patients after developing biliary strictures.

HEPATIC ARTERY STENOSIS AND/OR THROMBOSIS AFTER LIVER TRANSPLANTATION

Arterial blood flow disorders include hepatic artery thrombosis (HAT), hepatic artery stenosis (HAS), kinked or tortuous hepatic artery, and hepatic graft arterial steal syndrome.

Depending on the time interval between liver transplantation and HAT, early HAT (up to 4 weeks) and late HAT (over 4 weeks after transplantation) can be distinguished).

Bekker *et al.* [17] conducted a systematic literature review, which showed that the incidence of early HAT in children and adults after liver transplantation is 8.3% and 2.9%, respectively. Timely diagnosis and treatment of HAT can prevent injury to the biliary tract and liver transplant parenchyma.

RISK FACTORS FOR HEPATIC ARTERY GRAFT STENOSIS AND/OR THROMBOSIS

Donor risk factors

Atypical liver arterial anatomy on the donor side is a risk factor, especially in cases of "caliber" mismatch during formation of liver graft arterial anastomosis [18].

As for the graft-to-recipient weight ratio (GRWR), data obtained from various studies are still debatable. Sanada *et al.* [19] reported that grafts with GRWR <1.1% are a risk factor for hepatic artery thrombosis.

According to a study by Li *et al.* [20], grafts with GRWR ≥4% are significantly associated with development of HAT in related liver transplantation in children. However, Uchida *et al.* [21] showed that grafts with a GRWR ≥4% can be used safely in pediatric transplantation.

Besides, defects in surgical technique during graft retrieval are an important risk factor for HAS and/or HAT [22].

Recipient risk factors

Uchida *et al.* [21] also analyzed the risk factors for liver graft arterial blood flow disorders. It was established that gender (female), body weight (lower), and GRWR (higher) were associated with the risk of developing HAT.

A subject of disagreement is the recipient's low weight as a risk factor for thrombosis. Several studies have reported that the recipient's body weight is not a risk factor for HAT [19, 23]. Meanwhile, Desai *et al.* [24] found that the risk of developing HAT is higher in patients weighing less than 10 kg.

Atypical arterial anatomy of the recipient is also a risk factor for developing HAS and/or HAT [25].

Preoperative factors

According to Uchida *et al.*, long cold ischemia time and long warm ischemia time were factors leading to HAT [21].

However, according to the Organ Transplantation Center in Tianjin, China, there was no association found between high risk of HAT and prolonged cold ischemia time [26].

Intraoperative factors

With regard to transfusion risk factors, Uchida *et al.* [21] reported that over 6 doses of red blood cell suspension and/or transfusion of over 15 doses of fresh frozen plasma during surgery are risk factors for HAT.

According to some authors, the priority in using a microscope rather than conventional surgical optics during formation of hepatic artery anastomosis does not change HAT incidence [27, 28].

Backes *et al.* [29] reported that the use of a vascular insert in hepatic artery anastomosis is a useful option for pediatric liver transplantation. In contrast, however, Duffy *et al.* [30] reported that the use of a vascular insert is a significant independent risk factor for HAT.

According to Julka *et al.* [31] and Uchida *et al.* [21], multiple hepatic artery anastomoses had no effect on development of HAS and/or HAT. Nonetheless, Seda-Neto *et al.* [32] found a protective effect against development of thrombosis during formation of two arterial anastomoses.

Secondary edema of the liver graft resulting from ischemic reperfusion "stroke" is also a risk factor for HAT [33].

Postoperative factors

A group of authors believes that the presence of HAS and hepatic artery kinking (HAK) in the recipient after transplantation are initiating factors for development of HAT [34].

Early administration of aspirin has also been shown to be effective in preventing HAT [35].

DIAGNOSIS OF HEPATIC ARTERY STENOSIS AND/OR THROMBOSIS AFTER LIVER TRANSPLANTATION

Typically, the clinical presentation of HAS and/or HAT includes moderate increase in serum transaminase and bilirubin levels (75%), biliary complications (15%), fever and sepsis (6%), dysfunction or liver failure (4%) [36]. HAT can present as an isolated elevation in markers of cytolysis enzymes or as sepsis resulting from severe graft dysfunction.

HAS and/or HAT are most often detected by Doppler ultrasound followed by CT angiogram. According to some studies, Doppler ultrasound showed a 92–100% sensitivity and a 99.5% specificity [37].

The Doppler ultrasonic signs of HAS and/or HAT include peak hepatic artery systolic velocity <20.0 cm/s or resistance index <0.6 distal to the anastomosis region [38].

However, a vast majority of researchers believe that angiography is the gold standard for HAS and/or HAT diagnostics.

TECHNIQUES FOR MINIMALLY INVASIVE CORRECTION OF HEPATIC ARTERY STENOSIS AND/OR THROMBOSIS

A study by Chen *et al.* [18], as well as studies by Yanaga *et al.* [39] demonstrated that urgent revascularization in cases of early HAT after liver transplantation can significantly reduce graft loss and eliminate the need for retransplantation. The above studies showed that early revascularization achieved 55% graft recovery, whereas late revascularization was unsuccessful in 100% of cases.

Sanada *et al.* presented an extensive retrospective study in 2018 [40]. From May 2001 to September 2016, 279 related liver transplants were performed in 271 children.

Posttransplant hepatic artery complications were found in 15 cases (5.4%), which includes HAT and HAS in 14 (5.0%) cases and occlusion due to compression by fluid accumulation in one case (0.36%). Minimally invasive correction was the first-line treatment in seven cases (46.7%). The success rate in cases of minimally invasive techniques for HAS and/or HAT was 100%. Besides, graft survival rate in patients with vascular complications was 94.4% in the present study.

Techniques for performing endovascular correction of HAS and/or HAT have been widely used in adult patients after liver transplantation for many years; however, as for pediatric practice, these minimally invasive methods of treating complications are poorly transposed.

LIVER

Arterial hypoperfusion of a liver graft in the absence of hepatic artery occlusion was first described by Langer *et al.* in 1990 [41]. This complication occurs in the early postoperative period in more than 80% of diagnosed cases [54–74].

Angiography is the gold standard for diagnosing splenic steal syndrome. The diagnosis is determined by reduced blood flow through the hepatic artery in the absence of significant arterial anatomical defects, such as HAS, HAT or HAK [39, 42]

Thorough assessment of celiac trunk angiography to identify underlying vascular defects (HAS, HAT or HAK) is the first step [43]. Splenic artery embolization is considered to be the method of choice for shifting the hemodynamic balance in favor of the liver graft, as well as decreasing hyperdynamic portal blood flow [44].

POST-LIVER TRANSPLANT PORTAL VEIN STENOSIS

Interventional radiology is now widely used, and is considered a safe and effective treatment for portal vein stenosis (PVS) of hepatic graft [45]. Funaki *et al.* [46] reported that portal vein balloon angioplasty for treatment of PVS had a 50% recurrence rate on average at 6.3 months, while stenting showed 100% portal vein patency at 47 months of follow-up.

Portal inflow disorders after liver transplantation can be classified as early (detected within 3 months after liver transplantation) or late (detected more than 3 months after liver transplantation) [68]. As for portal vein thrombosis, endovascular methods have not been shown to be as effective as they should be [47]. In cases of early portal thrombosis, open thromboextraction is the appropriate treatment, and in cases of late portal thrombosis, formation of a mesenteric-portal bypass (Meso-Rex) shunt [48].

In cases of orthotopic whole liver transplantation, incidence of PVS is quite low in adult patients.

The most common indication for liver transplantation in children is biliary atresia [49]; in this disease, portal vein hypoplasia is quite common in patients. This factor provokes the development of PVS, and also complicates the formation of portal anastomosis due to the discrepancy between the "calibers" of the donor portal vein and the recipient's portal vein [50].

RISK FACTORS FOR PORTAL VEIN STENOSIS

The risk factors for portal vein complications include technical difficulties in anastomosis formation, young age, body weight <6 kg, recipient portal vein diameter <3.5 mm [51], graft rotation, simultaneous thrombectomy of preexisting portal vein thrombosis, and use of vascular conduits for portal reconstruction [50].

Several surgical techniques can play an important role in preventing PVS, especially in related liver transplantation or split liver transplantation. To overcome the discrepancy between the calibers of the donor and recipient portal veins, the use of "growth factor" in the vascular suture has proven to be an effective method.

Another method is to ensure adequate blood flow – ligation of the small portal branches on the recipient side. The use of large grafts can cause compromised blood flow during abdominal closure, which can lead to vascular thrombosis. In this case, delayed closure of the anterior abdominal wall is used to prevent a sharp increase in intra-abdominal pressure.

DIAGNOSIS AND TREATMENT OF PORTAL VEIN STENOSIS AFTER LIVER TRANSPLANTATION

The clinical manifestations of PVS range from asymptomatic to severe symptoms, including massive ascites, anemia, persistent splenomegaly with or without thrombocytopenia, and gastrointestinal bleeding [52]. Platelet counts may be below normal due to hypersplenism in patients with portal vein stenosis [53]. Portal vein stenosis is usually detected on routine Doppler ultrasound, CT scan, or MRI.

Currently, two types of endovascular approaches are widely used. The antegrade method – access to PVS is secured through the mesenteric vein system, from a mini-access. The second, less invasive approach is considered to be the retrograde method – access to the portal vein branches is secured by percutaneous transhepatic puncture of these branches under ultrasound control [54].

Among researchers of this complication, there is also an "opposition" to the methods of direct correction of stenosis, wondering whether balloon angioplasty or stenting is a necessary option for PVS treatment.

Sakamoto *et al.* [55] and Bertram *et al.* [56] demonstrated that balloon angioplasty is an effective and relatively safe method of treating PVS; however, 28–50% of patients develop PVS recurrence after the procedure. These studies suggest stenting and/or repeated balloon angioplasty to address this issue. Cheng *et al.* [47] also reported the efficacy of stenting for PVS in adults and children. Stent patency rate was 90.9% over a mean follow-up period of 12 months.

In contrast, other studies have raised concerns about the side effects of stenting [57, 58]. These side effects include intimal hyperplasia, size mismatch during retransplantation, and possibility of stent migration.

In 2019, Katano *et al.* [59] presented an extensive retrospective study at the Jichi Medical University, Japan. A related liver transplant was performed in 282 children. Portal vein complications occurred in 40 (14.2%) cases. In 36 cases, balloon angioplasty was performed. In 4 patients, portal vein stenting was carried out. Recurrence occurred in 27.5% of the patients after the initial treatment. Stent patency rate was 100%.

In 2017, a major collaborative study was conducted by Incheon St. Mary's Hospital, Korea and Asan Medical Center, Seoul, Korea [60]. Of the 296 patients, 55 (18.6%) developed PVS.

12 patients underwent balloon angioplasty, and 41 patients underwent stenting. There was 89% success with balloon angioplasty. Relapses occurred in 3 (25%) patients. Stenting was performed if, after angioplasty, the deployed balloon demonstrated waist deformation >50% or portal pressure gradient was >5 mm Hg. Satisfactory portal blood flow was observed in all patients who underwent stenting. The 1-, 5-, and 10-year primary stent patency rates were 90% (±7%), 90% (±7%), and 85% (±8%), respectively.

POST-LIVER TRANSPLANT BILIARY STRICTURES

Biliary strictures and biliary fistulas are the most common early post-transplant complications, with a 10–30% risk according to various sources [61, 62].

Biliary strictures are classified into anastomotic (AS) or non-anastomotic biliary strictures (NAS) [40].

RISK FACTORS FOR POST-LIVER TRANSPLANT BILIARY STRICTURES

The risk factors for biliary strictures (BS) are: cold ischemia time of the liver graft, impaired arterial blood supply to the graft, rejection, and cytomegalovirus (CMV) infection [63].

Several authors have provided studies proving that there is a relationship between BS occurrence and preexisting arterial complications. Thus, according to the data obtained by Darius *et al.* [64], HAT increased the risk of anastomotic BS in children ($p < 0.001$). In 2018, Fang-Min Liao *et al.* [38] demonstrated that children after liver transplantation with a hepatic artery resistance index according to Doppler ultrasound ≤0.57 had a higher risk of BS ($p = 0.001$). Feier *et al.* in 2016 reported that multiple arterial anastomoses can "protect" a child from developing biliary strictures [65].

DIAGNOSIS AND TREATMENT OF POST-LIVER TRANSPLANT BILIARY STRICTURES

Clinically, biliary strictures should be suspected in patients with signs of cholestasis or episodes of cholangitis. However, most patients have a nonspecific clinical picture, as well as discrete changes in liver enzyme levels. Ultrasound does not usually reveal significant

changes, while magnetic resonance cholangiography, an instrument superior to ultrasound, is a priority method in non-invasive imaging diagnosis of this complication [66].

Percutaneous transhepatic cholangiography plays a crucial role in the diagnosis of post-liver transplant biliary stricture, as it is considered the gold standard for detecting and quantifying stenosis [67].

Percutaneous transhepatic bilioplasty (PTB) is a minimally invasive method and has a success rate of 34% to 75%. Its outcomes are similar to those of surgical revision [61, 62]. The main disadvantage of PTB is the potential need for prolonged external biliary drainage and repeated procedures, with potential psychological consequences.

Belenky *et al.* [68] recommended that stent placement should be a priority treatment option in cases of late biliary strictures. This method provides long-term outcomes that are superior to those obtained with isolated dilation with balloon catheters.

There are no large cohort or randomized controlled trials to compare the short-term and long-term outcomes of PTB and surgery. In most published studies, only short-term or mid-term observations are available. In addition, different centers use different methods and/or therapeutic algorithms, making it difficult to compare outcomes and complications reported in the literature.

In 2008, Miraglia *et al.* reported PTB outcomes in 20 children who underwent liver transplantation between 2004 and 2007 [69]. Biliary anastomoses stricture was successfully completed in all patients, after which PTB was performed without major complications. The average number of balloon dilations performed was 4. Cholangiostomy drainage placement lasted for 5 months on average. Recurrent stenosis developed in 28%, which required a second series of PTB.

Normalization of liver enzymes and resolution of intrahepatic biliary dilatation are the endpoints used to measure technical success in the most recent series involving PTB or surgical reconstruction of biliary strictures [62, 63].

CONCLUSION

In cases of development of post-liver transplant complications in adult patients, endovascular and endobiliary methods for correcting these complications have become the first-line therapy, since they are less invasive and easier tolerated by recipients compared to volumetric reconstructive surgery. In recent years, with the development of minimally invasive technologies and techniques, the number of new cases of treatment of graft blood supply disorders and biliary strictures has increased. Although urgent retransplantation has long been considered the treatment of choice, minimally invasive interventional radiology is now being used as a first-line treatment for adult recipients in a number of leading centers.

However, in pediatric liver transplantation, endovascular technologies and techniques have not been as widely studied. Development of algorithms for minimally invasive interventional diagnosis and treatment of vascular complications and biliary strictures following a liver transplantation for pediatric practice is necessary to achieve long-term optimal outcomes and graft function.

The authors declare no conflict of interest.

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