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Arterial complications after liver transplantation: diagnosis, prevention and treatment

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Abstract

Background. *Liver transplantation is a life-saving surgery for patients with chronic end-stage liver diseases and individual patients with fulminant liver failure. Over the years, the procedure of the operation has undergone major changes. Recent advances in this field, including improved surgical techniques and the introduction of new*

immunosuppressive agents, have made it possible to achieve a 5-year survival rate of 87.6%.

Objective. *To analyze the current scientific literature on arterial complications after liver transplantation, their diagnosis and treatment methods.*

Material and methods. *The scientific articles, reviews and other literature on the topic of arterial complications after liver transplantation for the period from 2015 to the present, published in the databases Pubmed, Google Scholar, Medline, have been studied.*

Conclusion. *Arterial complications remain one of the most dangerous consequences of orthotopic liver transplantation, accompanied by a high risk of graft loss and death. The diagnosis and treatment of these complications is a significant challenge that requires a further search of approaches to improve the efficacy of liver transplantation.*

Keywords: liver transplantation, arterial complications, hepatic artery thrombosis, hepatic artery stenosis, risk factors, liver graft survival

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AC, arterial complication
BA, balloon angioplasty
CMV, cytomegalovirus
CT, computed tomography
HA, hepatic artery
HAA, hepatic artery aneurysm
HAS, hepatic artery stenosis
HAT, hepatic artery thrombosis
IATL, intra-arterial thrombolysis
LHA, left hepatic artery

LT, liver transplantation
PTLAP, percutaneous transluminal angioplasty
RHAA, reconstructed hepatic artery aneurysm
USE, ultrasound examination

Introduction

Liver transplantation (LT) is the only surgical treatment for end stages of liver failure, including fulminant forms [1]. Modern studies demonstrate high recipient survival rates: one-year survival reaches from 80–99%, and ten-year survival averages 71% [2]. However, postoperative arterial complications (ACs) remain a significant factor affecting the long-term prognosis and functional viability of the graft [3].

Despite the relatively low incidence, ACs are characterized by a high risk of graft loss and death due to impaired vascularization of the transplanted organ [4].

Statistical data show variability in the incidence of ACs depending on the type of donation: it is about 7% [3–5] in transplantation from a deceased donor, and it reaches 13% in transplantation from a living donor. The overall incidence of ACs in adult recipients varies in the range from 7.2–15%, while in the pediatric population, this parameter shows a tendency to increase [5, 6].

An analysis of modern scientific literature allows us to systematize ACs after LT into the following categories:

- Hepatic artery thrombosis (HAT), which incidence varies between 1.9–16.6%
- Hepatic artery stenosis (HAS) or arterial anastomotic stenosis observed in 0.8–13% of cases
- Hepatic artery aneurysm (HAA) occurring in 0–3% of cases
- Hepatic artery (HA) rupture recorded in 0.64% of cases [6, 7].

According to the time criterion, these complications are classified as early, developing within the first 4 weeks after transplantation, and later one, occurring at 4 weeks or more after surgery.

Arterial complications are relatively rare, but they pose the greatest threat to the patient, as they often lead to the graft dysfunction requiring retransplantation [8, 9].

Risk factors for the development of arterial complications

Hepatic artery thrombosis (HAT) is the most common AC after liver transplantation, accounting for up to 50% of all ACs. In most cases, the HAT development requires organ retransplantation. Regarding the occlusion nature, the occlusive thrombosis, typical for the early postoperative period should be distinguished from non-occlusive thrombosis, which mainly develops in the later period [10, 11]. This complication may develop due to a combination of surgical and non-surgical factors. Surgical factors include technical peculiarities of the vascular anastomosis formation, mismatch of the graft size, small artery diameter (less than 3 mm), mismatch in the diameters of the donor and recipient vessels, as well as anatomical characteristics of the vascular bed structure [11, 12]. Non-surgical factors include the presence of hereditary thrombophilia, donor cytomegalovirus (CMV) infection, graft cold and warm ischemia time, donor age and ABO incompatibility [12, 13].

HA stenosis develops as a result of vascular intima trauma during surgery, which leads to fibroblast proliferation. A significant role in the pathogenesis is played by an impaired blood supply to the vascular wall and immunological factors, in particular, an acute cellular rejection [13, 14].

The most unfavorable complication is HAA characterized by a mortality rate exceeding 50% [14, 15]. The aneurysm development is caused by both mechanical factors, including the characteristics of surgical

reconstruction, trauma to the vascular wall and disruption of its blood supply, and infectious complications associated with biliary disorders, failure of biliodigestive anastomoses and enteritis [13, 15].

Treatment of arterial complications

Currently, endovascular treatment methods, including selective thrombolysis, balloon angioplasty (BA) and stenting, are becoming the method of choice in the treatment of ACs after liver transplantation [16, 17]. The minimally invasive nature of these interventions performed under the control of computer visualization ensures better tolerability of the procedures by patients [16, 18]. Timely diagnosis and the use of endovascular methods are crucial for preserving the graft and improving recipient survival rates [18–20].

The blood supply to the liver is ensured from two sources: the portal vein and the HA, which provide adequate perfusion of both the organ parenchyma and the biliary system. The typical anatomical picture is represented by a single HA, which departs from the celiac trunk together with the splenic and left gastric arteries. The general HA is divided into the gastroduodenal and proper HA, which in turn forms the right and left branches [19, 20].

The HA variation anatomy classified according to Michels and Hiatt systems is represented by various anatomical configurations [20, 21]. The most common variations are the origin of the left hepatic artery (LHA) from the left gastric artery and that of the right hepatic artery from the superior mesenteric artery. Aberrant arteries can be either additional or substituting, which is determined by their role in the blood supply of the respective liver lobe [21].

The identification and proper evaluation of aberrant arteries are critical at the stage of donor organ procurement. However, it may be

difficult to verify the aberrant vessel nature (additional or replacing) intraoperatively due to the impossibility of examining intrahepatic branches [22]. The strategy of preserving aberrant arteries aimed at ensuring adequate blood supply to the graft may require multiple arterial anastomoses. This, in turn, increases the risk of postoperative complications, including stenosis, thrombosis, aneurysm formation, and bleeding [6, 23].

The phenomenon of arterial collaterals deserves special attention. Unlike the native liver, where the HA ligation can be compensated by collateral blood flow, in liver transplantation, the arterial blood supply disruption often leads to serious complications due to the absence of collateral circulation [24]. The graft viability in conditions of the compromised arterial blood flow is possible only with a sufficient portal blood flow and the development of new collaterals [17, 25, 26].

When planning surgical treatment of complications after liver transplantation, it is essential to consider the type of arterial anastomosis performed [24, 27]. The quality of arterial reconstruction plays a decisive role in preventing thrombosis, which can lead to biliary complications and a graft loss. This stage of surgery is often the most technically difficult and may require the use of microsurgical techniques [25, 27].

In the standard HA anatomy, an end-to-end anastomosis is traditionally performed between the gastroduodenal artery site of the graft and the recipient's common HA, creating a wider site by dissecting the latter [26, 27]. In cases of a short, small-diameter donor HA or the celiac trunk stenosis, an intermediate conduit can be used, most often that of an iliac artery allograft, creating an end-to-side anastomosis with the abdominal aorta [27, 28]. The use of conduits from donor gastroduodenal, splenic, right gastroepiploic, radial, superior and inferior mesenteric arteries, and the great saphenous vein has also been described [28, 29].

The HA variation anatomy requiring complex reconstruction potentially increases the incidence of ACs [6, 27]. Meanwhile, studies show that the best results are obtained with the shortest possible reconstructions [16, 29], although an adequate technical implementation allows for good long-term results to be achieved with any anatomical variations [6, 30].

Contemporary studies demonstrate that in the presence of an aberrant LHA, it is preferable to perform an additional anastomosis with a recipient branch, for example, with the gastroduodenal artery, instead of preserving the long native artery [31]. Excessive vessel length and an altered angle of its origin are significant risk factors for the development of thrombosis [5, 6].

It is important to note that arterial reconstruction in the presence of variation anatomy requires a longer warm ischemia time due to the need to perform multiple anastomoses before restoring blood flow [29, 32]. R. Montalti et al. proposed a method for preliminary preparation of the arteries of the graft and recipient to perform a single anastomosis while maintaining adequate blood supply to all parts of the donor liver [13], which reduces the incidence of postoperative complications [10, 11].

Research by R. Karakoyun et al. did not reveal significant differences in the incidence of biliary and arterial complications or in the graft and patient survival rates between the groups with standard and variation anatomy [33, 34]. At the same time, the incidence of HAT in arterial reconstruction in the presence of variation anatomy was 0.7% [35, 36].

In transplantation of a liver fragment from a living donor, performing a single anastomosis with the left lobe in the presence of two arteries did not affect the patient survival or the incidence of biliary complications [24, 37]. S. Yilmaz et al. recommend reconstructing both arteries if technically

possible, although reconstruction of a single artery is not a risk factor of the graft dysfunction [18, 19].

Hepatic artery thrombosis after liver transplantation

The etiology and pathogenesis of this complication remain quite controversial and, in most cases, unknown. Initially, this complication was most common in children. However, due to improvements in surgical technique, postoperative care, and immunosuppression methods, its incidence has decreased significantly. Nevertheless, it remains one of the most severe complications, which significantly increases morbidity, the risk of graft loss, and mortality. Currently, it accounts for more than 50% of all ACs and is still more common in children [24, 25].

The incidence of early HAT after LT varies from 1.9% to 16.6% [11, 26, 27]. The study by J. Bekker et al., which included 21,822 patients after LT, identified 843 cases of early HAT among both adults and children, which amounted to an overall incidence of 4.4% [15, 29, 30]. In adults, this figure was 2.9%. Before the implementation of microsurgical techniques, the incidence of HAT reached 14–25% [6, 29]. In 80% of patients, blood flow in the HA was ensured using a conduit made from donor's saphenous vein [36, 37].

Reconstructions of the HA in living donor liver transplantation differ significantly from similar surgery with using organs from deceased donors. There are multiple anatomical variations both in donor and recipient, small vessel diameters or damage to them are often encountered, which requires using a wide range of reconstructive techniques [27, 38].

The use of microvascular surgery has reduced the HAT incidence to 1.7%. The use of surgical six-fold magnifying lens has shown similar

or even better results in both adults and children compared to an operating microscope [5, 39].

The mean time to detect early HAT is 6.9 days, while later HAT is 6 months [5, 40]. Besides, no significant association was found between the HAT incidence and the choice of using organs either from deceased or living donors.

The prevalence of late HAT varies from 1% to 25% [4, 41]. J. Torras et al. reported an incidence of 7.5% (35 of 413 cases) [12]. Of these, 16 cases occurred within the first month after transplantation (early HAT), the diagnosis being made at days 1–13 after surgery. Later HAT diagnosed in 12 cases occurred 30 days or more after surgery, with a median detection time of approximately 5 months [8, 41].

Risk factors for hepatic artery thrombosis after liver transplantation

Risk factors for the HAT development have been identified, but systematic analyses of the impact of HA anatomical features and the type of reconstruction on LT results are still insufficient [7, 42]. The quality of vascular reconstruction plays a key role in the prevention of arterial thrombosis.

The development of HA-associated complications after transplantation is attributed to many causes. They are HA anatomical variations, small diameter of vessels, damage that occurs during organ removal or anastomosis, prolonged clamping, vessel kinking, excessive length, and arterial wall hematomas. In addition, the quality of the vessels and the discrepancy between the lumen diameters of the donor and recipient arteries require a careful approach to the choice of reconstructive technique and monitoring during surgery [19, 43].

In the early postoperative period, the number of arterial anastomoses performed may be an additional risk factor for HAT, while later complications more often have other causes. Up to 20% of HAT cases are associated with arterial anastomosis and difficulties in its implementation. Sometimes HA complex reconstructions using allografts are required. Additional risk factors may include poor quality of donor or recipient vessels and high resistance to microvascular outflow caused by the graft rejection or severe ischemic damage [20, 44].

As reported, HAT can develop within a few hours after liver fragment transplantation. Small diameter (<2 mm) of the vessel in the donor or recipient is one of the significant risk factors [8, 9, 45]. An additional risk factor for intimal dissection may be previous intra-arterial treatment of hepatocellular carcinoma before transplantation [8, 46].

Previous surgical interventions may also complicate the identification and incision of the recipient artery due to dense adhesions formed.

Among the non-surgical risk factors for HAT, the authors highlight the following: donor age over 60 years, prolonged cold and warm ischemia, ABO incompatibility, hypercoagulation diagnosed by thromboelastography, positive CMV status of the donor with a negative recipient status, episodes of graft rejection, retransplantation in primary sclerosing cholangitis [25, 47].

It has been noted that positive CMV status of the donor and negative status of the recipient may be associated with an increased risk of late HAT [3, 48].

However, some studies do not confirm a correlation between cold ischemia time, donor age, and the risk of HAT development [6]. This highlights the difficulty of accurately identifying risk factors, particularly for early HAT.

HAT is more common in clinics with little experience (less than 30 operations per year), but with an increase in the number of transplants performed, the incidence of complications decreases, indicating the importance of professional experience [25, 49].

Some researchers believe that the initiating factors for HAT development are stenosis and HA kinking, while others note the key role of hypercoagulation in the immediate postoperative period diagnosed by using thromboelastography [49].

A significant increase in HAT incidence is observed in patients with complicated arterial reconstructions (10.5% versus 2.0%) [6], although there are studies that do not confirm this correlation [2].

In their study, L.M. Marín-Gómez et al. found that intraoperative blood flow velocity in the HA may be a prognostic factor for HAT. A flow rate of less than 100 mL/min during surgery is associated with an increased risk of thrombosis [4].

The use of arterial allografts as conduits is also considered a risk factor requiring preventive measures [8]. In patients with hereditary thrombophilic diseases, the prevention of thrombotic complications is recommended.

Clinical presentation of hepatic artery thrombosis (early and late) after liver transplantation

Clinical manifestations of HAT after LT are characterized by a moderate increase in the level of alanine aminotransferase, aspartate aminotransferase, and bilirubin. Characteristic complications of this condition are biliary tract pathology (15%), pain, fatigue, fever, leukocytosis and sepsis (6%), a complete graft dysfunction or loss (4%) [8].

The presentation of HAT depends on both the time after transplantation and the presence of collateral circulation [8], which can

form as early as two weeks after surgery. Early HAT is usually accompanied by primary graft non-function or its severe dysfunction. Disruption of the blood supply to the epithelium of the bile ducts and hepatocytes leads to their damage [6, 46].

Thus, there are two main forms of HAT:

- **Early HAT (acute course)** accompanied by a severe clinical course with fever, significant leukocytosis, and increased liver enzymes. Biliary necrosis that occurs with early HAT can lead to uncontrolled septic shock and patient's death [25, 47].
- **Late HAT (delayed course)** is usually characterized by a milder clinical presentation, although it is often asymptomatic. In 50% of patients with late HAT, only an increase in functional liver tests is observed without obvious clinical signs [7, 48].

Pathophysiological features of hepatic artery thrombosis

Early HAT leads to the bile duct epithelial damage and massive necrosis of hepatocytes. This is associated with impaired arterial blood flow both in the main HA and in additional collaterals. As a result, a high incidence of biliary sepsis is observed in the early stages of the disease [25, 49].

Late HAT is likely due to ischemic or immunologic mechanisms and may be occult. Patients with late HAT often have biliary complications including recurrent cholangitis, bile duct stricture or stenosis, biliary fistulas, biliary necrosis, abscess formation accompanied by recurrent fever and bacteremia [25, 49].

In rare cases, late HAT can lead to ischemia and liver failure, aggravating the clinical situation [25].

Diagnosing the hepatic artery thrombosis after liver transplantation

Early diagnosis of HAT is critical to prevent a graft loss and initiate therapy in a timely manner. In most cases, early HAT is detected during a routine postoperative ultrasound examination (US) before complications develop. Ultrasound is a proven, noninvasive, and cost-effective diagnostic method. Absence of blood flow in the HA in color duplex ultrasonography is the most common finding in HAT. The diagnosis may also include the absence of blood flow in the intrahepatic branches of the artery. In 92% of cases, absent blood flow signals in the HA confirm the diagnosis [5].

To clarify the diagnosis, the contrast-enhanced abdominal computed tomography (CT) and/or the visceral angiography may be used. Ultrasound and CT angiography remain noninvasive and effective imaging tools, while traditional angiography is used to confirm the diagnosis and, if necessary, perform interventional procedures [9].

Protocols for monitoring the HA condition after liver transplantation vary among medical institutions. However, ultrasound allows for the detection of decreased or absent blood flow with high accuracy, which makes it possible to perform revascularization in a timely manner and save the graft and the patient [22].

The diagnosis is confirmed by CT angiography or conventional angiography, which also allow an accurate detection of anatomical abnormalities such as stenosis or kinking of the artery with high sensitivity and specificity [15].

E. Pareja et al. [7, 43] proposed an early screening protocol for HAT, including ultrasound examination within the first 48 hours after liver transplantation and a repeat examination after 7 days. If signs of ACs were detected during the first examination, the ultrasound with a

contrast agent or CT is recommended. If the diagnosis is confirmed, arteriography is performed [5, 42].

Regular postoperative ultrasound monitoring, including annual examinations, help to detect intimal hyperplasia, which can lead to progressive HA stenosis and precede the late HAT. In some cases, HA stimulates the development of collateral circulation, protecting the liver from ischemia in case of HAT [9, 33].

O. Abbasoglu et al. found that the sensitivity of ultrasound in detecting the HA stenosis reaches 85%. Early diagnosis using ultrasound demonstrates a sensitivity of 100%, specificity of 99.5%, positive predictive value of 95%, and overall diagnostic efficiency of up to 99.5% [25, 49].

To clarify the diagnosis, a multidetector computed tomographic angiography and the standard angiography, which is considered the “gold standard” for diagnosing HAS, are also used [9, 33].

The main methods of HAS treatment include transcutaneous transluminal angioplasty (PTLAP) with or without stent placement, surgical revision, and retransplantation.

PTLAP is a highly effective treatment method, but its use is accompanied by 7–12% complications, such as arterial dissection and rupture, restenosis (25%) or thrombosis. Moreover, 12% of procedures turn to be unsuccessful [6, 49].

Surgical revision and retransplantation provide a high success rate but are associated with a 20% mortality rate. Thus, the choice between surgical and endovascular treatment methods depends on the clinical situation, patient's condition, and efficacy of previous interventions [26, 29].

O. Abbasoglu et al. reported 35 cases of revision involving aortohepatic anastomosis using the iliac artery or saphenous vein after the resection of the stenotic segment. In all patients, the HA blood flow was

successfully restored. At a mean follow-up of 25 months, 67% of patients were free of complications and the liver function remained normal [25, 49].

In another study, six patients with HAS were treated with PTLAP. Five of them remained asymptomatic at a mean follow-up of 25 months [26, 38]. This confirms that HAS at advanced stages complicated by the failure of endovascular methods may be an indication for the re-examination, arterial reconstruction, or retransplantation [22].

Clinical manifestations of HAS vary from the normal graft function to the complete graft dysfunction with ischemia or necrosis. In most patients, HAS is asymptomatic and is detected by routine ultrasound. The main clinical sign, as reported by O. Abbasoglu et al., is an increase in the level of liver enzymes [25, 39].

Given the non-specific clinical presentation, a regular ultrasound examination is especially important for an early detection of HAS in asymptomatic patients. Screening is necessary when the results of liver function tests change, since a timely diagnosis of RAS improves the patient survival and graft integrity [27, 49].

Treatment and prevention of hepatic artery thrombosis after liver transplantation

To date, the choice of the most effective method remains a matter of debate and depends on the time of diagnosis. Early diagnosis, timely revascularization or retransplantation are considered key measures to save patients with early HAT. In most clinics, acute HAT diagnosed within the first 5 days after transplantation is treated surgically [17, 39].

The surgical treatment of early HAT includes methods such as thrombectomy, HA reanastomosis, or the anastomosis revision with plastic surgery. The simplest option for surgical revascularization is thrombectomy with a Fogarty catheter followed by HA reconstruction [7,

25]. However, with excessive tension of the artery, the vascular spasm and damage to the intima may develop, which may make the artery unsuitable for anastomosis. In such cases, extraanatomical reconstructions are used with the involvement of the gastroduodenal, gastric, splenic arteries or autovenous inserts [4, 16].

Although the surgical revascularization allows the graft preservation and does not reduce survival rates, biliary complications are observed in 50% of cases [19]. Historically, retransplantation has been considered the most reliable method, providing the best clinical results. However, the shortage of donor organs and the serious condition of the patient may limit this option [3, 8].

If thrombosis affects intrahepatic vessels, the surgical revascularization is often ineffective. In such cases, endovascular methods are increasingly used: intra-arterial thrombolysis (IATL), PTLAP, and stent placement. These methods allow the blood flow to be restored and in some cases achieve encouraging results.

As W.E. Saad et al. pointed out, the optimal time for performing IATL is from 1–3 weeks to 1–3 months after transplantation [22, 43]. In some cases, for example, when the surgical intervention is impossible, IATL can be performed during the first week, although this increases the risk of complications [27, 12]. The mean interval between transplantation and thrombolysis is 53 days, which ensures successful and safe performance of the procedure [7, 40].

Despite the advances in endovascular interventions, their widespread use is limited by the risk of complications such as bleeding, which occurs in 20% of patients. Intra-abdominal bleeding can rarely be fatal. However, a high concentration of thrombolytics near the thrombus minimizes systemic side effects [28, 33].

The main complications of thrombolysis include rethrombosis, vascular wall damage, and arterial ruptures. In such cases, a surgical intervention or retransplantation is required. Endovascular methods are more often used in asymptomatic patients to avoid more invasive procedures [23, 27].

Some patients with late HAT survive owing to the collateral circulation development distal to the occlusion, which allows avoiding revascularization or retransplantation. However, such cases are rare [17, 24].

The liver neovascularization is more often observed in late HAT, HAS development, or thrombosis at the level of the biliodigestive anastomosis. Thrombosis-induced liver hypoxia induces angiogenesis via vascular endothelial growth factors (VEGF) and hypoxia-inducible factor 1-α (HIF-1α), which promote the formation of new vessels [13].

Neovascularization of the omental, mesenteric, lumbar, renal, and iliac arteries has been reported [12, 13]. However, such angiogenesis is observed only in chronic or delayed ischemia.

Thrombolytic drugs such as urokinase, streptokinase, alteplase demonstrate relative efficacy and safety. However, there are still no standardized recommendations for the use of IATL, including optimal dosages and protocols [6, 11]. Some studies suggest combining thrombolysis with low doses of heparin, especially in the early postoperative period, despite the risk of bleeding.

PTLAP is often combined with BA or stent placement to treat anatomical defects such as kinks and anastomotic stenoses [23, 40]. These combined approaches show better results compared to using a single method.

Retransplantation remains the treatment of choice when revascularization or thrombolysis fails, particularly in patients with early HAT. The study by J.P. Duffy et al. showed that of 203 HAT patients,

retransplantation was performed in 153 (75%), which provided better clinical results [8, 38].

Surgical revascularization, endovascular interventions, and retransplantation have their own advantages and limitations. Despite the success of thrombolytic therapy, there is no single standard for the HAT treatment. Further research is needed to develop optimal protocols [8, 42].

Incidence of arterial complications in liver transplantation and their impact on survival

The incidence of HAT has a significant impact on the graft and patient survival. According to M.A. Silva et al., mortality after liver transplantation in the presence of HAT development reaches 23% [21, 31]. In a meta-analysis by J. Bekker et al., early HAT was reported as the main cause of a graft loss (53.1%) and mortality (33.3%) [15]. The patient survival after revascularization was 40% among symptomatic patients, compared to 82% in asymptomatic patients [8, 16].

Due to the shortage of donor organs and high mortality associated with retransplantation, the endovascular therapy has become the treatment of choice in a number of cases. However, in the early postoperative period, patients with severe graft dysfunction most often require retransplantation. Retransplantation rates range from 25% to 83% in patients who have not undergone revascularization and from 28 to 35% in those who have undergone this procedure [16].

HAS is a lumen narrowing of the liver graft reconstructed arteries (in most cases, the reconstructed hepatic artery), which leads to graft ischemia and is usually accompanied by changes in liver biochemical parameters [8]. In most cases, HAS is caused by technical errors leading to the vessel intima damage, necrosis, and scar formation. HAS reduces the blood flow and increases the risk of the HAT development.

Angioplasty performed in the early postoperative period may result in a suture rupture or intimal damage, which may have catastrophic consequences [17].

Risk factors for HAS remain poorly understood, but they may be associated with technical peculiarities of making the anastomosis (rough handling of vessels, clamps, damage to the intima), anatomical features of the donor and recipient (length of the reconstructed HA, kinks, angles of inclination, difference in vessel diameter), external compression or damage to the "vasa vasorum", acute cellular rejection of the graft [25, 33].

Clinically significant HAS is defined as a stenosis of the arterial lumen of more than 50% accompanied by a resistance index of less than 0.5 and a peak systolic velocity greater than 400 cm/s at ultrasound examination [3, 17]. If HAS is not treated, it progresses HAT within six months in 65% of cases. HAS and HAT are two interrelated components of graft ischemia [3, 8].

The incidence of biliary tract complications with HAS is significantly lower than with HAT, but can reach 67% [26]. E. Volpin et al. reported cases where untreated HA anastomotic stenoses progressed to HAT [30].

According to O. Abbasoglu et al., the HAS incidence was 4.8% in a cohort of 857 patients who underwent liver transplantation from 1988 to 1995 [25]. The mean time to diagnosis was 100 days (range 1–1220 days); that was supported by the data obtained by A.L. Denys et al., where the mean time to diagnosis was 94 days [27, 38].

HAS is classified into early HAS (diagnosed within 30 days after transplantation), and late HAS (diagnosed 30 days after transplantation).

J. Chen et al. reported that the HAS overall incidence was 2.8%, with early HAS occurring in 40% of cases and late HAS in 60%. The mean time between transplantation and diagnosis was 91 days [39].

Anastomotic stenosis most often develops within the first three months after transplantation and is the most common site of stenosis formation [22].

Delayed complications and efficacy of percutaneous transluminal angioplasty

Delayed complications of HAS after PTLAP, including recurrent stenosis, occur in 5% of cases within 30 days after the procedure. The rate of recurrent stenosis reaches 75%, indicating the need for long-term monitoring [25].

A.L. Denys et al. reported a low incidence of HAT among 13 patients who had a stent placed in the HA. These patients were receiving anticoagulant or antiplatelet therapy. Among them, there was one case of HAT and four cases of in-stent restenosis, which were successfully treated [27].

The best time to perform the endovascular intervention after liver transplantation remains a matter of debate. However, the method provides an acceptable benefit-risk ratio, allowing the intervention to be performed at any stage of the postoperative period [6, 19].

N. Rostambeigi et al. noted that PTLAP, both alone and in combination with a stent placement, demonstrates a high efficacy, reducing the retransplantation rates and preserving the graft [29].

Prognosis in hepatic artery stenosis after liver transplantation

O. Abbasoglu et al. reported that the 20% overall mortality of patients with HAS, predominantly in the surgical group [25]. Retransplantation was performed in 19% of patients, with 5 of them having a chronic rejection that was not diagnosed before the HA revision, suggesting that HAS may be an early sign of a chronic rejection [26, 42].

Screening for the chronic rejection is recommended when diagnosing HAS. The patient and graft survival rates after 4 years were 65% and 56%, respectively, which did not differ from the comparison group [26].

Aneurysm of the reconstructed hepatic artery

The reconstructed hepatic artery aneurysm (RHAA) is a HA dilation associated with damage to the arterial wall, which most often occurs as a result of iatrogenic injuries. The RHAA incidence after liver transplantation varies from 0.27 to 3% [27, 39].

Most aneurysms are located extrahepatically and their development is observed mainly in the first 35 days after transplantation. The average time of RHAA manifestation is 13 days [20, 49].

Risk factors for the RHAA development include technical errors while making the anastomosis, damage to the "vasa vasorum", an excessive length of the HA with kinks, acute cellular rejection [30, 44].

The incidence of RHAA infection reaches 81%. Early recognition and treatment allow achieving successful results in 100% of cases [30, 45].

Treatment of the reconstructed hepatic artery aneurysm

RHAA treatment can be performed using surgical methods or interventional radiology. In a study by E. Volpin et al. [30], the emergency laparotomy and ligation of HA were performed in 5 patients. Three of them died in the immediate postoperative period, and 2 survivors developed biliary complications. In 5 patients, the HA was restored, including with cryopreserved allografts in 2. Among all patients in this subgroup, biliary complications were seen in 3 cases. The overall mortality rate was 28%, but no complications occurred among 66% of successfully

treated patients, demonstrating promising results for emergency surgery [30, 43].

Two patients in this study were treated with interventional radiology. One of them underwent a coil embolization followed by the HA occlusion. After 10.5 years of follow-up, the patient maintained a good liver function without biliary complications. In the second patient, the aneurysm was occluded with a stent; the liver function also remained stable after 10 years [29].

In some cases, the RHAA treatment after liver transplantation has been performed using N-butyl-2-cyanoacrylate (Nbca) embolization. This method has been successful owing to the formation of a collateral vascular network around the graft and bile duct. Nbca is most commonly used to embolize aneurysms, arteriovenous malformations, and venous varices. Nbca is mixed with lipiodol, and when released into the bloodstream, the reagent polymerizes, effectively cutting off the blood supply [30].

The advantage of using Nbca over coil embolization is the ability to be used in tortuous vessels, as well as the minimal risk of recanalization after the injection [30].

Prognosis for the reconstructed hepatic artery aneurysm after liver transplantation

The HAA rupture is accompanied by massive hemorrhage, which leads to the disruption of the arterial blood supply to the graft, its loss, and high mortality. This condition is one of the most severe complications and requires an emergency surgical intervention.

The role of the infectious factor in the RHAA development has been repeatedly emphasized in the literature. In half of the cases, the infection remains unrecognized until complications develop, but it always requires an immediate intervention [6, 46].

Discussion

Analyzing the literature data, it becomes obvious that the prevention and timely diagnosis of ACs after liver transplantation are key aspects in ensuring successful clinical outcomes. In the context of a rapid increase in the number of transplants, the expanded indications for surgery, and improved availability of this procedure, ACs are becoming a major problem requiring constant attention from specialists.

One of the critical factors in preventing complications is taking into account the individual anatomical features of the liver vascular bed at the stage of preoperative planning. The HA variation anatomy, as shown in numerous studies, significantly increases the risk of thrombosis, stenosis and other complications. Thus, the choice of reconstruction technique should be based on an accurate preoperative assessment, which will minimize the likelihood of ACs.

The improvement of diagnostic methods is also important. An early detection of complications using ultrasound, contrast-enhanced CT, and angiography allows a timely treatment, preventing serious consequences. Protocols for monitoring the condition of the graft vessels should be standardized and include regular postoperative examinations, especially in high-risk patients.

The treatment of already developed ACs is a complex task requiring an individual approach. Modern treatment methods, such as RHAA, the stent placement and selective thrombolysis, demonstrate high efficiency in a number of cases. However, the success of these methods depends on the timeliness of their application and the competence of medical personnel. Surgical revascularization and retransplantation remain reserve methods that are used when endovascular interventions are ineffective.

An important area is the development of technologies aimed at minimizing postoperative complications. The use of microvascular surgery, the use of new materials for vascular conduits, the improvement of thrombolysis methods and the development of angioplasty protocols can significantly improve treatment results. In addition, the use of innovative thrombolytic drugs in combination with anticoagulant therapy opens up additional opportunities to reduce the risk of thrombosis.

The problem of ACs after liver transplantation is becoming increasingly important due to the increase in the number of surgeries and their increasing accessibility. In this context, it is extremely important both to develop new treatment methods, and also to focus on improving preventive strategies. Training of specialists, standardization of diagnostic and treatment protocols, implementation of modern technologies and a personalized approach to each patient play a key role in improving the long-term outcomes of liver transplantation.

Thus, the fight against ACs requires a multidisciplinary approach, including the efforts of surgeons, radiologists, anesthesiologists, and intensive care specialists. Only through joint efforts can optimal clinical outcomes be achieved, ensuring the graft preservation and improving the quality of life of patients.

Conclusion

Arterial complications after orthotopic liver transplantation, despite their relatively rare incidence, represent one of the most serious threats to the patient and graft survival rates. They threaten the critical disruption of the blood supply to the graft, which can lead to both liver parenchyma ischemia and severe biliary complications. The high risk of graft loss and death makes this problem one of the key ones in transplant medicine.

Diagnosis and treatment of such complications remain a serious clinical challenge. Particularly challenging is the need for timely detection of early signs of complications, since the delay can significantly worsen the prognosis. Ultrasound Dopplerography, contrast multidetector computed tomography with, and angiography are the main tools for early diagnosis of vascular disorders. However, these methods require standardization of application protocols, which will improve the accuracy and efficiency of detecting complications.

Treatment of arterial complications requires an individual approach, taking into account the anatomical features of the patient and the technical feasibility of performing endovascular or surgical interventions. Modern methods, such as percutaneous transluminal angioplasty, stent placement and intra-arterial thrombolysis significantly expand the possibilities of minimally invasive restoration of blood flow. However, despite their promise, these methods require highly qualified specialists and the availability of appropriate equipment. Surgical revascularization and retransplantation remain extreme measures, but play an important role in saving patients when less invasive approaches fail.

An equally important task is the prevention of arterial complications. The key factor in this direction is a thorough preoperative assessment of the vascular bed anatomy, which allows choosing the optimal reconstruction strategy. In addition, the improvement of surgical techniques, the use of microsurgical methods, and the introduction of modern materials for vascular conduits significantly reduce the risk of complications.

Arterial complications after liver transplantation require a multidisciplinary approach, including the efforts of surgeons, radiologists, anesthesiologists, hepatologists and intensive care specialists. The joint

work of these specialists allows for timely diagnosis, effective treatment and, ultimately, increased patient survival.

Thus, arterial complications remain the central challenge of current transplant medicine, defining the limits of the possibilities of surgery and postoperative patient management. The fight for arterial anastomosis is not just another technical task, it is a fundamental issue, where the triumph of science, skill and innovation is the saved life of a patient and his long, full life.

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