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VASCULAR COMPLICATIONS AFTER LIVER TRANSPLANTATION: CONTEMPORARY APPROACHES TO DETECTION AND TREATMENT. A LITERATURE REVIEW

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Vascular complications (VCs) after liver transplantation (LT) are rare but are one of the most dreaded conditions that can potentially lead to graft loss and recipient death. This paper has analyzed the international experience in the early diagnosis of various VCs that can develop following LT, as well as the optimal timing and methods of treatment of these complications.

Keywords: liver transplant, vascular complications.

INTRODUCTION

LT is a very complex and comprehensive method of treating end-stage liver disease, and it has proven to be the only method that can significantly prolong the life of incurable patients [1]. However, this surgical procedure is associated with significant risks, such as VCs [2].

The overall incidence of VCs varies in different world centers, with a cumulative incidence of about 7% in orthotopic LT from deceased donors and about 13% in liver fragment transplantation from living donors in both adult and pediatric cohorts of recipients [3–7].

Since VCs carry the greatest threat of graft loss, their diagnosis and treatment are a serious aspect in terms of graft and recipient survival. This explains why many transplant teams nowadays closely monitor all vascular anastomoses using Doppler ultrasonography for early detection and treatment of these complications before the liver graft is irretrievably lost [8–10].

Indeed, VCs can suddenly interrupt blood supply to the liver with a high probability of graft loss.

Generally, regardless of the type of complication, treatment measures include:
– conservative therapy;
– endovascular plasty/stenting/thrombolysis;
– percutaneous transhepatic therapies;
– open surgical recanalization;
– retransplantation.

Although open surgery has been considered the primary choice for graft revascularization, advances in endovascular surgery have enabled less invasive and very effective revascularization. In recent decades, huge advances in interventional radiology have radically changed diagnostic and therapeutic approaches to the management of patients with post-LT vascular complications [1, 5, 11–18].

In fact, percutaneous endovascular interventions (i.e. thrombolytic procedures, balloon angioplasty and stenting) performed by experienced endovascular surgeons have become increasingly used and are gradually replacing open surgery, becoming the surgical method of choice in the treatment of VCs following liver transplantation [18–20].

Further, during the narrative, we will dwell on each VC in detail and consider optimal methods of detecting the complication and its treatment based on data from world literature sources.

1. HYPERCOAGULABLE STATES IN PATIENTS WITH CIRRHOSIS

Recent studies have shown that bleeding is not the only risk in LT for cirrhosis of various etiologies. Several risk factors must be considered in the setting of liver surgery, such as vascular constriction, veno-venous bypass, presence of central venous catheters, use of antifibrinolytic drugs, tissue ischemic injury, venous stasis, etiology of liver disease, endothelial damage, and ischemia time. All these factors may increase the likelihood of thrombotic complications [21–23].

The end stage of liver disease is itself a risk factor for thrombosis. In liver disease and liver surgery, conventional coagulation tests often fail or provide incorrect information about the status of hemostasis. For example, prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR) are only indicative of procoagulant factors and are insensitive to plasma levels of anticoagulant factors. So, these tests are not always reliable for describing the hemostatic status of patients with terminal liver disease.

A marked decrease in both procoagulant factors (factors II, V, VII, IX, X, XI, XII) and anticoagulant factors

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(antithrombin III, protein C, and protein S), an increase in von Willebrand factor (vWF), and ADAMTS13, a protease that cleaves on vWF, are characteristic features of the course of cirrhosis and lead the patient to a new hemostatic balance [11]. vWF fulfills its hemostatic functions by binding to factor VIII and connective tissue components, and also promotes platelet adhesion to endothelial surfaces and platelet aggregation [24].

Thrombocytopenia resulting from hypersplenism in patients with portal hypertension, abnormal thrombopoietin metabolism, increased platelet destruction mediated by antiaggregant antibodies, and alcohol-induced bone marrow suppression, antiviral and immunosuppressive therapy, is another condition that develops in cirrhosis [25]. Unless the platelet count is very low ($<50 \times 10^9$/L), thrombocytopenia does not pose an increased risk of intraoperative bleeding. Such a platelet count is usually sufficient to guarantee normal thrombin formation, and the low platelet count is compensated for by a higher level of vWF, which is responsible for platelet adhesion [26, 27]. Hyperfibrinolysis is another described sign of end-stage liver disease, but its role in the coagulopathy of cirrhosis is still debated [28]. Elevated levels of tissue plasminogen activator and deficiency of thrombin-activated fibrinolysis inhibitor have been associated with laboratory changes that are typical of hyperfibrinolysis and increased risk of bleeding [29]. However, liver cirrhosis is also associated with decreased fibrinolysis, as evidenced by decreased plasminogen levels and increased plasminogen activator inhibitor levels. These contrasting results explain the ongoing debate regarding the absence or presence of a hyperfibrinolytic state in patients with liver disease, even though the balance of fibrinolysis is probably restored by parallel changes in profibrinolytic and antifibrinolytic factors (see Table 1) [30].

The above factors affecting the imbalance of the coagulation and anticoagulation systems may have a direct impact on the risks of post-liver transplant VCs. 95% CI:

2. ARTERIAL COMPLICATIONS

Arterial complications remain the most formidable and leading causes of morbidity and mortality after orthotopic LT [20, 34, 173]. Generally, the liver graft is supplied by the portal vein, hepatic artery (or several hepatic arteries). The hepatic artery (HA) plays an extremely important role as it supplies blood to both the liver parenchyma and the biliary tree. Absence of or reduction in arterial blood flow often leads to biliary complications due to ischemic processes, with the formation of biliary necrosis, liver abscesses, which leads to graft dysfunction, septic complications and graft loss, leading recipient death [20, 34, 174]. That is why extremely rapid detection of this problem and early treatment are very important.

The main arterial complications following liver transplantation are:

- HA thrombosis (1.9%–16.6% incidence rate) [10];
- stricture of the arterial anastomosis (0.8–9.3%) [175];
- splenic artery steal syndrome (≤10.1%) [26–31];
- HA pseudoaneurysm (0.1%–3%) [18];
- HA rupture (arterial bleeding, incidence <1%) [176].

Based on timing, these complications are divided into early (complications occurring in the first month after LT), and late complications (complications developing after one month following LT).

Special attention should be paid to early complications because they are associated with graft loss and high mortality. The timing of early and late complications continues to be debated in different studies. Most authors have defined late complications as those that occurred within the first four weeks of transplantation and others within the first six months [13, 32–34]. However, according to the most recent international consensus, early complications are defined as those that occur within the first month after LT [8, 13, 18, 33–34].

2.1. Hepatic artery thrombosis

Transplant hepatic artery thrombosis (HAT) occurs when a blood clot forms in the HA that provides blood flow to the liver. According to the classification of VCs described above, there are early and late HAT [8, 13, 18, 32–34].

HAT is the most common (about 50% of all VCs) and the most severe arterial complication that can develop

Table 1

Balance of antihemostatic and prohemostatic factors in cirrhosis

	Antihemostatic factors	Prohemostatic factors
Primary hemostasis	Platelet dysfunction	Increased von Willebrand factor
	Thrombocytopenia	Decreased ADAMTS 13
	Reduced thrombopoietin synthesis	Platelet reactivation
Coagulation	Reduced synthesis of factors II, V, VII, IX, X, XI	Increased factor VIII
	Vitamin K deficiency	Decreased anticoagulant protein C, protein S, and antithrombin III
	Hypodysfibrinogenemia	Procoagulant changes in fibrin structure
Fibrinolysis	Low levels of alpha 2-antiplasmin and factor XIII, decreased thrombin activatable fibrinolysis inhibitor	Low plasminogen
		Increased plasminogen activator inhibitor 1

after LT. This complication is one of the main causes of primary graft dysfunction, which can lead to graft loss and patient death in the early postoperative period [37]. Arterial thrombosis is more common in younger recipients [7, 8, 16, 17, 34–37].

If not diagnosed in time, the chances of graft loss are extremely high. The only method of treatment in this situation is liver re-transplantation. Indeed, the frequency of re-transplantation is very high in case of late graft revascularization and, according to reports, it is 25%–83%. Methods of early revascularization with the help of endovascular intervention have been actively introduced in recent years [5, 8, 13, 16, 17, 36, 38–45].

The true incidence of early HAT is unknown, but it varies widely (0% to 12%) [7, 32, 36, 43, 46, 47, 175]. Becker et al. in 2009 reported that based on an analysis of 21,822 patients who underwent orthotopic LT, there were 843 cases (3.9%, adults and children) of early HAT [34]. Also, this analysis showed that the number of VCs decreased slightly as new surgical techniques emerged year after year. Among other things, this report shows that the number of HAT cases remains approximately the same worldwide, regardless of the clinics. The mean time to detection (development) of HAT ranged from 1 to 18 days from the time of transplantation. Late complications occurred on average six months after LT [34].

There is no clear evidence in the literature on whether the incidence of HAT depends on whether the transplantation was performed from a deceased donor or from a living donor. Many studies show conflicting results. A meta-analysis of data from several major transplant centers found no significant difference in the development of thrombosis (3.1% in related transplants, 4.6% in liver transplants from a postmortem donor) [3, 17, 34, 47, 60, 175]. In addition, it has been reported that when surgical microscopes were used during arterial anastomosis, the incidence of HAT remained the same [17, 34, 47, 102, 175].

Risk factors

There are several factors that increase the risk of developing HAT.

The risk factors for early HAT include surgical problems, namely [7, 8, 17, 19, 32–34, 40, 48, 49]:
- difficulties with arterial reconstruction;
- small HA diameter;
- large HA tortuosity;
- arterial dissection to create a site for arterial anastomosis;
- multiple arteries feeding the graft;
- arterial anomalies requiring complex arterial reconstructions, including the use of vascular grafts;
- poor quality of donor/recipient vessels.

It has been shown that the more experienced the operating team is, the lower the risk of early HAT. So, surgical causes are probably not a major risk factor for early HAT [17, 34, 35, 37, 43]. Also, minimally invasive techniques in related liver donation have been developed recently. For example, laparoscopy-assisted liver graft harvesting is widely used in related liver transplantation. Evidence has been analyzed, showing that the graft harvesting technique does not affect the incidence of early arterial complications [50–53].

Also, the transarterial chemoembolization procedure in hepatocellular liver cancer can be attributed to risk factors. For instance, Panaro in 2014 showed that patients with a history of selective transarterial HA chemoembolization are prone to HA intima damage, which, in turn, may lead to HAT in the early postoperative period [54].

Factors influencing the development of late HAT include [33, 40, 49, 55]:
- cytomegalovirus infection;
- hepatitis C;
- female donors;
- male recipients;
- tobacco smoking;
- retransplantation.

Also, many authors believe that a hypercoagulable state may be the main cause of HAT [7, 9, 17, 35, 49, 55].

Clinical presentation

The clinical presentation of HAT ranges from mild elevation of cytolytic enzymes and bilirubin in peripheral blood serum to acute liver failure. Elevation of cytolytic enzymes (AST and ALT) occurs in 75% of patients with HAT; development of biliary complications on the background of HAT occurs in about 15% of cases. Fever and sepsis develop in 6% of HAT cases. Acute graft dysfunction or liver failure occurs in 4% of cases [7].

The severity of clinical manifestations depends on the time HAT develops, as well as on how developed the hepatic arterial collaterals are [7, 32, 33].

Biliary complications, such as bile duct strictures or bile leakage, sometimes leading to liver abscesses, are more often, but not exclusively, associated with late HAT, while early graft dysfunction (liver failure) is most often associated with early HAT [17, 33, 46].

Also, the severity of clinical manifestations depends on the presence of arterial collaterals, which can develop already within two weeks after LT. Therefore, the following are the two main forms of HAT [32]:
1. Early HAT characterized by a severe clinical course;
2. Late HAT, characterized by a milder clinical course.

In almost every case, early HAT is clinically manifested by fever, leukocytosis, and elevated liver enzymes [20, 34, 173]. Ischemia of bile ducts and hepatocytes with subsequent necrotization often develops, which leads to multiple liver abscesses, followed by uncontrolled septic shock (against the background of immunosuppressive therapy) and patient death [32–34, 37, 41, 43].

If HAT develops late after LT, clinical manifestations are usually associated with biliary complications [8, 19,

33, 41]. In 50% of cases, late HAT manifests asymptomatically, only a biochemical test can detect a slight increase in cytolysis markers. Subsequently, patients suffer from recurrent cholangitis, some of them develop bile duct strictures or bile leakage. Also, intrahepatic bile duct necrosis with formation of liver abscesses develops. As a rule, late HAT symptoms and signs are insidious and require special attention by physicians [17, 33, 34, 41, 43, 56].

Diagnosis of hepatic artery thrombosis

Early diagnosis of HAT is critical because of the high risk of graft loss. Diagnostic procedures include biochemical tests (increase in cytolysis enzymes) and Doppler flowmetry. If necessary, bolus contrast-enhanced multislice computed tomography (MSCT) is performed to assess blood flow through the arterial bed of the graft, or angiography is carried out [17, 173].

Ultrasound Doppler flowmetry is a non-invasive method and is the gold standard for diagnosis. Doppler flowmetry detects decreased arterial blood flow and increased resistive index (Ri). Ultrasound should be used as a screening method for early diagnosis of HAT and it should be performed at least once a day in all liver transplant patients [17, 32, 43]. That said, in some transplant centers, routine ultrasound examination is performed every 6 hours after transplantation for 7 days (acute period) for early detection of occlusion and initiation of immediate treatment [57, 173]. The protocol used in the Russian Federation and Central Asia is presented in Fig. 1. This protocol is also used in the pediatric liver transplantation program [173].

If the patient has elevated hepatic enzymes (ALT and AST) and changes in HA indices, it makes sense to perform bolus contrast-enhanced MSCT and/or selective angiography of the celiac trunk (celiacography) [43, 173].

Treatment of hepatic artery thrombosis

There are several classic treatments for HAT:

– administration of anticoagulants/antiplatelet agents and dynamic monitoring;
– revascularization (surgical or endovascular);
– retransplantation;

At present, the most effective treatment approach remains contentious, and the choice of any of these treatments depends on the timing of diagnosis. Early diag-

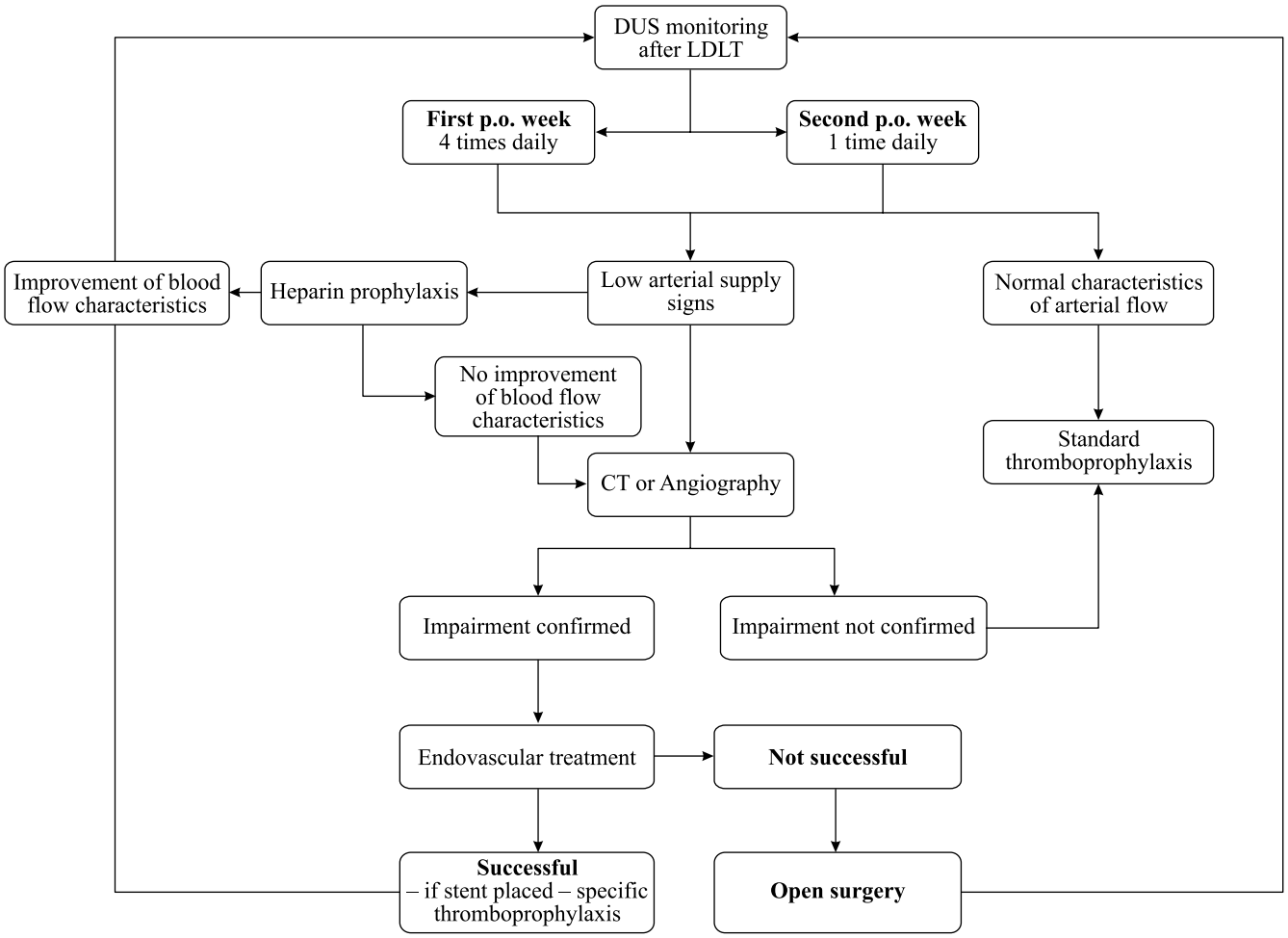

Fig. 1. Ultrasound monitoring protocol for screening of vascular complications after liver transplantation and plan of action in case of complications detection [57]

nosis, conservative therapy, surgical revascularization or retransplantation are considered the only solution to save patients with HAT [2, 8, 10, 17, 20, 173].

Some patients receive anticoagulant/antiplatelet, thrombolytic therapy for the treatment/prevention of thrombosis. Acetylsalicylic acid, clopidogrel, rivaroxaban, apixaban, urokinase, streptokinase, alteplase, calcium nadroparin, and heparin are used [17, 20, 58]. A reliably better protocol is not yet known, and there are currently no specific guidelines for the use of thrombolytic therapy in these patients. Nevertheless, when thrombosis is reliably detected, thrombolytic therapy is actively used in many surgical centers despite the high risk of postoperative bleeding [2, 17, 20, 57–59]. Indeed, bleeding is the most frequent side effect of thrombolytic therapy and occurs in approximately 20% of patients. Bleeding manifests itself in a variety of ways – from a mild hemorrhagic discharge through safety drains to intra-abdominal hemorrhage, which can be fatal in some cases. Bleeding on the background of thrombolytic therapy typically occurs in the early postoperative period [16, 20].

Thrombosis prevention protocols at different transplant centers are presented in Table 2.

There is selective endovascular thrombolysis (drugs are injected directly into the HA). This method offers several advantages, such as lower thrombolytic dose, high local concentration of drugs and relatively small effect on systemic coagulation (Fig. 2) [11, 20, 162].

As reported from various sources, combined therapy has a good effect: endovascular balloon correction, blood flow correction (with or without artery stenting), together with the administration of anticoagulants/antiplatelet agents.

In some centers, early after transplantation, permanent heparinization under aPTT control is used if arterial thrombosis is suspected based on ultrasound diagnostics. The authors note that if this method is ineffective, it

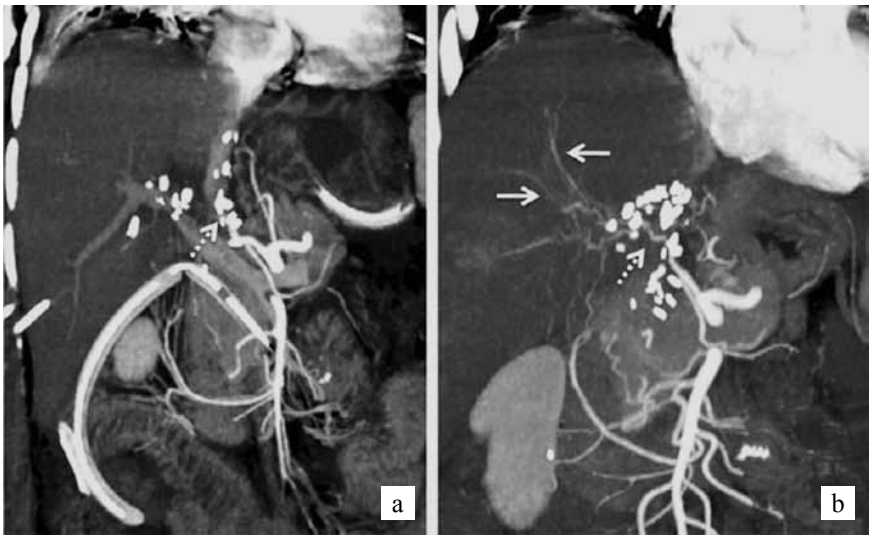

Fig. 2. Frontal projection when performing celiacography. A, The dotted arrow indicates the site of hepatic artery thrombosis after liver transplantation. B, The white arrows indicate the presence of arterial blood supply to the graft after thrombolytic therapy [162]

Table 2

Comparison of thromboprophylaxis protocols among transplant centers

Study	Thromboprophylaxis protocol	Number of cases	Vascular complications	Bleeding
Gautier, Monakhov et al. 2021 [173]	Prostaglandin E1, intraoperatively for 7 days; Enoxaparin, on day 1 after surgery in the absence of thrombocytopenia $<70 \times 10^9$/L within 14 days; Acetylsalicylic acid, with the start of oral nutrition or on postoperative day 4 for 3 months. If thrombosis/stenosis of afferent vessels is suspected, heparin is administered, targeting an aPTT of 60–80 seconds	416 patients, children, transplantation of various liver fragments from a living related donor and split liver transplantation	Arterial thrombosis (17; 4%) Portal vein thrombosis (no information)	No information

End of table 2

Study	Thromboprophylaxis protocol	Number of cases	Vascular complications	Bleeding
Blasi et al. 2016 [168]	Enoxaparin or not routinely administered unless the patient has had an intraoperative thrombectomy or the patient was on anticoagulant treatment prior to liver transplantation. No thromboprophylaxis if platelet count is below 30×10^9/L	328 patients, adults, cadaveric liver transplantation	Portal vein thrombosis (8; 2.4%) Arterial thrombosis/ stenosis (no information)	No information
Kaneko et al. 2005 [169]	Administration of dalteparin, target activated coagulation time (ACT) is 130–160 seconds	128 patients, adults, right lobe liver transplantation from a living donor	Arterial thrombosis (2; 1.5%) Portal vein thrombosis (1; 0.78%) Arterial thrombosis + portal vein thrombosis (1; 0.78%)	11 (8.5%) surgical revisions and 8 (6.25%) patients with hemorrhagic complications were treated conservatively
Gad et al. 2016 [170]	Heparin infusion up to 180–200 U/kg/day, adjusted depending on ACT (target levels, 180–200 seconds) and/or APTT (target levels, 50–70 seconds)	186 patients, transplantation of various liver fragments from a living donor	Arterial thrombosis (4; 1.8%) Portal vein thrombosis (5; 2.3%) Arterial thrombosis + portal vein thrombosis (4; 1.8%)	4 (1.8%)
Semash et al. 2023 [57]	Prostaglandin E1, intraoperatively for 5 days; Enoxaparin, on postoperative day 1 in the absence of thrombocytopenia $<50 \times 10^9$/L for 14 days; Acetylsalicylic acid, with the start of oral nutrition or on postoperative day 4 for 3 months. If thrombosis/stenosis of afferent vessels is suspected, heparin is administered, targeting an aPTT of 60–80 seconds	30 patients, adults, right lobe liver transplantation from a living donor	Arterial thrombosis (0) Portal vein thrombosis (no information	No information
Sugawara et al. 2002 [171]	Enoxaparin, Prostaglandin E1 (0.01 g/kg/hour) immediately after transplantation, administration of protease inhibitors	172 patients, adults, right lobe liver transplantation from a living donor	Arterial thrombosis (7; 4%) Portal vein thrombosis (4; 2.3%)	No information
Mori et al. 2017 [172]	Heparin infusion at a dose of 5 U/kg/h during the first week after liver transplantation	282 patients, adults, right lobe liver transplantation from living donor, 48 patients with portal vein thrombosis	Arterial thrombosis/ stenosis (no information) Portal vein thrombosis (8; 17%)	No information
Yip et al. 2016 [183]	Heparin injection, 5000 units subcutaneously every 8 hours	999 patients, adults, cadaveric liver transplantation	No information	No information
Vivarelli et al. 2007 [184]	100 mg aspirin orally	838 patients, adults, cadaveric liver transplantation (236 received thromboprophylaxis and 592 did not receive thromboprophylaxis)	Arterial thrombosis (1; 0.4%) in the thromboprophylaxis group and 13 (2.2%) in the comparison group. Portal vein thrombosis (no information	0%
Uchikawa et al. 2009[185]	Continuous infusion of dalteparin administered in the non-hepatic phase to maintain ACT at 140 to 150 seconds (Group A) versus continuous intravenous infusion of dalteparin administered immediately after surgery and adjusted according to clinical data (Group B)	42 patients, adults, cadaveric liver transplantation. Group A, 10 patients; Group B, 32 patients	Arterial thrombosis: 5 (15.6%) in group A, 0 in group B. Portal vein thrombosis: 5 (15.6%) in group A, 0 in group B	1 (3.1%) in group A and 0% in group B

is necessary to perform emergency revascularization. Moreover, if the thrombosis was successfully resolved against the background of permanent heparinization, which was confirmed by ultrasound and/or contrast-enhanced MSCT, revascularization was not performed, these patients were subsequently prescribed a prophylactic course of antiplatelet drugs [57, 173].

Historically, retransplantation in patients with post-LT occlusive HAT has been shown to have the best patient survival outcomes [7, 16]. On the other hand, percutaneous liver biopsy techniques for blood flow correction have been strongly developed recently and show decent outcomes. Currently, balloon angioplasty and/or stenting of graft artery, with subsequent administration of antiplatelet and anticoagulant therapy, are being actively performed. According to evidence from recent studies, good graft survival rate was achieved using the above-described method [2, 59, 173].

There are also cases when patients do not undergo any intervention, and the graft survives due to the arterial collaterals developing in it. The percentage of such cases is extremely small [17, 20, 43, 44, 46]. However, open surgical revascularization or retransplantation may also be ineffective. Despite the encouraging outcomes of endovascular interventions, these treatments also have a downside, as complications may occur during endovascular procedures. Moreover, after failed attempts at endovascular revascularization, either open interventions or retransplantation are required. Thus, urgent revascularization with endovascular interventions as a primary option may offer a chance to avoid retransplantation, but it is not successful in all cases [8, 10, 17, 20].

Below are the complications of endovascular correction of HA thrombosis:
– recurrent thrombosis;
– extravasation (minor damage to the artery);
– HA rupture with subsequent bleeding.

Any of these may require open revascularization or liver retransplantation [17, 20, 59, 173].

Open surgical revascularization for HAT is another type of treatment for graft salvage. Open surgical revascularization can be performed in different ways depending on the length and integrity of the artery. Fogarty catheter thrombectomy is used, and hepatic arterial anastomosis is transposed [16].

Meta-analysis of treatment methods has shown that liver retransplantation for early HAT shows the best patient survival compared to conservative therapy and revascularization in different variants. At the same time, some patients with late HAT survive without revascularization or retransplantation due to collateral circulation in the graft [7, 8].

Prognosis

With revascularization, patient survival in HAT is 40%. Survival reaches 85% when revascularization is performed with combined use of antiplatelet/anticoagulant/thrombolytic therapy. There have been different reports on an overall mortality reaching 23%–33% in patients with early HAT. The risk of graft loss in HAT according to some studies may be as high as 53.1%. The most effective prognosis of graft survival depends on the time of HAT detection and the speed of revascularization [17, 33, 34].

HAT develops quite rarely but represents the most common vascular complication after liver transplantation. A definitive diagnosis is established by angiography, during which therapeutic manipulations can also be performed using endovascular procedures, such as balloon angioplasty and/or arterial stenting.

Currently, it seems advisable to perform endovascular treatment first, mainly because of organ shortage and the high mortality rate associated with retransplantation. However, it has been proven that patients with early HAT with severe graft dysfunction require liver retransplantation.

2.2. Transplant hepatic artery stenosis

Transplant hepatic artery stenosis (HAS) is a narrowing of the lumen of a liver transplant artery leading to reduced arterial blood flow and partial ischemization of the graft. Significant HAS is a narrowing of the lumen of the graft artery by more than 50%. HAS along with HAT, are the most common arterial complications with high morbidity and mortality [4, 16, 36, 61, 63–66].

According to various reports, HAS develops in 2% to 13% after LT [4, 16, 36, 61–67]. There are cases where HAS is in turn complicated by thrombosis [4].

Similar to HAT, HAS is divided into early (developing within the first 30 days of LT) and late (developing after 30 days of LT).

Based on a meta-analysis, early HAS is statistically less common than late HAS (40% *vs.* 60%, respectively). The mean time to diagnosis of HAS is 94–160 days after LT (1–1220 days) [68].

The anastomotic portion of the liver graft artery has been shown to be the most common site for HAS within three months after LT [69].

Also, transplant HA kinking is considered to be a narrowing of the liver transplant artery [17]. In turn, arterial kinking can lead to HAT [7, 17].

Risk factors

The risk factors associated with HAS are not really known and seem to have a multifactorial origin [68]. The authors believe that technical factors such as arterial injury (during clamping, intima separation, improper anastomotic sutures), anatomical features of the donor and recipient arteries (excessive length, arterial kinking, diameter difference between donor and recipient arteries), impaired vascular blood supply to the artery, coagulation injury to the vessels, etc. may increase the risks

of arterial occlusive complications, including transplant hepatic artery stenosis, etc [66].

Clinical presentation

The clinical presentation of HAS varies from asymptomatic disease to graft dysfunction associated with ischemia and necrosis. Moreover, HAS can lead to graft dysfunction both early and late postoperatively. Many patients with asymptomatic course may demonstrate minor deviations from normal blood biochemistry (cytolysis, cholestasis) [16, 61, 66–68, 70, 71]. Mostly, in patients with asymptomatic HAS, the diagnosis is established incidentally, during Doppler ultrasound (DU) screening. That is why regular DU screening at early and late periods after LT is so important [57, 173].

The risk of biliary complications is less common in HAS than in HAT. Ideally, HAS should be diagnosed before biliary complications occur because, according to reports, they develop in 67% of HAS cases [70, 71].

Diagnosis of hepatic artery stenosis

Ultrasound is a well-established noninvasive and inexpensive method of assessing liver graft arterial patency; it has been reported in many studies to be effective in the early diagnosis of HAS [57, 61, 66, 173]. Velocity of blood flow through the artery is assessed in combination with resistive index (Ri) – in arterial stenosis, it decreases <0.5, delayed systole and "rounding" of the systolic peak occur. Sometimes, on the contrary, when peripheral resistance increases, Ri increases, the diagnostic criterion is Ri >0.85 [173]. Some authors describe turbulent blood flow through the HA – an increase in velocity >100 cm/sec (Fig. 3) [163]. Many transplant teams also use contrast-enhanced MSCT and direct angiography to confirm the diagnosis, which is the gold standard for diagnosing HAS [8, 72, 73].

Treatment of hepatic artery stenosis

As with HAT, treatment of HAS includes:

– revascularization (surgical or endovascular);
– retransplantation.

In patients with asymptomatic HAS, endovascular angioplasty (balloon vasodilatation and/or stenting of the graft artery) is performed [59, 62, 67, 71, 74, 173, 177]. A positive effect was achieved in 87% of patients [61]. In 7% of patients who underwent endovascular angioplasty, complications developed, including arterial rupture, requiring open revision. Some authors have noted the development of HAT after endovascular correction of arterial stenoses. This is typically associated with inadequate postoperative management of patients (inappropriately selected antiplatelet/anticoagulant therapy after arterial stenting) [61, 66]. According to a meta-analysis report, open revascularization with excision of the anastomotic narrowing, with the use of vascular grafts in some cases, showed a 100% restoration of blood flow through the HA [61].

Despite this, a case series meta-analysis published in 2015 showed that interventional techniques for correcting arterial blood flow are highly effective for early HAS, they do not differ in complication rates compared to open arterial reconstructions, and they help to reduce the number of liver retransplantations [75].

With timely early diagnosis of HAS by DU screening and regular laboratory monitoring of blood biochemical parameters (liver panel) and early revascularization by either method, the risk of graft loss and retransplantation is reduced significantly.

When endovascular intervention fails to restore blood flow through the HA, surgical revascularization should be undertaken before considering retransplantation, which has a lower postoperative survival rate, given that HAS is associated with subsequent biliary complications.

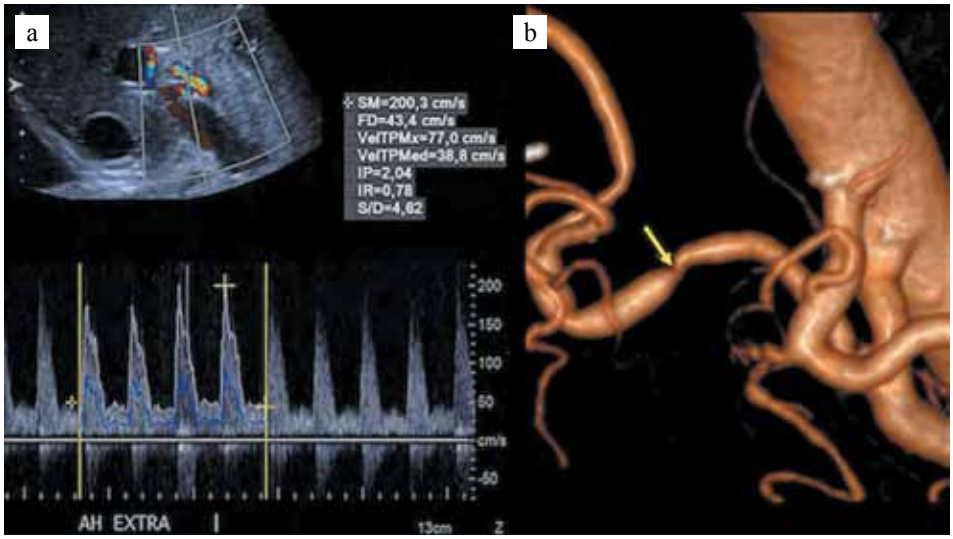

Fig. 3. Visualization of liver graft artery stenosis: a, triplex ultrasound of the liver graft, turbulent arterial blood flow in flowmetry; b, volume rendered image from a CT angiography. The arrow indicates the site of hepatic artery stenosis [163]

Carefully performed arterial anastomosis during transplantation appears to prevent arterial stenosis.

2.3. Splenic artery steal syndrome

Splenic artery steal syndrome (SASS) is another cause of graft hypoxia or ischemia. SASS can be described as a decrease in blood flow into the HA in the absence of HAT or HAS. This condition is associated with increased arterial inflow through the enlarged splenic artery, since the liver and spleen in most cases are supplied from the same basin. According to world reports, SASS remained without attention from surgeons for a long time because the actual surgical problem in the arterial anastomosis was not revealed. But it was shown that SASS can reliably lead to graft hypoxia/ischemia against the background of hepatic hypoperfusion and is a threatening complication, which, in turn, can lead to irreversible consequences, up to graft loss and patient death [57, 76–80].

Risk factors

A complex combination of factors, including HA hypoperfusion and portal hyperperfusion, can lead to SASS. The first and the main risk factor is portal hypertension, against which the volume of the spleen and vessels feeding it increases. Some authors cite the following pattern: a difference between splenic and HA diameters of 1.5 times or more in favor of the splenic artery is a risk factor for SASS. Some authors consider the splenic artery diameter of more than 5 mm as a risk factor regardless of the difference in the diameter of the liver and spleen vessels [57, 81, 177]. There are also works that determine an increased risk of SASS when the graft-to-recipient weight ratio (GRWR) is less than 0.9% [57, 82, 83].

Clinical presentation

Similar to HAS, the clinical presentation of SASS can be diverse. This condition is often asymptomatic, but hepatic failure, graft ischemia and necrosis may develop if diagnosis is delayed. Patients with asymptomatic SASS may show minor deviations from normal blood biochemistry parameters (cytolysis, increased bilirubin, alkaline phosphatase, and gamma-glutamyl transferase) [76–80, 84].

Diagnosis of splenic artery steal syndrome

Most often, diagnosis is made early after transplantation and is detected through routine use of ultrasonography. Ultrasound signs of SASS include difficulties in visualizing the HA at the anastomosis level and in the graft parenchyma, and decreased total blood flow velocity of less than 15 cm/sec, while blood flow indices may be normal. If SASS is suspected, contrast-enhanced MSCT or selective angiography of the celiac trunk (celiacography) should be performed [25]. During angiography, there will be increased discharge of contrast agent into the enlarged splenic artery, while the inflow into the liver graft artery will be reduced [57, 84, 173, 181].

Prevention of splenic artery steal syndrome

Currently, there are several SASS prevention techniques. According to numerous reports, endovascular methods of prophylaxis are used, for example, selective splenic artery embolization before LT [182]. At the stage of examining the recipient in preparation for transplantation, if signs of hypersplenism and enlarged splenic artery are detected, the above procedure is performed. According to world reports, this method reduces the risk of SASS [79]. However, the procedure is not always effective. Cases have been described where after splenic artery embolization, a powerful collateral blood flow developed in the spleen, and patients experienced SASS after transplantation [57].

Intraoperative splenic artery ligation is another described method for preventing SASS. During transplantation, the celiac trunk and its branches are skeletonized and the splenic artery is ligated. According to reports from studies describing this technique, there was not a single case of SASS developing after splenic artery ligation. At the same time, ischemic disorders in the spleen were practically not described and they were asymptomatic [57, 81, 178–180].

Treatment of splenic artery steal syndrome

The mainstay of treatment for SASS is currently endovascular selective embolization of the splenic artery (Fig. 4) [25, 57, 76–80, 83–85, 173, 181]. Embolization is performed either with emboli or coils. At the same time, cases have been described where, in the early stages after LT during angiography, it was technically impossible to perform cannulation of the splenic artery for embolization; in such cases, relaparotomy and open ligation of the splenic artery were performed to restore adequate arterial perfusion of the liver graft [57]. Also, there were cases where during embolization, the guidewire migrated into the HA and caused dissection of the arterial anastomosis, which made it necessary to perform open surgery to stop bleeding and open splenic artery ligation [57].

With early diagnosis of SASS using ultrasound methods and timely selective splenic artery embolization, the risk of graft loss and retransplantation is significantly reduced.

It is recommended to use prophylactic methods (splenic artery embolization before transplantation, splenic artery ligation during transplantation) to prevent the risks of SASS.

2.4. Hepatic artery pseudoaneurysm

Hepatic artery pseudoaneurysm is a formation resulting from a breach of the integrity of the arterial wall and

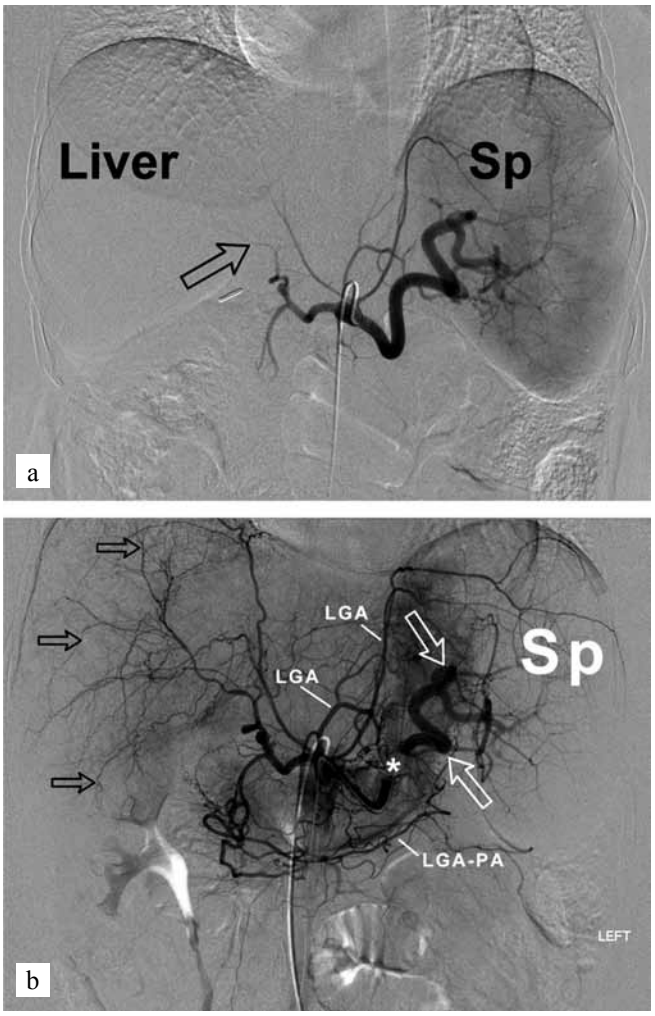

Fig. 4. Celiacography in splenic artery steal syndrome: a, no evidence of hepatic artery stenosis/thrombosis, with reduced blood flow to the liver (indicated by the black arrow), the splenic artery is enlarged in diameter; b, white asterisk marks the place of spiral placement in the splenic artery. Black arrows indicate active filling of the graft along the arterial channel after splenic artery embolization [177]

ongoing bleeding. The spilled blood accumulates in the tissues around the artery forming a tumor-like formation. As a rule, pseudoaneurysms in a liver graft are iatrogenic. Their incidence according to different data varies from 0.27% to 3% [36, 86–96, 176].

A retrospective meta-analysis by Volpin et al. on 787 liver transplants performed between January 1990 and December 31, 2005 reported an incidence of 2.5%, evenly distributed over a 16-year period. The authors showed that this complication did not significantly affect any specific laboratory findings in patients after LT [96]. In 16 patients, the anatomical location of the pseudoaneurysms was extrahepatic and developed early after liver transplantation. In fact, most pseudoaneurysms developed in the early postoperative period within an average of one month after transplantation: 69% were diagnosed within 20 days and 81% within 35 days after LT. The average time for the development of pseudoaneurysms was 13 days [36, 93, 96, 176].

Risk factors

Several predisposing factors for the development of HA pseudoaneurysms have been suggested, including peritoneal infections, technical difficulties in arterial anastomosis, and bile duct leaks [36, 97–101, 176]. In patients with extrahepatic localization of pseudoaneurysms, the rate of detection of bacteria and fungi in the culture of abdominal contents was very high: according to various literature sources from 81% to 100% bacterial or fungal growth was detected [96].

It has also been reported that some patients with bile duct leaks who underwent biliodigestive anastomosis during LT developed HA pseudoaneurysms [36, 96–101, 176].

Clinical presentation

The clinical presentation of pseudoaneurysms ranges from asymptomatic course and incidental diagnosis on DU, MSCT, or angiography to abdominal pain combined with fever, gastrointestinal bleeding (25% of cases), massive intra-abdominal bleeding in the early postoperative period (31% of cases), and acute hemorrhagic shock (81% of cases) [96].

Diagnosis of hepatic artery pseudoaneurysm

The diagnosis of pseudoaneurysm is established based on instrumental diagnostic methods (Fig. 5) [96]:
- DU;
- Bolus contrast-enhanced MSCT;
- MRI;
- Angiography.

Treatment of hepatic artery pseudoaneurysm

Open surgery or endovascular correction are the main methods of treating pseudoaneurysms [90, 93, 96, 100, 161, 176]. Thus, according to literature data, HA ligation was performed as a surgical benefit for some patients. Postoperative mortality in such patients reached 85%, and those patients who survived developed biliary complications and liver abscesses, which eventually led to the need for retransplantation [176]. Another group of patients underwent excision of the arterial defect with subsequent arterial reconstruction, including the use of shunt grafts. Postoperative mortality in this group was 28%; 66% of patients did not develop any postoperative complications. The remaining 6% developed biliary complications. Two patients underwent endovascular intervention. One was embolization of the pseudoaneurysm. The second one was placement of a covered stent. Both patients have been alive for more than 10 years and have not had any complications after the endovascular intervention [96].

Prognosis

In the literature, HA pseudoaneurysm is associated with a high mortality rate of 69% to 100% [36, 96–101, 176].

It should be noted that early detection of HA pseudoaneurysm in high-risk patients (patient with peritoneal infection, bacteremia, biliary leakage, biliodigestive anastomosis) is crucial for diagnostic evaluation and subsequent treatment with endovascular correction methods.

Open surgery should be followed by immediate revascularization even in the infected area if endovascular treatment fails. Detection of the pseudoaneurysm before it ruptures should ensure a successful outcome in 100% of cases.

It is worth keeping in mind that pseudoaneurysms are usually asymptomatic until they rupture, most commonly in the first five weeks after LT.

DU is not a highly effective method of diagnosing a HA pseudoaneurysm. Contrast-enhanced MSCT, MRI, or angiography should be performed.

2.5. Hepatic arterial rupture

Hepatic arterial rupture (HAR) is defined as the development of bleeding from the hepatic artery trunk. This is a rather severe complication leading to both impaired blood supply to the graft and the risk of patient death from bleeding [176].

HAR is a rare complication (0.64%); in most cases, it typically develops against the background of graft arterial pseudoaneurysm accompanied by infection, or occurs iatrogenically after endovascular interventions on the liver graft artery [88, 93, 96]. A ruptured hepatic artery leads to high patient mortality; therefore, it requires emergency surgical treatment [103].

Clinical presentation and diagnosis

The clinical presentation has always been accompanied by sudden bleeding: hemoperitoneum (58.8%), gastrointestinal bleeding (29.4%), hematoma (5.9%) and hemobilia in one patient (5.7%). The presence of fungal infection in the arterial wall was confirmed in 35% of patients. Bile leakage occurred in 41% of patients [176].

Treatment of hepatic arterial rupture

Since a ruptured hepatic artery is accompanied by acute bleeding, many surgical treatment options are available. In case of graft artery rupture, the following are performed: endovascular correction with embolization, arterial stenting, open arterial reconstruction, aorto-hepatic bypass, graft artery ligation, and retransplantation [176].

To date, the mortality rate in ruptured hepatic artery remains high, so there is no definite consensus on the choice of specific surgical treatment tactics.

Early postoperative mortality in such patients is 35%, the main causes being recurrent bleeding or sepsis [176].

A retrospective analysis has shown that revascularization in hepatic artery ruptures is not always indicated because one of the main causes of rupture is infection, and such patients subsequently die of sepsis. A study by Boleslawski et al. (2013) showed that HA ligation is efficient. Their study showed that 83% of patients who underwent revascularization died within 90 days of revascularization, with all patients who underwent ligation surviving 90 days after HA ligation. The one-year and three-year survival rates of patients after HA ligation were 100% and 80%, respectively, while the survival rates of patients who underwent revascularization were 14% and 14%, respectively [176].

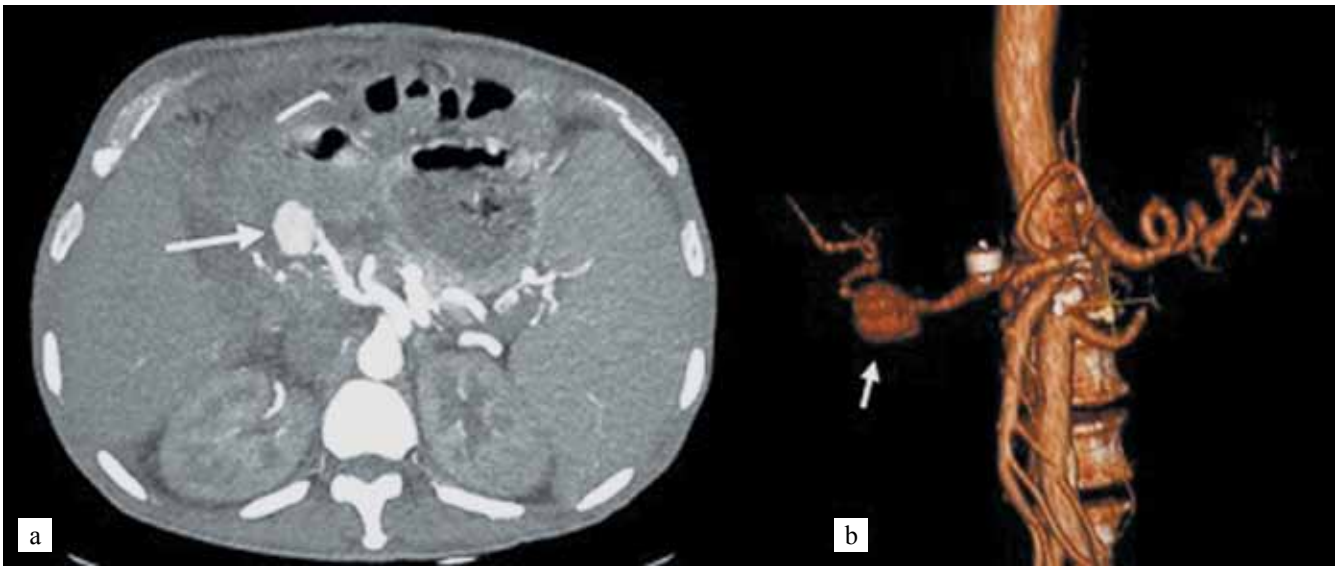

Fig. 5. Contrast-enhanced multislice computed tomography. The arrow indicates the site where hepatic artery pseudoaneurysm developed after liver transplantation. a, coronal projection; b, volume rendering [161]

3. VENOUS COMPLICATIONS

Compared to arterial complications, venous complications are less common with an established overall incidence of about 3%. They can be potentially dangerous, can lead to liver graft dysfunction and therefore represent an important source of post-LT morbidity and mortality, especially if they occur in the early postoperative period [6, 7, 10, 104–106, 175]. Numerous scientific studies have shown that the incidence of venous complications in pediatric transplants is higher than in adult patients [2, 72, 105, 107, 107, 108, 175]. Venous complications include portal vein complications and complications of the inferior vena cava and hepatic veins [7, 10, 109, 175].

3.1. Portal vein complications

The incidence of portal vein complications after liver transplantation is low (1%–3% of patients). These complications are more common after split transplantation, as well as in liver fragment transplantation from living donors, and in pediatric recipients [6, 7, 10, 109–112, 175].

3.1.1. Portal vein thrombosis

The incidence of portal vein thrombosis (PVT) after liver transplantation ranges from 0.3% to 2.6% [3, 111]. However, PVT incidence in patients who received a graft from a living donor is approximately 4%. This is due to technically more complicated venous reconstruction of the portal vein during transplantation, as well as to the fact that it is not always possible to take a liver fragment with a long section of the portal vein from a living related donor, especially when transplanting the right lobe of the liver [113]. PVT occurs more often in the early postoperative period, with 73% of all PVTs after liver transplantation occur in the first three months after surgery [113].

Risk factors

The most common causes of PVT are technical errors associated with an excessively long portal vein and its kinks and/or stenosis of the portal anastomosis [111].

Other risk factors are [36, 111–117]:

– previous surgical interventions on the portal vein;
– portal vein thrombosis before liver transplantation, requiring thrombectomy during surgery,
– small portal vein diameter (<5 mm);
– history of splenectomy;
– pepatic microvascular dysplasia;
– portosystemic shunts;
– use of vascular grafts for portal vein reconstruction.

Additional risk factors in patients who received a transplant from a living donor:

– Small size of the portal vein (length and/or diameter);
– Spatial position of the liver graft in the abdominal cavity.

Clinical presentation

The clinical presentation depends on the timing of thrombosis [36]. When PVT occurs early postoperatively, acute graft dysfunction predominates. If thrombosis occurs late, the clinical symptoms depend on the degree of collateral venous circulation [36, 111, 112].

The most important clinical manifestations of late PVT are manifestations of portal hypertension, including ascites, splenomegaly, cytopenia, and gastrointestinal bleeding from esophageal varices [36, 111].

Diagnosis of portal vein thrombosis after liver transplantation

Ultrasound monitoring should be performed regularly after LT to assess portal vein patency. Ultrasound is the easiest way to assess the patency of the portal vein of the graft, the speed of its blood flow, as well as the presence of blood clots in the portal system. Doppler flowmetry is used intraoperatively, as well as methods for measuring volumetric blood flow to exclude portal vein thrombosis, as well as to determine indications for modulation of portal blood flow [165]. Portal pressure is measured by direct cannulation of the portal vein or its tributaries, such as the inferior mesenteric vein or other mesenteric veins. It should be noted that high central venous pressure can affect portal vein pressure (PVP), and the PVP values in this case may be erroneous [165–167]. Most postoperative ultrasound monitoring protocols vary worldwide, with the belief that graft vessel DU monitoring should be performed daily for the first 5–7 days after LT [2, 59, 118–120]. If PVT is suspected, it is advisable to perform contrast-enhanced MSCT (Fig. 6) [121, 164]. Some authors have suggested the use of contrast-enhanced magnetic resonance imaging (MRI) of the liver with the administration of gadolinium, an MRI contrast agent. Also, there are protocols for the use of high-contrast ultrasound [119, 121].

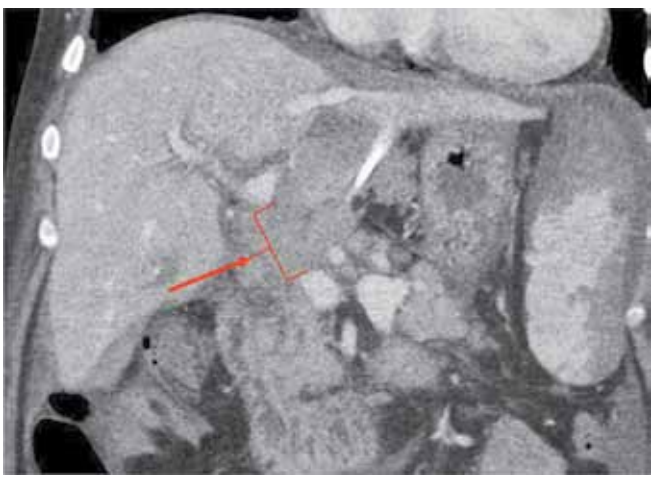

Fig. 6. Contrast-enhanced multispiral computed tomography. The arrow indicates the site of portal vein thrombosis in the patient after liver transplantation [164]

Treatment of portal vein thrombosis after liver transplantation

Treatment protocols include various methods of treatment: from anticoagulant administration to open portal reconstruction surgery. Currently, percutaneous transhepatic correction of portal vein blood flow is actively used. Interventional techniques include balloon angioplasty, portal vein stenting, anticoagulant administration into the portal vein system via a transjugular intrahepatic portosystemic shunt (TIPS) [122–125, 129, 130].

Among other things, the approach to PVT treatment differs depending on how long after LT it develops. Thus, in early PVT (in the first 72 hours after LT), accompanied by acute graft dysfunction, open revision of the portal anastomosis, thrombectomy, and reconstruction of the portal anastomosis must be performed. In PVT developing from postoperative day 3 to 30, treatment is initiated with the use of systemic anticoagulants. Endovascular correction of portal blood flow is also performed [122, 124, 126–128].

In late PVT, in the late period after LT, the main manifestations of portal vein thrombosis will be the development of portal hypertension syndrome (splenomegaly, ascites, esophageal varices, formation of collateral shunts, with the development of gastrointestinal bleeding) [36, 111]. As a rule, late after transplantation, endovascular techniques are used first, followed by various variants of portosystemic shunting, including TIPS, or open surgical reconstruction. In addition, treatment may require endoscopic hemostasis for bleeding from esophageal varices [122–125, 128–131].

It is worth noting that when TIPS is performed or when portal vein stenting is performed, different thrombolytic protocols (antiplatelet/anticoagulant therapy) are administered to patients [124].

Prognosis

PVT is fraught with graft loss and patient death. However, when PVT is diagnosed and treated early, real-world data show a good outcome with a survival rate >89% [139].

PVT is a rare but serious complication, especially when it develops in the early postoperative period. The physician's goal is to detect PVT as early as possible using ultrasound screening protocols. Open thrombectomy is required for PVT detected early posttransplant, but percutaneous interventions are gradually becoming the best therapeutic option with good outcomes and safety.

3.1.2. Portal vein stenosis

The true incidence of portal vein stenosis (PVS) after LT is not reliably known. The few data reported in the literature suggest an incidence of <3% [109].

Risk factors

Like PVT, the main risk factors are surgical technical errors during LT. Stenosis most often develops in portal anastomosis that is technically difficult to perform. Most often, such difficulties occur when there is a difference between the diameters of the portal vein of the donor and the recipient. This is often the case when transplanting liver fragments to children [109, 133, 134].

Clinical presentation

PVS is characterized by the clinical picture of portal hypertension syndrome and/or graft dysfunction [105, 106, 133, 135]. In practice, most patients with PVS have no complaints, and the diagnosis of stenosis is an incidental finding discovered during routine screening ultrasound [109, 133].

If patients develop a clinical picture, it is usually consistent with that of portal hypertension. These patients may develop gastrointestinal bleeding, ascites, and splenomegaly. Laboratory changes in the biochemical panel are not consistent and, therefore, are not specifically significant for PVS diagnosis [105, 106, 133, 135].

Diagnosis of portal vein stenosis after liver transplantation

The main methods of diagnosis and screening also include ultrasound (DU). The criteria for diagnosis by ultrasonography include [118]:

– the presence of a narrowing site in the portal vein;
– normal or reduced blood flow velocity through the portal vein to the narrowing site;
– post-stenotic portal vein dilatation;
– increased blood flow velocity (turbulent blood flow) after the portal vein narrowing site.

At the same time, according to some scientific papers, ultrasound has been considered as a sensitive method of investigation in relation to PVS, but not specific. In view of this, ultrasound criteria for PVS after LT have been calculated [118]:

1. Ratio of portal vein diameters before narrowing and after narrowing ≥50%;
2. Blood flow velocity is greater after the narrowing site than before the site >3:1.

If both criteria are present, contrast-enhanced MSCT is indicated for additional diagnosis and confirmation of PVS [118, 134, 136].

Treatment of portal vein stenosis

Surgical treatment, including anastomotic revision or retransplantation, is usually performed when PVS develops early after LT [137]. In case of asymptomatic PVS, patients with normal graft function should be followed up (systematic DU screening) without any intervention [114].

In case of clinical manifestations, the method of choice is percutaneous transhepatic methods of blood

flow correction (Fig. 7) [105, 106, 115–117, 133–135, 138–141]. Both balloon angioplasty and portal vein stenting, followed by antiplatelet therapy are performed. A disadvantage of stenting may be stent thrombosis, which may subsequently lead to the need for retransplantation. However, it has been shown that the risk of stent thrombosis can be significantly reduced with anticoagulants/antiplatelet agents [133, 134, 138–141].

Also, percutaneous interventions may pose a risk of complications, such as bleeding due to liver vessel injury, hemobilia due to ductal injury [137, 139].

PVS is a rather rare venous complication following LT. It develops most commonly after pediatric liver fragment transplantation or after transplantation of liver fragments from living donors. Ultrasound is an important diagnostic tool to assist the clinician because most asymptomatic cases may progress until PVS is clinically manifested by signs of portal hypertension. This in turn will adversely affect the prognosis of graft survival and ultimately patient survival.

Percutaneous intervention with stent placement has been shown to be the preferred treatment modality with a high success rate and low recurrence and/or complication rate.

3.2. Complications of the inferior vena cava and hepatic veins

Currently, impaired blood outflow from the liver graft by kinking, stenosis or thrombosis of the inferior vena cava (IVC) or hepatic veins, especially in transplants from living donors, are rare post-LT complications with a reported incidence of less than 3% [142, 143].

Clinical presentation and diagnosis

The clinical manifestations are diverse: lower limb edema, hepatomegaly, ascites, hydrothorax, Budd–Chiari syndrome, polyserositis, liver dysfunction, multiple organ failure, which may eventually lead to graft loss and patient death [6, 110, 144].

The main risk factor leading to caval complications is technical errors committed while performing caval anastomosis, which lead to kinking or thrombosis in the early postoperative period. Many authors have developed technical intraoperative techniques to prevent these complications. Thus, the piggyback technique and a modified version of the piggyback, i.e., hepatectomy techniques with preservation of the recipient inferior vena cava and formation of caval anastomosis directly with the recipient's hepatic veins, have been developed [145–151].

In the late postoperative period, chronic stenosis at the caval anastomosis site is the result of fibrosis, hyperplasia and/or external compression due to liver graft enlargement [6, 110, 144].

The diagnosis is made on the basis of DU, contrast-enhanced CT scan (Fig. 8) and with the help of transjugular cavography, which enables therapeutic manipulations during the procedure [152].

Treatment of caval complications

The treatment methods depend on the extent to which complications related to impaired blood flow from the transplant have developed in the long-term period. In case of severe graft dysfunction or if multiple organ failure develops, retransplantation is always indicated [132, 152].

In addition, minimally invasive endovascular interventions are the treatment of choice because the mortality rate after minimally invasive surgery is 11.1% compared with 41.6% for retransplantation.

Angioplasty by balloon-assisted transjugular intrahepatic portosystemic shunt placement can restore anasto-

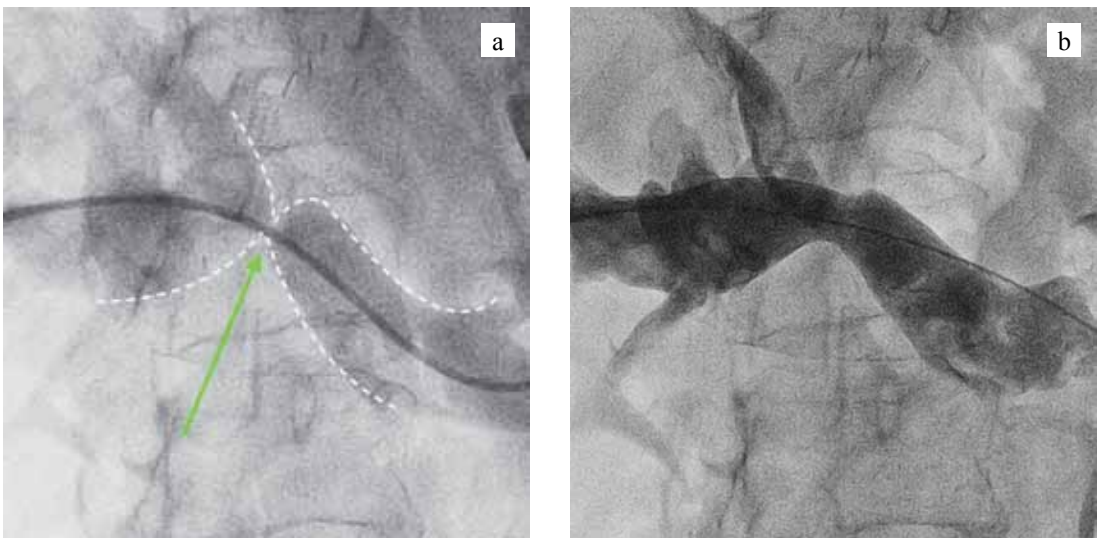

Fig. 7. Percutaneous retrograde portography: a, the arrow indicates the site of portal vein stenosis in percutaneous transhepatic portography; b, effect after balloon angioplasty of the site of portal vein stenosis [106]

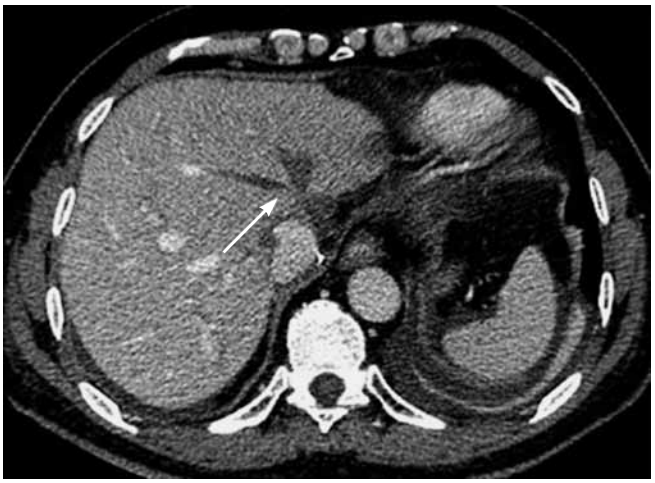

Fig. 8. Contrast-enhanced multislice computed tomography. The arrow indicates the site of hepatic vein thrombosis in the area of caval anastomosis [75]

motic patency in almost 100% of cases, but recurrence of stenosis is quite common and repeated angioplasties may be required [152].

Reports suggest that stenting of the caval anastomosis may be the best treatment option with a high success rate ranging from 73% to 100%. This method is safe and shows good long-term outcomes [132, 139, 152–160].

4. CONCLUSION

Vascular complications remain a major problem after liver transplantation. They are associated with a high mortality rate, especially if they manifest in the early postoperative period (first month after transplantation) and if they are not diagnosed in time.

The only solution to reduce the severity of VCs is to prevent them by controlling risk factors and, if this is not possible, early diagnosis is necessary, even in asymptomatic patients.

Many transplant centers around the world advocate the use of routine screening investigations such as ultrasound (DU) and, if in doubt, to perform contrast-enhanced CT scan or angiography, which is the standard.

If a VC is identified and the patient does not have severe liver graft dysfunction, it is more appropriate to attempt to resolve the complication by endovascular blood flow correction, as this method has demonstrated effective and safe outcomes.

Conversely, if there are serious consequences for the liver graft, the most effective therapeutic procedure is emergency retransplantation, which shows better outcomes in terms of efficacy and survival. However, organ shortage severely limits this treatment option.

The authors declare no conflict of interest.

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