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Diagnosis and treatment of resistant ascites in liver recipients in the early post-transplant period

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Abstract

Introduction. *Resistant ascites after liver transplantation is a relatively rare complication. At the same time, its presence significantly affects the prognosis and quality of life. Early diagnosis and successful treatment of resistant ascites can improve the long-term outcome. However, the aetiology of post-transplant ascites is heterogeneous, and the identification of the aetiological factor and the choice of treatment method in most cases is a significant problem for clinicians.*

Objective: *To present the review on methods of diagnosis and treatment of resistant ascites in liver recipients in the early post-transplant period.*

Material and methods. *The authors have reviewed the publications covering the main causes of ascites development after liver transplantation, the efficiency of instrumental diagnostic methods and surgical interventions in liver recipients with resistant ascites. The article has also discussed the authors' own observations of severe clinical cases of post-transplant ascites.*

Conclusions. *The preoperative status of the patient, the characteristics of the donor organ and the peculiarities of the surgical intervention should be taken into account in diagnosing the post-transplant ascites aetiology. In the absence of obvious predisposing factors, the patient should be evaluated sequentially to exclude vascular, intrahepatic and extrahepatic causes of ascites. The understanding of the main mechanisms of post-transplant ascites development and a consistent patient evaluation may help clinicians in choosing the treatment method.*

Keywords: liver transplantation, resistant ascites, splenic artery embolization, transjugular intrahepatic portosystemic shunt, hepatic venous outflow obstruction, arterioportal fistula, veno-occlusive disease, sinusoidal obstruction syndrome

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Abbreviations

APF, arterioportal fistula

CKD, chronic kidney disease
CT, computed tomography
DUS, Doppler ultrasonography
HA, hepatic artery
HVOO, hepatic venous outflow obstruction
HVPG, hepatic venous pressure gradient
IVC, inferior vena cava
LT, liver transplantation
MSCT, multislice computed tomography
PV, portal vein
RA, resistant ascites
SA, splenic artery
SASS, splenic artery steal syndrome
SFF, small-for-flow syndrome
SFS, small-for-size syndrome
VOD/SOS, veno-occlusive disease/sinusoidal obstruction syndrome

Introduction

Ascites is the most common manifestation of decompensated liver cirrhosis. The main cause of this complication is visceral arterial vasodilation, which leads to a decrease in the effective circulatory volume and sodium retention by the kidneys [1]. The appearance of ascites is associated with a poor prognosis: one-year survival is only 50%, and liver transplantation (LT) is the only definitive treatment method [2].

Hemodynamic changes that contribute to the development of ascites in liver cirrhosis are reversible after LT. On average, ascites resolves spontaneously or with diuretic therapy within 2–4 weeks. However, 3.4–7% of liver transplant recipients have resistant ascites (RA), which is not amenable to therapeutic treatments [3, 4]. Although this complication is relatively rare, it is associated with worse outcomes, including a decrease in a 1-year survival [5]. While the mechanisms of ascites development in patients with liver cirrhosis are well understood, RA after LT is a serious clinical problem and requires a comprehensive approach to diagnosis and treatment. The etiologic factors of RA are diverse and can be divided into three main groups: vascular, intrahepatic, and extrahepatic.

The objective was to present a literature review on the methods for diagnosis and treatment of resistant ascites in liver recipients in the early post-transplant period.

Vascular causes

Hepatic venous outflow obstruction

The most common cause of resistant ascites after LT is hepatic venous outflow obstruction (HVOO). The HVOO incidence varies from 0.5% to 9.5%, with a higher rate observed after living donor LT [6]. Previously, mechanical venous outflow obstruction was associated with the use of the piggyback surgical technique. However, further observations showed that the incidence of HVOO is more dependent on surgical experience than on the type of anastomosis to the recipient's inferior vena cava (IVC) [7].

The detection of HVOO signs in the early postoperative period may be associated with stenosis of the cavo-caval anastomosis, kinking, thrombosis of the hepatic veins or the IVC compression by the liver graft. At later stages after LT (more than 3 months), the HVOO development is more often associated with the stenosis formation in the area of the caval anastomosis as a result of intima hyperplasia or fibrosis. In liver fragment transplantation, the organ rotation and the deformation of hepatic veins may occur against the regeneration and an increase in the graft size, with possible subsequent impairment of venous outflow. As a differential diagnosis, it is necessary to exclude the HVOO cardiological etiology (the right heart chamber insufficiency, tricuspid regurgitation, regurgitation, etc.) [8–11].

The HVOO common clinical manifestations are ascites, hydrothorax, lower limb edema, renal failure, and/or abnormal liver biochemical test results [12]. Ultrasound imaging and Doppler

sonography remain important tools for screening for HVOO. Possible signs of the impaired venous outflow from the liver include dilated hepatic veins and decreased phasic blood flow in them (venous pulsatility index less than 0.45), which is due to the absence of the right atrial wave transmission through the IVC [13, 14]. However, a preserved three-phase blood flow waveform excludes HVOO. Additional signs include stenotic change in the area of the IVC anastomosis with turbulent blood flow and an increase in peak velocity in this segment while the blood flow velocity in the hepatic veins is reduced (less than 10 cm/s). The absence of blood flow registration at Doppler ultrasonography and the presence of echogenic masses in the lumen may indicate thrombosis of the hepatic veins [15, 16].

The characteristic signs at a computed tomography (CT) with intravenous contrast enhancement include a typical dilation of the hepatic veins that are not contrast-enhanced during the venous phase, or contrast-enhancement defects in the area of the caval anastomosis [9]. In cardiac causes of HVOO, a retrograde reflux of contrast agent from the right atrium into the IVC and hepatic veins may be observed during the arterial phase [17].

The most important diagnostic method of examination is venography, which allows both to evaluate not only the anatomical and hemodynamic features in the area of the IVC anastomosis, and also to conduct differential diagnostics and to assume the level of venous outflow obstruction. The stenosis detection and registration of clinically significant transanastomotic pressure gradient (the difference between free pressure in the hepatic vein and pressure in the right atrium) constitute the diagnostic criterion of HVOO. Currently, there is no consensus regarding the threshold value of the pressure gradient, and this

parameter value varies from 3-20 mm Hg. However, a gradient of more than 10 mm Hg is most often used to diagnose HVOO [18].

Treatment of HVOO depends on the etiologic factor and the period after LT. Balloon angioplasty and stenting are preferable for stenosis of the cavo-caval anastomosis due to minimal invasiveness and favorable long-term results of endovascular treatment. However, angioplasty in the early postoperative period is associated with the risk of extravasation and restenosis [19]. Relaparotomy can be considered if the signs of venous outflow obstruction occur in the first weeks after LT, while in the long term, reconstructive surgeries are associated with high risks of complications. Another indication for surgical intervention is thrombosis of the hepatic veins due to the high risks of thromboembolism during endovascular interventions. Surgical treatment options include revision and reconstruction of the anastomosis, thrombectomy, hepatopexy to prevent rotation and kinking [9, 18, 20].

The literature also describes alternative methods of surgical treatment of HVOO when the IVC is compressed by a large-size graft (large-for-size syndrome). M. Gastaca et al. published their experience of treating 3 patients using a breast implant placement retrohepatically, which helped to fix the graft in a midline position and maintain an adequate outflow from the hepatic veins. Resolution of resistant ascites was observed in all cases within 2 weeks after surgery. In addition, other centers have reported the use of inflatable balloons (Foley catheter, Blackmore catheter) and surgical gloves as a fixing elastic material [21–23].

A group of specialists from the Netherlands reported a case of successful treatment for RA in terms of HVOO by using a temporary implantation of a cava filter in the area of the IVC compression. A gradual resolution of ascites was observed on the 16th day after the

procedure, and the removal of the cava filter 3 weeks after its implantation was not accompanied by ascites recurrence [24].

Obstruction of portal blood flow to the liver

Impaired blood flow to the liver is a rare cause of post-transplant ascites and in most cases is caused by portal vein (PV) thrombosis or stenosis [25]. PV thrombosis occurs in 1–2% of liver recipients. However, among patients with previous portal thrombosis, the incidence of this complication varies from 4.7% to 36% [26]. PV stenosis is observed in 5% of cases and is typical mainly for the liver fragment recipients. The most common risk factors for complications at the PV level are macroscopic changes in the PV wall, previous splenectomy, mismatch between the anastomosed areas of the donor and recipient PV, the use of venous conduits during portal reconstruction, and a decrease in the portal blood flow velocity to less than 20 cm/s. The debut of the liver venous inflow occlusion in the first month after transplantation is often accompanied by the graft dysfunction, while in the late postoperative period, the manifestations of portal hypertension predominate: RA and bleeding from esophageal varices [26–29].

The most informative instrumental diagnostic techniques for the portal vein occlusion are Doppler ultrasonography (DUS) and multislice computed tomography (MSCT) with intravenous contrast enhancement. The ultrasonography pattern of portal thrombosis is characterized by absent blood flow mapping of the main trunk of the portal vein and its intrahepatic branches, the presence of echogenic masses in the portal vein lumen and an increased hepatic arterial signal [30]. However, the DUS sensitivity depends on the grade of PV thrombosis as assessed by Yerdel's classification (2000) and makes 48% at grade I, 82% at grade I, 100% grades III and IV. Thus, a low information value of the method is seen in

partial portal thrombosis [31]. The criteria for diagnosing the PV stenosis when using the imaging diagnostic techniques include stenosis of the hepatic vein altered segment by more than 50% according to MSCT, an increase in the blood flow velocity above 125 cm/s the depletion of intrahepatic portal blood flow, and an increase in the stenotic/prestenotic blood flow velocity ratio by 3 times or more according to ultrasound Doppler imaging [13, 32, 33].

The portal obstruction is confirmed and is treated in most cases by means of percutaneous transhepatic direct portovenography. Transjugular, transsplenic, and intraoperative mesenteric venous accesses have also been described. An increase in the transstenotic pressure gradient exceeding is significant and requires portal angioplasty and stenting. The technical success of these procedures ranges from 66–100% [28]. If endovascular treatment methods are ineffective, bypass operations or retransplantation may be considered. A surgical revision is advisable in the event of portal occlusion in the early postoperative period (no more than 1 week after LT) and in relation to technical causes (tension, kinking and(or) PV external compression) [27].

Arterioportal fistula

The resistant ascites can develop in case of fistula occurrence between the hepatic artery (HA) and a PV branch [25]. The main cause for the formation of arterioportal fistulas (APF) is the implementation of percutaneous transhepatic procedures such as liver biopsy and cholangiostomy with or without bile duct drainage [34]. The hemodynamic significance of arteriovenous shunt can be determined as based on a specific image pattern at Doppler ultrasonography or angiography. The main criteria in ultrasound examination include the visualization of a turbulent focus at the site of arterioportal shunt, reduced

arterial resistive indices (RI<0.50) of the supplying hepatic artery and hepatofugal blood flow in the PV with or without arterialization. Hemodynamically significant arterioportal fistulas (APFs), according to the results of angiography, are defined as visualization/contrast filling of the main trunk of the graft PV or its first-order branch [34, 35]. The PV arterialization and an increased portal pressure lead to the RA development.

Treatment options for APF include the embolization of the hepatic artery involved branch, segmentectomy/lobectomy of the liver graft, and in some cases, repeated LT [36, 37].

Local hemodynamic alterations

In addition to the obvious mechanisms of blood flow impairment in the graft, in recent years much attention has been paid to numerous hemodynamic syndromes that can also cause RA [7]. These include the “splenic artery steal syndrome” (SASS), small-for-flow (SFF) syndrome, and small-for-size (SFS) syndrome.

In SASS, there is a redistribution of arterial blood flow from the HA to the splenic artery (SA), which is associated with an increase in the splenic arterial bed and a decrease in its vascular resistance against the portal hypertension and splenomegaly in patients with liver cirrhosis. The development of liver hypoperfusion leads to a graft dysfunction and, in some cases, to the ascites development [38].

The SFS and SFF pathophysiology is explained by an increased portal pressure due to the use of a small graft (graft to recipient body weight ratio GRWR less than 0.8%) or the effect of previously formed excess portal blood flow on the graft in the presence of liver cirrhosis. Portal hyperperfusion has an inverse relationship with the hepatic artery

blood flow, being the so-called the hepatic artery buffer response. Blood flow is diverted to the SA system with the SASS development of [39].

The main factors for the SASS development include a spleen volume of more than 830 cm^3, the SA diameter more than 4 mm and/or SA/HA diameter ratio exceeding 1.5. DUS results are not specific, but an increase in the HA resistance index more than 0.80 is often reported [40]. The “gold standard” for diagnosing SASS is angiography. The diagnosis is based on recording a decrease in the HA blood flow velocity (a subjective assessment of a relative flow in the SA) in the absence of significant arterial anatomical defects, such as stenosis, thrombosis, and/or kinking with a decrease in the artery diameter by more than 50%. In severe SASS forms, the HA can be visualized in the portal venous phase of angiography. In other words, the contrast agent reaches the intrahepatic PV through the splenic vasculature simultaneously with the contrast-enhanced HA, which is associated with a significant decrease in the arterial blood flow [38].

The SA embolization is the main treatment of RA associated with impaired local hemodynamics of liver transplantation. Reduction of the SA blood flow makes it possible to reduce portal hyperperfusion and increase the HA blood flow. In case the endovascular treatment turns ineffective, the spleen devascularization or splenectomy should be considered [41–43].

Intrahepatic causes

Veno-occlusive disease or sinusoidal obstruction syndrome (VOD/SOS) is characterized by the development of hepatomegaly, ascites, and/or hyperbilirubinemia resulted from non-thrombotic obliteration of small centrilobular hepatic veins due to edema or fibrosis. The VOD/SOS pathophysiology is associated with endothelial cell injury caused by cytotoxic agents. The main etiologic factors of VOD/SOS after

LT include an acute cellular rejection, antibody-mediated rejection, and treatment with tacrolimus or azathioprine [44–47]. Cold ischemia time is an additional factor of the RA development. Liver tissue ischemia is reported to be accompanied by sinusoidal endothelial injury and hepatocyte edema, leading to vascular obliteration and increased intrahepatic vascular resistance [48].

VOB/SOS is characterized by specific changes suggested by CT or magnetic resonance imaging signs: heterogeneity/mosaic pattern of the liver parenchyma, stenosis of the hepatic veins, accentuated contours of the IVC and intrahepatic PV branches in the portal phase as a result of interstitial edema. However, such “congestive” changes in the liver are also possible with suprahepatic blood outflow obstruction, which requires HVOO exclusion of [49]. The main method of differential diagnosis is transjugular manometry with calculation of the hepatic venous pressure gradient (HVPG). HVPG values greater than 10 mm Hg indicate a clinically significant intrahepatic venous outflow block due to sinusoidal obstruction. The diagnosis of VOD/SOS can be confirmed by performing a liver biopsy (percutaneous/transjugular). However, one should bear in mind possible complications and weigh the risks of this invasive procedure against its diagnostic value [50].

In most cases, it is very difficult to identify the VOD/SOS etiologic factor. If an acute graft rejection or the cytotoxic effect of immunosuppressive therapy is excluded, the method of choice for the surgical treatment may be transjugular intrahepatic portosystemic shunt (TIPS). The efficacy of endovascular intervention in post-transplant RA reaches 86%. However, it is worth considering the possible complications when performing the TIPS procedure. The development of hepatic encephalopathy is reported in a third of liver recipients. Also, portal blood flow shunting can increase the graft ischemia and hepatocellular

insufficiency progression. Thus, TIPS should be performed after a thorough examination and exclusion of other causes of ascites [6, 51, 52].

RA can develop with relapse of viral hepatitis C in a liver graft even at early stages, without pronounced liver fibrosis or cirrhosis. In most cases, the histological examination of the patient liver biopsy can show perisinusoidal fibrosis [43]. Moreover, the presence of cryoglobulinemia in HCV-positive patients increases the risk of RA by 7 times, which confirms the assumption about the role of microangiopathy in the pathogenesis of ascites. The main method of RA treatment in this group of patients is a timely antiviral therapy using direct-acting antiviral drugs [25, 53].

Extrahepatic causes

In the absence of obvious surgical causes, the examination of patients with post-transplant RA should be aimed at excluding extrahepatic causes of ascites, such as heart failure, chronic kidney disease, and bacterial peritonitis. Single cases of enteropathy due to cytomegalovirus (CMV) infection and cryptosporidiosis with severe hypoalbuminemia due to malabsorption, which led to the development of RA, have also been described in the literature. A complete resolution of ascites was observed after the administration of etiotropic treatment [54, 55]. Finally, the RA etiology after LT may remain unknown. One hypothesis is that this type of ascites may be the result of persistent peripheral vasodilation and volume redistribution. Moreover, studies have shown that patients with an unknown cause of post-transplant RA have a significantly higher rate of spontaneous resolution of ascites [25].

Analysis of clinical cases

In our center practice, the RA incidence after LT was 3.2% (n=5/157). In 4 (80%) of these patients, the RA onset was observed in the early

postoperative period (less than 3 months after LT). The RA causes included SOS, SASS syndrome, APF, severe proteinuria resulted from the everolimus intake in a patient with chronic kidney disease (CKD); in one patient the RA etiology remained unknown. In all cases, a persistent resolution of ascites was achieved. Endovascular interventions were performed in 3 patients. The development of severe nephrotic syndrome in the course of the underlying CKD required the initiation of renal replacement therapy, and further kidney transplantation. In a patient with an unclear etiology, a spontaneous resolution of RA was noted within 6 months.

Below we present clinical cases that, in our opinion, deserve the most attention.

Clinical case #1

Patient B., 51 years old, underwent orthotopic transplantation for alcoholic liver cirrhosis receiving the whole liver from a blood-type-compatible cadaveric donor. By the time of surgery, the abstinence period had been for over 2 years. The liver cirrhosis was scored 11 by the Model for End-Stage Liver Disease (MELD), and assessed as Child-Pugh class C scored 10 points. Clinically, the liver cirrhosis decompensation was manifested by uncontrolled ascites and grade 2 esophageal varices. Intraoperatively, 7,500 ml of ascitic fluid was removed, the cold ischemia time was 5.9 hours. Caval reconstruction was performed with the IVC replacement. No hepatic steatosis was detected in the donor liver biopsy. The postoperative period was uneventful. The chosen immunosuppressive therapy included extended-release tacrolimus in combination with mycophenolic acid. Four weeks after LT, ascites relapsed. After three weeks of ineffective high-dose diuretic therapy, diagnostic-therapeutic laparocentesis was performed. Analysis of ascitic fluid excluded an infectious RA etiology. Further, an increased serum creatinine to 206

μmol/L, blood level tacrolimus up to 18.8 ng/mL, and alanine aminotransferase up to 200 U/L were noted. The tacrolimus dose was adjusted to reduce its blood level to 6.6 ng/mL; the serum creatinine level and blood transaminase decreased to normal. Due to RA persistence, abdominal CT scanning was performed, which revealed the signs of congestive changes in the liver, stenosis of hepatic veins and IVC at the level of the intrahepatic section (Fig. 1).

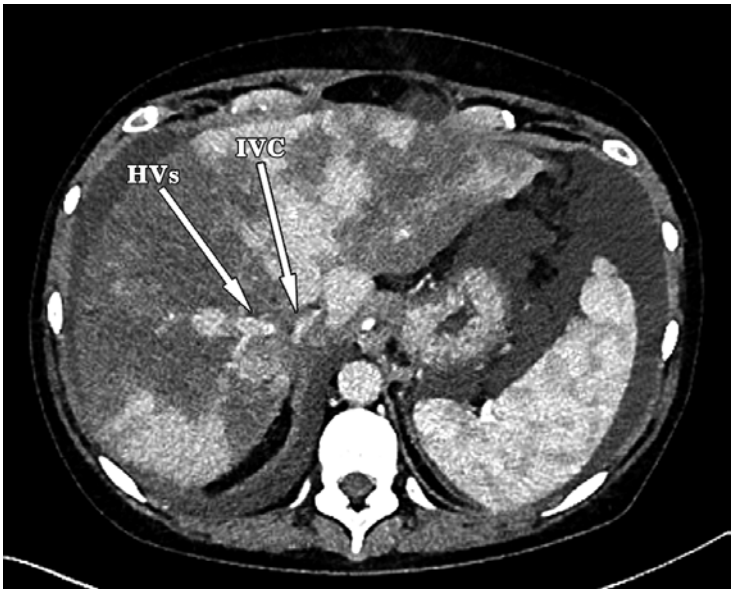

Fig. 1. Computed tomography scan of abdominal organs. Signs of congestive abnormalities in the liver are manifested by diffuse heterogeneous mottling appearance of the liver parenchyma, narrowed hepatic veins and the inferior vena cava at the level of the intrahepatic section (indicated by white arrows)

Venography was performed; an increase in HVPG to 23 mmHg was recorded in the absence of hemodynamically significant venous outflow obstruction; based on its results, the SOS/VOB was diagnosed. Liver biopsy was not performed. As conservative therapy was ineffective, 113 days after LT, a TIPS procedure was performed with the implantation of a bare metal stent (8 mm in diameter). For the month after the TIPS

placement, a progressive reduction of ascites was observed, and diuretic treatment was gradually discontinued. No hemodynamic impairments, graft dysfunction, or encephalopathy were observed thereafter. No RA recurrence was observed during 11 months of follow-up after TIPS.

Clinical case #2

Patient V., 50 years old had the previous history of liver cirrhosis due to viral hepatitis C. Despite the elimination of the hepatitis C virus with successful antiviral therapy, the manifestations of liver function decompensation persisted: RA, type 2 hepatorenal syndrome (HRS-CKD), and grade 2–3esophageal varices. The MELD status scored 20. The patient underwent orthotopic whole liver transplantation from a cadaveric donor (classical technique with IVC replacement). In the postoperative period, CKD manifestations persisted: serum creatinine up to 200 μmol/L, daily proteinuria 0.33 g/day. For the purpose of nephro- and cardioprotection, a combination immunosuppressive therapy was chosen: everolimus with the maintained blood serum level of the drug in the range of 6-8 ng/mL in combination with mycophenolic acid at a dose of 1 g per day. Due to satisfactory graft function, the patient was discharged from the hospital on the 20^{th} day. Six weeks after LT, the patient, having a satisfactory liver graft function, experienced a relapse of ascites, which, despite the treatment with diuretics, required repeated laparocenteses. The investigations performed excluded infection of ascitic fluid; markers of viral hepatitis C, B, CMV and Epstein-Barr virus were negative; the results of an echocardiography revealed no significant hemodynamic disorders, daily loss of protein in the urine did not reach the nephrotic level (0.6 g/day); serum albumin was 33 g/L. The diagnostic imaging techniques (Doppler ultrasonography, MSCT) showed no signs of venous outflow obstruction in the liver graft. Noteworthy

signs were the spleen area of 1560 cm^3 (over 830 cm^3), the SA/HA diameter ratio of 1.6 (SA was 8 mm, HA was 5 mm), and the registered contrast-enhanced HA in the portal phase, which suggested the presence of SASS syndrome. On day 80 after LT, celiacography was performed: a delayed HA contrast-enhancement with a predominant discharge of contrast towards the well-developed SA was recorded. Two-level occlusion of the SA was performed with the implantation of embolization coils in the projection of the splenic hilum and the SA proximal third (Fig. 2). The postoperative period was uneventful. During the first week after the procedure, a progressive decrease in ascites was noted, which allowed the discontinuation of diuretics without the need for further paracentesis. At 10 months after SA embolization, no recurrence of ascites was observed.

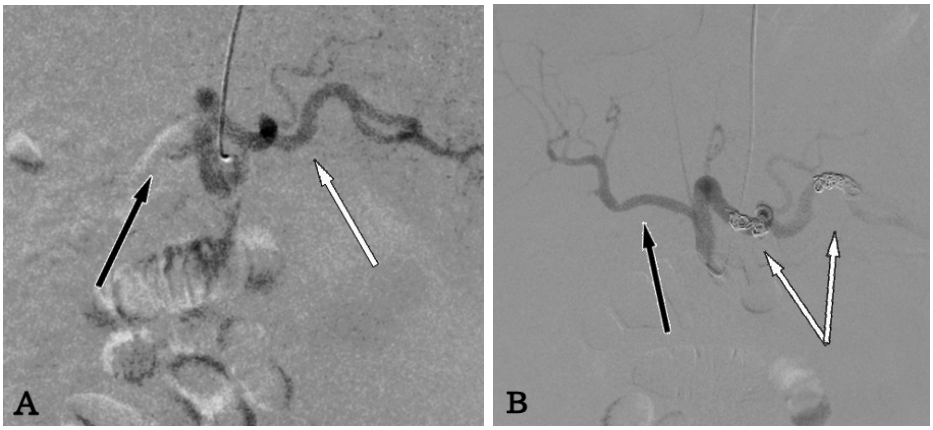

Fig. 2. A. Celiacography: large branches of the splenic artery up to 8 mm in diameter are visualized (white arrows); antegrade filling of the common hepatic artery does not occur (black arrow). B. Control celiacography: the embolization coils are visualized being located at two levels: in the splenic hilum and in the proximal third of the splenic artery (white paired arrows). The resumed antegrade blood flow in the common hepatic artery system is also visualized. (black arrows)

Clinical case #3

Patient M., 29 years old had a history of liver fragment retransplantation from a cadaveric donor on the 7th day after living donor liver transplantation due to HA thrombosis. The postoperative period was further complicated by biliary anastomosis failure, arrosive arterial bleeding, and arterial thrombosis of the liver graft. Complications were successfully coped with by a consistent use of open surgical, endovascular, and minimally invasive interventions, including: thromboextraction and HA stenting, percutaneous transhepatic drainage of the bile ducts [56, 57]. Eleven months after LT, ascites, bilateral hydrothorax, and episodes of encephalopathy were observed. Doppler ultrasonography showed signs of arterioportal shunt: dilatation of the HA branches and hepatofugal blood flow in the PV. Subsequent angiography revealed simultaneous contrast-enhancement of the PV and HA systems. It was not possible to clearly visualize the APF location, and therefore the graft HA embolization was performed. Subsequently, a steady resolution of all symptoms of portal hypertension was observed without laboratory test signs of increasing liver graft dysfunction. At the time of publication, the period after LT was more than 5 years.

Conclusion

To determine the etiology of resistant ascites after liver transplantation, it is necessary to exclude potential ascite-predisposing factors. Pretransplant predictors of ascites development after liver transplantation are the following: resistant ascites, hepatorenal syndrome, hepatic encephalopathy, and spontaneous bacterial peritonitis. These complications are often accompanied by irreversible changes in the portal blood flow amid pathological hemodynamics, which is in splenomegaly,

the splenic artery hypertrophy and the presence of collaterals. As a result, portal hyperperfusion may persist in the postoperative period, which supports the pathophysiological mechanisms of the resistant ascites development. Thus, in this category of patients, the presence of local hemodynamic disorders can be suspected. Previous spontaneous bacterial peritonitis may also be accompanied by infectious complications with the development of ascites in the postoperative period.

In liver fragment transplantation, the probable cause of resistant ascites is SFS syndrome, especially when using a small-for-size graft (GRWR less than 0.8). In addition, the complexity of vascular reconstruction and the unfitting diameters of the anastomosed vessels predispose to stenosis formation and impaired venous outflow. Surgical risk factors also include the surgery of the operation, the volume of blood loss, and cold ischemia time. The donor age and the severity of donor liver steatosis additionally increase the graft susceptibility to ischemia-reperfusion injury. Subsequently, an impaired intrahepatic microcirculation provokes the development of sinusoidal obstruction and the liver graft dysfunction.

In case of unclear etiology of resistant ascites, the investigation should start with a diagnostic laparocentesis to exclude infection and determine the nature of ascitic fluid. The next step should include imaging studies, which include Doppler ultrasonography and/or multislice computed tomography of the liver with contrast enhancement to assess the graft blood flow. Echocardiography is indicated to exclude cardiac causes. Invasive investigations may be required later: venography with manometry to assess pressure gradients, celiacography and/or liver biopsy (percutaneous/transjugular).

The treatment of resistant ascites is based on the elimination of the etiologic factor and the pathophysiological mechanism of its development

(Table). If conservative therapy is ineffective or the cause of post-transplant ascites has not been identified, it is possible to use interventional treatment methods, such as balloon angioplasty, splenic artery embolization, and the TIPS procedure after a thorough risk assessment of these invasive interventions.

Table. Diagnostic and treatment methods for resistant ascites

Etiology	Investigating technique	Treatment options
Vascular		
Surgical causes of hepatic venous outflow obstruction	Doppler ultrasound, contrast-enhanced MSCT, venography with manometry	Balloon angioplasty ± stenting, reconstructive surgeries
Obstruction of blood flow to the liver	+ Percutaneous transhepatic direct portovenography	
Arterioportal fistula	+ HA angiography	Embolization of the PA branch
Local hemodynamic abnormalities: Splenic artery steal syndrome Small-for-sizes graft (small-for-sizes syndrome)	Celiacography	SA embolization
Intrahepatic		
Sinusoidal obstruction syndrome (veno-occlusive disease)	Venography + manometry	Etiological therapy, if ineffective, TIPS
Acute liver rejection	Liver function tests + liver biopsy	Modification of immunosuppressive therapy
Hepatitis C recurrence in liver graft	+ Diagnostic PCR for hepatitis C	Antiviral therapy
Extrahepatic		
Cardiac	Echocardiography, NTproBNP	Treatment of the underlying disease according to the specialist's recommendations
Chronic kidney disease	Evaluation of renal function, ultrasound examination of the kidneys	
Infectious complications	Diagnostic laparocentesis, identifying the location of the infectious process	Antibacterial therapy
Uncertain etiology	Conduct a full range of investigations	Conservative therapy (diuretics, laparocentesis); if ineffective, consider splenic artery embolization or TIPS

Notes: HA, hepatic artery; SA, splenic artery; NTproBNP, N-terminal pro-B-type natriuretic peptide; PCR, polymerase chain reaction

Conclusion

When verifying the etiology of resistant ascites after liver transplantation, it is necessary to consider the patient's preoperative status, donor organ characteristics, and surgical intervention characteristics. In the absence of obvious predisposing factors, the patient should be evaluated sequentially to exclude vascular, intrahepatic and extrahepatic causes of ascites. Understanding the underlying mechanisms of resistant ascites and a consistent evaluation of patients with posttransplant ascites may help clinicians in choosing a treatment method.

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