https://doi.org/10.23873/2074-0506-2022-14-2-159-173

Role of portocaval shunts in development of complications after liver transplantation

D.L. Tsoy✉[1,2], M.I. Prudkov[1,2], O.G. Orlov[1], E.N. Bessonova[1,2], I.G. Leshchenko[1], L.V. Kardopoltsev[1], A.N. Ageev[1], S.I. Solodushkin[3]

[1]*Sverdlovsk Regional Clinical Hospital No. 1,*

3 Repin St., Ekaterinburg 620028 Russia;

[2]*Ural State Medical University,*

185 Volgogradskaya St., Ekaterinburg 620102 Russia;

[3]*Ural Federal University,*

19 Mira St., 620002 Ekaterinburg Russia

✉Corresponding author: Dmitriy L. Tsoy, Physician of the Department of Abdominal Oncology and Surgery, Sverdlovsk Regional Clinical Hospital No. 1; Medical Assistant at the Department of Surgery, Coloproctology and Endoscopy, Ural State Medical University, tsoy.transpl@gmail.com

Abstract

Rationale. *Portal blood flow is a key component in the viability of the liver transplant.*

Portocaval shunts formed on the background of the liver cirrhosis before transplantation can cause portal vein steal syndrome, with subsequent development of ischemic necrosis of the graft.

To date, the tactics of treating patients with portal vein steal syndrome during liver transplantation has not been sufficiently developed.

This paper presents a literature review and our own experience on this important, but little-studied issue.

Purpose. *The purpose of this research is to study the role of portocaval shunts in the development of complications after liver transplantation, based on a retrospective analysis of clinical cases.*

Conclusions. *In liver transplantation, portocaval shunts can cause the development of portal vein steal syndrome with subsequent development of liver failure. For the diagnosis of portal vein steal syndrome, it is important to use the data obtained at all stages of liver transplantation. Surgical correction of portal vein steal syndrome can be performed during liver transplantation and in the early postoperative period.*

Keywords: orthotopic liver transplantation, liver transplantation, portocaval shunts, portal vein steal syndrome

Conflict of interests Authors declare no conflict of interest

Financing The study was performed without external funding

For citation: Tsoy DL, Prudkov MI, Orlov OG, Bessonova EN, Leshchenko IG, Kardopoltsev LV, et al. Role of portocaval shunts in development of complications after liver transplantation. *Transplantologiya. The Russian Journal of Transplantation.* 2022;14(2):159–173. (In Russ.). https://doi.org/10.23873/2074-0506-2022-14-2-159-173

ALT, alanine aminotransferase

AST, aspartate aminotransferase

CT, computed tomography

DUS, Doppler ultrasound

IMV, inferior mesenteric vein

IVC, inferior vena cava

LHV, left hepatic vein

MELD, Model for the End-stage Liver Disease

MSCT, multi-slice spiral computed tomography

NO, nitric oxide

OLT, orthotopic liver transplant

PCS, portocaval shunts

PSS, portal vein steal syndrome

SMV, superior mesenteric vein

US examination, ultrasound examination

VEGF, vascular endothelial growth factor

Introduction

According to a number of authors, 15–40% of patients on the waiting list for liver transplantation are subjected to developing the spontaneous portocaval shunts (PCS).

When the liver transplantation is performed, the hepatopetal blood flow through the portal vein is normally noted; but under absence of any obstruction to the blood flow, the spontaneous shunts are obliterated by their own. However, under presence of the shunts with a diameter of more than 10 mm or in impaired liver venous outflow, the shunt obliteration may be delayed or not occur at all. In this case, the portal vein steal syndrome (PSS) develops. This condition is potentially the life-threatening due to the key importance of the portal blood flow in the functional state of the liver graft.

When analyzing the literature data, we found that today there is no uniform approach or algorithm in the treatment of patients with the PSS during liver transplantation.

In the paper, we present an analysis of the literature and our own experience on such important and, at the same time, little studied issue.

Pathogenesis of portal vein steal syndrome after liver transplantation

The pathophysiological basis of the portal vein steal syndrome (PSS) in the liver transplantation is the functioning portocaval shunts (PCS). Thanks to the functioning PCS, the blood is drained from the

portal system (where high blood pressure occurs in liver cirrhosis) into the system of the inferior vena cava (IVC) [1].

In addition to the pressure gradient between the portal and vena cava systems, the intrahepatic endothelial cells that produce the nitric oxide (NO) are involved in formation of the PCS, which leads to developing the splanchnic vasodilatation and increase in the portal pressure [2]. On the other hand, the release of the vascular endothelial growth factor (VEGF) by the endothelial cells of the portal system induces the neoangiogenesis [2].

More often, the development of PCS occurs in the splenorenal region, or the varicose veins of the left gastric vein are observed in isolation. Less often, the development PCS is noted in the retroperitoneal space, or the isolated varicose vein of the inferior mesenteric vein occurs.

In liver transplantation, after the implantation of a hepatic graft, in an absent intrahepatic obstruction, there is an increase in the portal blood flow velocity up to 1.8–2.8 L/min that happens due to the portal hypertension [3-6]. The unhindered blood flow contributes to the obliteration of the spontaneous shunts as a result of decrease in pressure in them. If the shunt diameter is more than 10 mm, as well as in the presence of a large total number of portocaval anastomoses, the spontaneous regression of the shunt or shunts can be slowed down [7, 8].

Moreover, under presence of factors that affect the development of intrahepatic edema and, as a result, of decrease in the blood outflow in the hepatic sinusoids, the spontaneous shunt obliteration may appear. These factors include: the acute rejection, venous congestion due to an impaired outflow from the graft, small liver volume syndrome, and severe ischemia-reperfusion injury [9, 10].

Prolonged persistence of the PCS (and, as a result, the portal hypoperfusion experienced by the graft) subsequently leads to

development of the severe steatosis and the portal vein thrombosis with the eventual loss of liver graft function [10-13].

Diagnosis of the portal vein steal syndrome in liver transplantation

When assessing clinical parameters before the liver transplantation, the hepatic encephalopathy (that is caused by the hyperammonemia in the systemic circulation) is of the particular importance [14]. In the study by Riggio et al., it was found that the large shunts in patients with the liver cirrhosis are often accompanied by the recurrent episodes of the encephalopathy [15]. Tarantino et al. demonstrated in their study that the hyperammonemia is a biochemical indicator of the presence of portocaval collaterals [16].

Thus, when detecting encephalopathy and elevated levels of ammonia in the blood, one can suspect the presence of the functioning PCS. However, it is important to note that the presence of PCS does not indicate the subsequent mandatory development of the PSS after the liver transplantation. To judge the hemodynamic significance of the PCS, it is important to assess its size and location, according to the computed tomography (CT) data.

The preoperative contrast-enhanced CT allows the portal system to be assessed for the presence of the PCS, to determine their exact location and diameter. For example, Aucejo et al. demonstrated that the preoperative CT is a useful method in identifying the potentially problematic shunts [17].

In the study by Simon-Talero et al., the prevalence of the PCS was demonstrated in patients with cirrhosis. So, this study included 1729 patients where 60% of them had the PCS. Meanwhile, half of the shunts had a diameter of 8 mm or more [18].

Thus, using the CT before liver transplantation makes it possible to assess the location and size of the PCS that in some cases allows planning the course of the operation with taking into account the need to separate them.

However, the CT diagnostics has limited possibilities in the case of severe tortuous course of the shunts and in a situation where the shunts are in a state of hypoperfusion during the time of the study [19].

The intraoperative diagnosis of the PSS consists of assessing the clinical signs and data obtained, using certain specific technologies. The clinical signs of the hemodynamically significant PCS include the hypoplastic portal vein (diameter <10 mm), visually accessible large shunts in the abdominal cavity, and an enlarged spleen. The clinical signs of the PCS presence are complemented by special instrumental studies such as the intraoperative Doppler ultrasound (DUS) and flowmetry.

The main criterion for presence of the PCS during the ultrasound examination is the hepatofugal blood flow. At the same time, the ultrasound does not make it possible to estimate the volume of afferent blood flow per unit of time.

This shortcoming is devoid in the intraoperative flowmetry. This method allows measuring simultaneously the flow of afferent blood flow and pressure in the portal vein system during surgery. The indisputable advantage of the flowmetry over the ultrasound is the relative simplicity of the technique and its independence from operator's skill. As early as in 1997, Danish transplantologists Rasmussen et al. showed the effectiveness of using this method in diagnosing the PSS [20].

In addition to ultrasound and flowmetry, the portography supplemented by the intraoperative ultrasonography can be used intraoperatively. Moon et al. demonstrated that this combined diagnostic approach is justified, since the intraoperative ultrasonography is not

always able to detect the PCS. Moreover, the intraoperative portography allows one more adequate control of shunt disintegration [19].

The main method for detecting the vascular complications after the liver transplantation is the ultrasound examination of the liver by the DUS [21].

This method makes it possible to non-invasively assess the patency of the portal vein and its velocity characteristics, which, in turn, allows us to make a timely assumption about the occurrence of the PSS and the early thrombosis of the portal vein. If there are signs of a decreased blood flow and the portal vein thrombosis, the CT angiography and direct portography are justified to visualize and assess the PCS at dynamic examination [22].

Clinical Case No. 1

Patient B, 35 years old, was admitted to Sverdlovsk Regional Clinical Hospital No.1 for liver transplantation. He had the liver cirrhosis of viral origin: HBV+HDV, Child-Pugh class C, MELD 18, portal hypertension syndrome, and hepatic encephalopathy stage I.

On March 20, 2019, orthotopic liver transplantation (OLT) was performed according to the method proposed by T.E. Starzl.

On the 3rd day, at DUS examination of the graft, the low blood flow (5 cm/sec) in the portal vein was noted. Transaminase levels were: AST 407 U/L, ALT 195 U/L.

On the 7th postoperative day, the ultrasound data were obtained and indicated the non-occlusive parietal thrombosis of the portal vein against the background of the low afferent blood flow. Transaminase levels made AST 55 U/L; ALT 89 U/L.

During the entire follow-up period, the results of laboratory tests were within acceptable range of values considering the duration and volume of the operation.

On the 14th postoperative day, the abdominal CT confirmed the portal vein thrombosis and revealed presence of a large splenorenal shunt.

In connection with the thrombosis and the clinical presentation of PSS, a decision was made on expediently performing the surgery to uncouple the splenorenal shunt.

The relaparotomy and ligation of the splenorenal shunt were implemented. The postoperative DUS examination of the graft revealed a clear improvement in the portal blood flow with increasing its rate up to 20 cm/sec.

After the operation, the patient had a decrease in the level of cytolysis, and cholestasis parameters.

However, 2 days after the ligation of the splenorenal shunt, a decrease in the blood flow in the portal vein to 5 cm/sec and signs of the portal vein thrombosis were noted again. In connection with the suspicion of a functioning splenorenal shunt, the cavography was performed that revealed a large splenorenal shunt (Fig. 1).

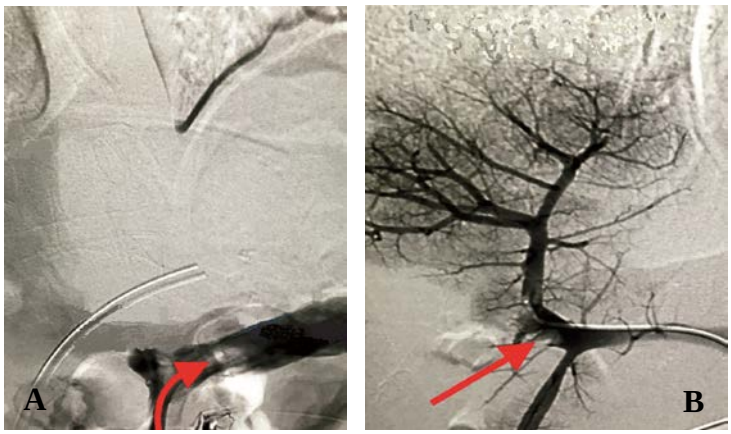

Fig. 1. Cavography of patient 1. (A) Contrast enhancement of the splenic vein through a splenorenal shunt (the red arrow indicates blood flow direction). (B) Portogram obtained through the splenic vein (red arrow indicates the portal vein)

In order to ensure the graft viability, it was decided to perform the second relaparotomy, ligation of the splenorenal shunt, and resection of the splenic vein.

A repeated ligation of the splenorenal shunt (Fig. 2), and the ligation of the splenic vein were performed.

The 1st day after the repeated relaparotomy was spent in conditions of the intensive care unit. The DUS examination of the graft demonstrated an improvement in the portal blood flow velocity up to 25 cm/sec.

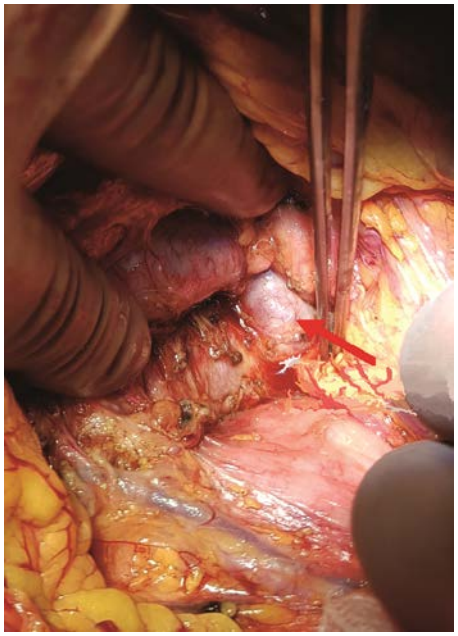

Fig. 2. Isolation of the splenorenal shunt (the red arrow indicates the splenorenal shunt)

On the 2nd day after the repeated relaparotomy, the patient was urgently taken to the operating room due to the clinical manifestations of the intra-abdominal bleeding. The intra-abdominal bleeding from the tail of the pancreas was stopped. In a severe condition due to a serious blood loss, the patient was transferred to the Intensive Care Unit.

Further, there was a recurrence of the intra-abdominal bleeding. In connection with the intraoperatively developed hemorrhagic shock, the hemorrhage was stopped by the tamponade of the pancreas tail.

After the stabilization of the patient's condition and the relief of the shock clinical symptoms, the patient was taken to the operating room. There the splenic vein stump was sutured and bursoomentostomy was formed. The DUS after the last operation revealed a satisfactory blood flow in the portal vein.

The CT scan of the head demonstrated the signs of brain damage.

After the operations performed, it was possible to achieve the adequate blood flow in the portal vein and satisfactory graft function. However, since the backdrop of the past bleeding episodes, the patient developed irreversible ischemic brain damage. As a result, the patient died.

Clinical Case No. 2

Patient K, 46 years old, was admitted to Sverdlovsk Regional Clinical Hospital No. 1 for liver transplantation for cirrhosis as a result of the chronic viral hepatitis B + D, Child-Pugh Class C, MELD 11, portal hypertension syndrome, resistance ascites, esophageal varices stage I, hypersplenism, and encephalopathy II degree.

On August 22, 2019, an operation was performed: orthotopic liver transplantation (OLT) from a cadaveric donor according to the Starzl technique without using the veno-venous bypass grafting. Intraoperatively, large venous collaterals were found in the lesser omentum and at the hilum of the spleen; and a large venous shunt of 2 cm in diameter was identified along the upper edge of the pancreas.

On the 1st day after the operation, pronounced cytolysis was noted with AST 6576 U/L, and ALT 2495 U/L. The liver ultrasound

examination revealed the non-occlusive portal vein thrombosis. The blood flow velocity in the portal vein was 12 cm/sec.

On the 2nd day, there was a progression of the hepatocyte cytolysis: the AST was 8204 U/L, ALT was 2841 U/L. The abdominal CT scan confirmed the portal vein thrombosis. Relaparotomy and the thrombectomy from the portal vein were performed.

After the operation, there was a slight positive trend in the form of regression of the cytolysis. Meanwhile, the DUS of the liver again indicated the signs of the portal vein thrombosis.

On the 4th day after transplantation, a repeated CT scan of the abdominal cavity was performed. On its basis, the thrombosis of the portal vein and its right lobar branch were diagnosed, and the large splenorenal shunt was detected (Fig. 3).

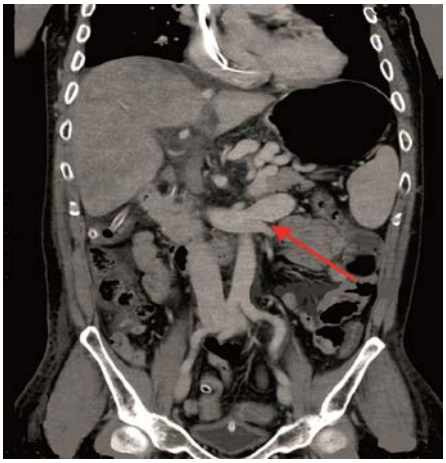

Fig. 3. The computed tomography image of patient No. 2 on day 4 after surgery (the red arrow indicates the splenorenal shunt)

As a result of the taken therapeutic measures, the patient's condition was stabilized. The hepatocyte cytolysis syndrome significantly regressed to AST of 458 U/L, ALT of 574 U/L.

On the 14th day after OLT, the condition worsened and manifested in the progressive hepatic encephalopathy. Despite the ongoing therapy, the patient's condition worsened significantly. According to laboratory tests, AST was 1424 U/L, ALT was 1709 U/L.

On the 19th day after OLT, the patient died as a result of the progressive multiple organ failure that occurred against the background of the acute ischemic hepatitis of the liver graft and the occlusive portal vein thrombosis.

Thus, the unrepaired large splenorenal shunt caused the development of the PSS followed by development of the portal vein thrombosis resulting in necrosis of the graft parenchyma.

Clinical Case No. 3

Patient K., 44 years old, was admitted to Sverdlovsk Regional Clinical Hospital No. 1 for liver transplantation for the liver cirrhosis of mixed etiology: (HCV + steatohepatitis), Child-Pugh class C, MELD 15, portal hypertension syndrome, esophageal varices stage I, ascites, and splenomegaly, hypersplenism.

The preoperative abdominal CT revealed a large splenorenal shunt up to 16 mm in diameter and a portocaval shunt in the left iliac fossa (Fig. 4).

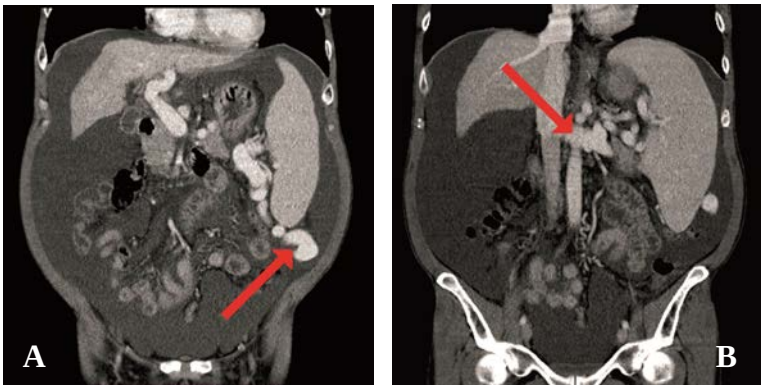

Fig. 4. Preoperative computed tomography of patient No. 3. (A) The arrow indicates the portocaval shunt in the left iliac fossa. (B) The arrow indicates the splenorenal shunt

On 12.10.2019, the orthotopic liver transplantation from a cadaveric donor was performed according to the Starzl technique. During the operation, 3 liters of ascites were drained, a significantly enlarged spleen, and the portal vein with a diameter of 14 mm were seen.

Moreover, the portocaval shunt of convoluted shape up to 12 mm in diameter was identified (Fig. 5).

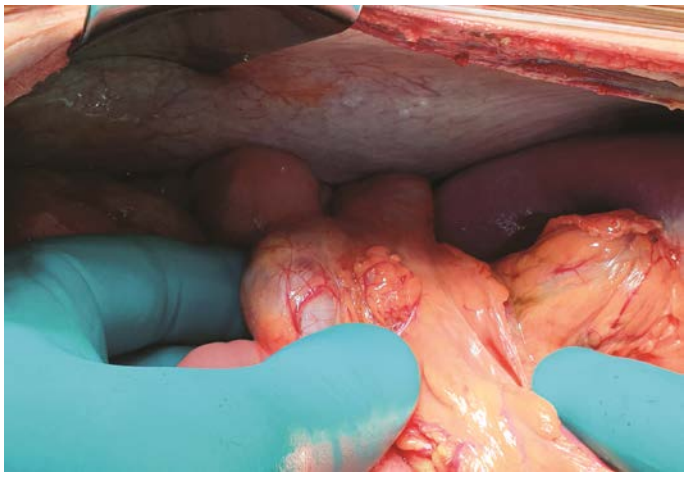

Fig. 5. The portocaval shunt in the left iliac fossa, intraoperative image

Taking into account the preoperative CT data, a decision was made to ligate the splenorenal shunt. The shunt was isolated at 3 cm laterally to the confluence of the left renal vein taken to the tourniquet (Fig. 6).

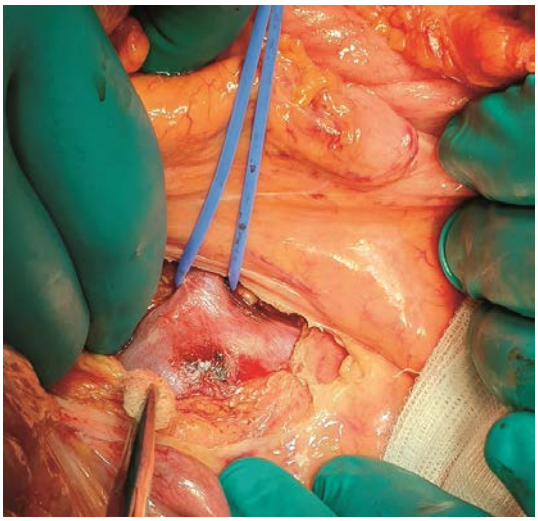

Fig. 6. The splenorenal shunt taken on the tourniquet, intraoperatively

The clamp was removed from the portal vein without clamping the splenorenal shunt, the blood flow was weakened. A vascular clamp was applied to the splenorenal shunt, which resulted in the significant increase in the blood flow.

The splenorenal shunt was ligated twice (Fig. 7A) and sutured with Prolene 3.0.

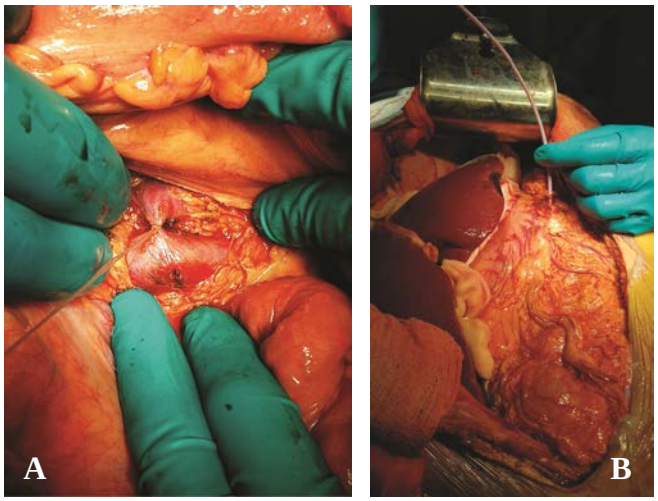

Fig. 7. Disconnection of the splenorenal shunt followed by the portal pressure measurement. (A) Ligation of the splenorenal shunt. (B) Catheterization of the gastroepiploic vein

Additionally, to assess the hemodynamic significance of the shunt in the right iliac region, the gastroepiploic vein was catheterized according to Seldinger: the 7Fr catheter was installed (Fig. 7B).

The portal vein pressure was recorded as 27 mmHg. It was decided that there was no need to ligate the portocaval shunt in the left iliac region.

On the 1st day after the operation, there was a maximum rise in liver enzymes: AST 4386 U/L and ALT-419 U/L. Further, there was a significant decrease in cytolysis.

Two weeks after the operation, the enzymes became AST 30 U/L; ALT 85 U/L.

The graft blood flow parameters on the first day after surgery were within acceptable limits: RI was 0.72; the blood flow velocity in the portal vein was 50 cm/sec. Further, at ultrasound examination, no signs of impairments in the afferent blood flow of the liver were revealed. The postoperative period was uneventful.

On the 25th day, the patient was discharged from the hospital.

Clinical Case No. 4

Patient V., 48 years old, was admitted to Sverdlovsk Regional Clinical Hospital No. 1 for the liver transplantation for liver cirrhosis as a result of the primary sclerosing cholangitis, Child-Pugh class B, MELD 5, portal hypertension syndrome, stage II esophageal varices, splenomegaly, and hepatic encephalopathy.

Before the operation, the ligation of the esophagus veins was performed.

The preoperative abdominal CT revealed the CT signs of the portal vein hypoplasia (Fig. 8A), and also an enlarged inferior mesenteric vein

(IMV) up to 13 mm in diameter flowing into the left iliac vessels (Fig. 8B).

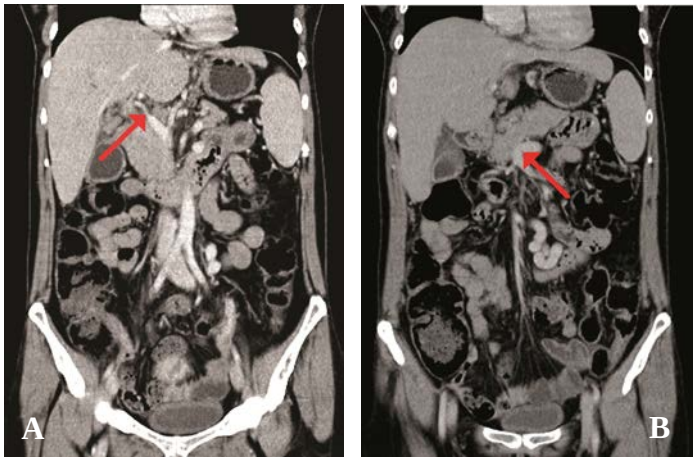

Fig. 8. Preoperative computed tomography of patient No. 4. (A) The arrow indicates the portal vein. (B) The arrow indicates the confluence of the inferior mesenteric vein with the superior mesenteric vein

On 29.11.2019, orthotopic liver transplantation from a cadaveric donor was performed according to the Starzl technique. During the operation, 500 ml of ascites was drained, a moderately enlarged spleen was recorded, as well as, the signs of the portal vein stenosis, which diameter was 7 mm (Fig. 9).

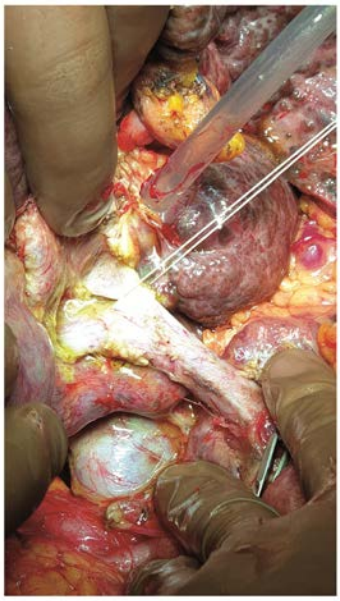

Fig. 9. Intraoperative image: portal vein is not dilated, up to 7 mm in diameter

After the hepatectomy, the clamp was removed from the portal vein to perform a subjective assessment of the portal blood flow. The blood flow was found to be reduced. In order to objectively assess the portal blood flow, a catheter was introduced in the trunk of the portal vein for a direct invasive pressure measurement (Fig. 10A). The pressure in the portal vein was 10 mm Hg. Based on the obtained CT data on the presence of the portocaval shunt and intraoperative portometry results, a decision was made to ligate the IMV.

Under the lower edge of the pancreas, an enlarged IMV was exposed (Fig. 10B) and clamped. The pressure in the portal vein with the clamped IMV was 20 mm Hg. Also, the clamp was removed from the portal vein again, the blood flow increased. At the next stage, the ligation and transection of the IMV was performed.

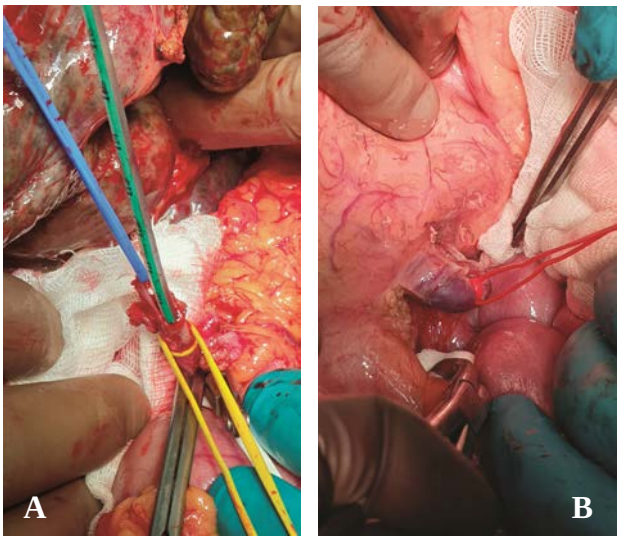

Fig. 10. Stages of the surgical correction of the portal vein steal syndrome in patient No. 4. (A) The catheter was placed in the lumen of the portal vein to measure invasive pressure. (B) The inferior mesenteric vein under the inferior rim of the pancreas was taken on a tourniquet

The rest of the operation passed uneventfully. An immediate flow of bile from the choledoch after the reperfusion was noted. The hepaticojejunostomy was formed on the excluded by Roux-en-Y loop of the small intestine. The early postoperative period was uneventful.

On the 1st day after the operation, the AST and ALT levels were 688 U/L, and 621 U/L, respectively. The portal blood flow velocity was 30 cm/sec. On the 7th day after the operation, AST was 47 U/L, ALT was 241 U/L at a portal blood flow rate of 43 cm/sec.

Parameters of the afferent blood flow in the hepatic artery and portal vein were satisfactory from the 1st day. The portal blood flow velocity was 30 cm/sec on the first day, 43 cm/sec on the seventh day.

The blood flow velocity in the hepatic artery was 50 cm/sec on the 1st day, 50 cm/sec on the 7th day; the resistive index was 0.77 and 0.61, respectively.

On the 16th day after the operation, the patient was discharged from the hospital with satisfactory graft function.

Clinical Case No. 5

Patient T., 38 years old, was admitted to the Sverdlovsk Regional Clinical Hospital No. 1 for the liver transplantation for the liver cirrhosis of HCV viral origin, Child-Pugh class C, MELD 22, portal hypertension syndrome, ascites, stage II esophageal varices, splenomegaly, hypersplenism, and hepatic encephalopathy.

The abdominal CT scanning before surgery revealed a large PCS of 15 mm up to 20 mm in diameter between the mesenteric veins and the iliac and rectal veins from the side of the IVC system.

On 27.01.2020, orthotopic liver transplantation from a cadaveric donor was performed by the classic technique.

About 3 liters of ascites were drained intraoperatively. The portal vein diameter was 10 mm. Taking into account the availability of the CT data on large PCS, the decision was made to isolate and ligate them regardless of the the invasive pressure values in the portal vein system. After the dissection of the mesentery of the small intestine, a large portocaval shunt (of more than 20 mm in diameter) was isolated and ligated (Fig. 11A, B).

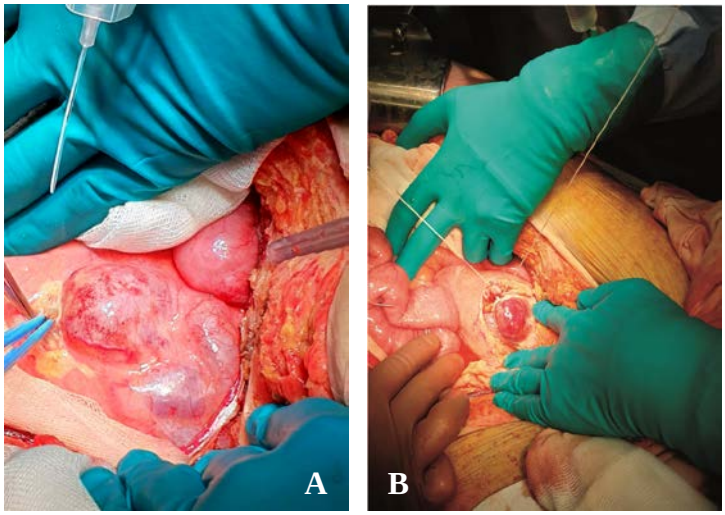

Fig. 11. Stages of surgical correction of the portal vein steal syndrome in patient No. 5. (A) The large portocaval shunt is exposed in the mesentery of the small intestine. (B) The portocaval shunt is ligated

After the ligation of the portocaval shunt, the pressure in the portal vein was measured by catheterization of the main trunk. The pressure was 20-21 mm Hg, which was regarded as a satisfactory value.

The first three days in the intensive care were uneventful. On the 3rd day, the parameters were as follows: AST 96 U/, ALT 167 U/L. A satisfactory synthetic function of the liver was noted. According to the DUS examination of the graft, the blood flow in the portal vein was 38 cm/sec on the 1st day and 60 cm/sec on the 3rd day; the blood flow through the hepatic artery was satisfactory. Meanwhile, on the fourth day after the OLT, the increasing hepatic encephalopathy was noted. A contrast-enhanced abdominal CT scanning was performed. It revealed the extended parietal thrombus of the superior mesenteric vein (SMV), and a large shunt between the IMV and IVC.

In the Angiography Department, the cavagraphy was urgently performed that confirmed the presence of the pathological shunt of the IMV flowing into the IVC below the confluence of the right renal vein (Fig. 12A). In order to prevent pulmonary embolism, a retrievable ALN

vena cava filter was introduced in the IVC below the orifices of the renal veins (Fig. 12B). The portocaval shunt was embolized with microcoils: two Cosmos Complex coils of 20 mm/65cm and two Interloc coils of 22 mm/60cm. The control cavography showed that the blood flow through the portocaval shunt had been terminated (Fig. 12C).

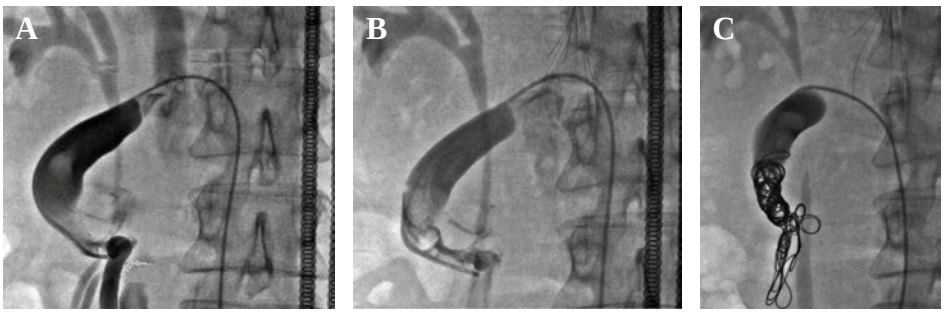

Fig. 12. Stages of endovascular intervention in patient No. 5. (A) Cavography examination revealed the presence of a portocaval shunt. (B) Before embolization, the cava filter was placed into the inferior vena cava. (C) Embolization of the portocaval graft with microcoils

The blood flow velocity in the portal vein was 68 cm/sec on the 1st day after the shunt embolization, and no portal vein thrombosis was observed. The vena cava filter was removed on the 10th day after the angiographic operation.

One month after the OLT, an episode of an acute cellular rejection occurred. It was arrested by the pulse therapy with glucocorticosteroids. Further, the graft function was satisfactory.

Discussion

Analysis of the available literature and our own clinical experience demonstrate the relevance of the problem of the portal vein steal syndrome after liver transplantation.

The first two clinical cases showed the irreversible development of the portal vein thrombosis in the presence of a functioning splenorenal shunt. Unfortunately, we did not pay due attention to the preoperative CT results that could have helped to change the intraoperative tactics.

In the 3rd case, we managed to avoid the PSS development thanks to preoperative planning that took into account the CT results, and to using the intraoperative measurement of pressure in the portal vein. These measures made it possible to assess the PCS impact on hemodynamics.

According to the literature, the splenorenal shunt is more common in liver cirrhosis than other PCS. In patients on the waiting list, the incidence of this shunt varies from 20% to 30% [23].

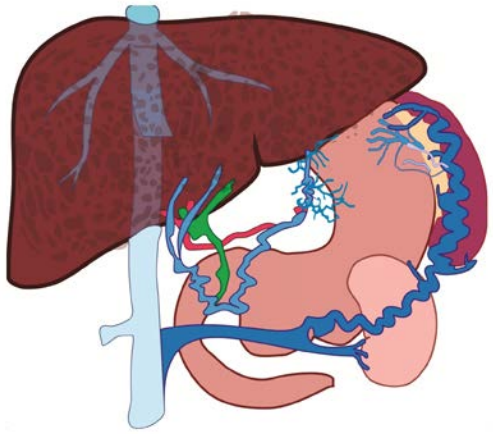

Fig. 13. A schematic illustration of a splenorenal shunt

In order to separate the portal vein system and the IVC in case of a functioning splenorenal shunt, various surgical techniques have been used, such as: a direct ligation of the splenorenal shunt and the ligation of the left renal vein.

H. Kim et al. consider the method of the direct ligation of the splenorenal shunt to be an effective and relatively safe method of the PSS surgical correction. Meanwhile, the main disadvantage of this method is

the risk pancreatic parenchyma damage with a subsequent risk of developing the arrosive bleeding [24] that became a fatal complication in the Clinical Case No. 1.

The most commonly used operation for disconnection of the pathological reset of the portal blood flow into the IVC system is ligation of the LPV. The first mention of ligation of the left renal vein dates back to 2000 in a study by Castilo-Suescun et al. [25].

Technically, the Kocher maneuver is performed, in which the duodenum and the head of the pancreas are retracted to the left to visualize the site where the LRV flows into the IVC. Before transecting the left renal vein, the vascular clamp should be applied on it, and the increase in the portal blood flow be confirmed. A number of authors have noted that after this procedure, there is a significant improvement in the portal blood flow parameters [26].

It is logical to assume that the renal pathology may develop after the LRV ligation. After more than 1 year of follow-up in patients, who underwent the LRV ligation, Samson et al. found that of 36 patients, only two had an increase in the creatinine and a decrease in the glomerular filtration rate [27].

The data obtained as a result of the largest series of the LRV ligation in Asan Medical Center, Seoul, South Korea are of a great practical interest.

The specialists of our Center performed 44 such operations in the course of the related donor liver transplantations. The mean diameter of the splenorenal shunt was 1.73 cm, the portal flow before the LRV ligation was 10.2 cm/sec; and after the vein ligation, it was 53.4 cm/sec. The mean creatinine level (3 months after surgery) was 133 μmol/L. In 4 cases, the patients required postoperative hemodialysis procedures.

Subsequently, kidney function recovered. All four patients had pre-existing renal pathology [28].

Basing on the described data, we can conclude that the LRV ligation of is effective in the restoring portal blood flow and has less risk of pancreatic damage compared to the direct ligation of the splenorenal shunt. In addition, it is relatively safe in relation to the development of the renal complications. The most preferred is the LRV ligation in close proximity to the IVC.

But we should note that the LRV ligation has its limitations. These include uncorrectable portal vein thrombosis and its hemodynamically significant stenosis. In case when the technical correction of the portal vein thrombosis is feasible, it is important to combine thrombectomy with the LRV ligation [29].

Thus, in deciding on the issue of surgical intervention in the splenorenal region (the LRV ligation or the splenorenal shunt ligation), the status of the receiving channel, that is, the portal vein, is of decisive importance. With existing thrombosis or stenosis, the important role is played by correction of the thrombectomy or stenting of the portal vein, which can be performed on time or after eliminating the PSS.

In addition to open surgical interventions, the endovascular techniques are currently used to blockade the left renal vein.

In order to treat the hepatic encephalopathy, a group of Japanese authors led by Matake applied a transhepatic access for obliteration of the splenorenal shunt using the double-ballon balloon technique [30].

A group of American authors used the percutaneous transfemoral embolization of the splenorenal shunt [31].

Our Clinical Cases No. 4 and 5, we encountered a problem of a significantly enlarged IMV, which had communication with the IVC system via iliac vessels. Portocaval collaterals formed by the superior and

inferior mesenteric veins are much less common compared to a splenorenal shunt) [18].

B. Kim et al. presented the case, in which the anomalous dilatation of the inferior mesenteric vein resulted in portal vein steal syndrome. In this case, embolization of the IMV with lipiodol coils through the transhepatic approach was successfully used [32].

Our experience of the PSS surgical treatment in case of enlarged IMV has shown that the direct ligation is an effective method of treatment. In the presence of an additional PSS, it is advisable to use the interventional radiology techniques for the purpose of their embolization.

The need for surgical correction of the pathologically dilated left gastric vein after the liver transplantation is much less common. Despite this, Gupta et al. published 3 cases of the gastric coronary vein PSS after the full-length liver graft transplantation. The flowmetry has been the main method to determine a compromised portal blood flow after the reperfusion. Meanwhile, the authors note that any decrease in the portal blood flow velocity below 1000 ml/min should prompt active measures for PCS detection.

It was the routine intraoperative use of flowmetry that allowed the authors to identify a hemodynamically significant dilation of the coronary vein and timely uncouple the pathological blood shunt. However, in one case, due to the satisfactory blood in the portal vein that was 1360 ml/min after the reperfusion, the large coronary vein varix was not disconnected. In the postoperative period, the patient suffered from several episodes of the acute cellular rejection and early relapse of the viral hepatitis C (HCV). Eleven months after the operation, the cholestasis and cytolysis syndromes were noted with CT- and ultrasound-confirmed presence of the hemodynamically significant varix of the coronary vein. Thus, according to the authors, a complete obliteration of the coronary vein

varix did not occur due to increase in the vascular intraparenchymal resistance that arose due to episodes of the acute cellular rejection and the HCV relapse [33].

The literature review and our own accumulated experience have shown that making the diagnosis of portal vein steal syndrome is a complex procedure that requires a thorough assessment of clinical and instrumental parameters at pre-, intra- and postoperative stages. In the absence of generally accepted algorithms and guidelines for the management of patients with portal vein steal syndrome after liver transplantation, further studies are needed to investigate in detail the pathogenesis of this condition, its diagnosis, and the choice of optimal surgical tactics.

Conclusions

1. Based on a retrospective analysis of clinical cases, we concluded that for the diagnosis of portal vein steal syndrome after liver transplantation, it is important to take into account the data of preoperative CT angiography, data on intraoperative pressure in the portal vein system, and the characteristics of the blood flow velocity in the portal vein according to Doppler ultrasound.

2. Hemodynamically significant portocaval shunts, if not eliminated, can cause the development of the portal vein steal syndrome followed by the occurrence of portal vein thrombosis.

3. When the portal vein steal syndrome is timely surgically treated, the early postoperative period is uneventful.

References

1. Colle I, Geerts AM, Van Steenkiste C, Van Vlierberghe H. Hemodynamic changes in splanchnic blood vessels in portal hypertension. *Anat Rec (Hoboken).* 2008;291(6):699–713. PMID: 18484617 https://doi.org/10.1002/ar.20667

2. Iwakiri Y, Groszmann RJ. The hyperdynamic circulation of chronic liver diseases: from the patient to the molecule. *Hepatology*. 2006;43(2 Suppl1):S121–131. PMID: 16447289 https://doi.org/10.1002/hep.20993

3. Bolognesi M, Sacerdoti D, Bombonato G, Merkel C, Sartori G, Merenda R, et al. Change in portal flow after liver transplantation: effect on hepatic arterial resistance indices and role of spleen size. *Hepatology*. 2002;35(3):601–608. PMID: 11870373 https://doi.org/10.1053/jhep.2002.31352

4. Henderson JM, Gilmore GT, Mac-kay GJ, Galloway JR, Dodson TF, Kutner MH, et al. Hemodynamics during liver transplantation: the interactions between cardiac output and portal venous and hepatic arterial flows. *Hepatology*. 1992;16(3):715–718. PMID: 1505914 https://doi.org/10.1002/hep.1840160316

5. Paulsen AW, Klintmalm GB. Direct measurement of hepatic blood flow in native and transplanted organs, with accompanying systemic hemodynamics. *Hepatology*. 1992;16(1):100–111. PMID: 1618464 https://doi.org/10.1002/hep.1840160118

6. Doi R, Inoue K, Kogire M, Sumi S, Takaori K, Suzuki T, et al. Simulta-neous measurement of hepatic arterial and portal venous flows by transit time ultrasonic volume flowmetry. *Surg Gynecol Obstet.* 1988;167(1):65–69. PMID: 2968000

7. Braun MM, Bar-Nathan N, Shaharabani E, Aizner S, Tur-Kaspa R, Belenky A, et al. Postshunt hepatic encephalopathy in liver transplant

recipients. *Transplantation.* 2009;87(5):734–739. PMID: 19295319 https://doi.org/10.1097/TP.0b013e318196340d

8. De Carlis L, Del Favero E, Rondinara G, Belli LS, Sansalone CV, Zani B, et al. The role of spontaneous portosystemic shunts in the course of orthotopic liver transplantation. *Transpl Int.* 1992;5(1):9–14. PMID: 1580990 https://doi.org/10.1007/BF00337182

9. Duffy JP, Hong JC, Farmer DG, Ghobrial RM, Yersiz H, Hiatt JR, et al. Vascular complications of orthotopic liver transplantation: experience in more than 4,200 patients. *J Am Coll Surg.* 2009;208(5):896–903. PMID: 19476857 https://doi.org/10.1016/j.jamcollsurg.2008.12.032

10. Sadamori H, Yagi T, Matsukawa H, Matsuda H, Shinoura S, Umeda Y, et al. The outcome of living donor liver transplantation with prior spontaneous large portosystemic shunts. *Transpl Int.* 2008;21(2):156–162. PMID: 18005086 https://doi.org/10.1111/j.1432-2277.2007.00593.x

11. Ploeg RJ, D'Alessandro AM, Stegall MD, Wojtowycz M, Sproat IA, Knechtle SJ, et al. Effect of surgical and spontaneous portasystemic shunts on liver transplantation. *Transplant Proc.* 1993;25(2):1946–1948. PMID: 8470237

12. Sekido H, Matsuo K, Takeda K. Severe fatty change of the graft liver caused by a portosystemic shunt of mesenteric varices. *Transpl Int.* 2002;15(5):259–262. PMID: 12012048 https://doi: 10.1007/s00147-002-0409-x

13. Tallón Aguilar L, Jiménez Riera G, Suárez Artacho G, Marín Gómez LM, Serrano Díaz-Canedo J, Gómez Bravo MA. Posttransplantation portal thrombosis secondary to splenorenal shunt persistence. *Transplant Proc.* 2010;42(8):3169–3170. PMID: 20970639 https://doi.org/10.1016/j.transproceed.2010.05.075

14. Blei AT, Cordoba J. Hepatic Encephalopathy. *Am J Gastroenterol.* 2001;96(7):1968–1976. PMID: 11467622 https://doi.org/10.1111/j.1572-0241.2001.03964.x

15. Riggio O, Efrati C, Catalano C, Pediconi F, Mecarelli O, Accornero N, et al. High prevalence of spontaneous portal-systemic shunts in persistent hepatic encephalopathy: a case-control study. *Hepatology.* 2005;42(5):1158–1165. PMID: 16250033 https://doi.org/10.1002/hep.20905

16. Tarantino G, Citro V, Esposito P, Giaquinto S, de Leone A, Milan G, et al. Blood ammonia levels in liver cirrhosis: a clue for the presence of portosystemic collateral veins. *BMC gastroenterology*. 2009;9:21. PMID: 19292923 https://doi.org/10.1186/1471-230X-9-21

17. Aucejo FN, Hashimoto K, Quintini C. Triple-phase computed tomography and intraoperative flow measurements improve the management of portosystemic shunts during liver transplantation. *Liver Transpl.* 2008;14(1):96–99. PMID: 18161777 https://doi.org/10.1002/lt.21377

18. Simón-Talero M, Roccarina D, Martínez J, Lampichler K, Baiges A, Low G, et al. Association between portosystemic shunts and increased complications and mortality in patients with cirrhosis. *Gastroentero-logy.* 2018;154(6):1694–1705. PMID: 29360462 https://doi.org/10.1053/j.gastro.2018.01.028

19. Moon DB, Lee SG, Ahn C, Hwang S, Kim KH, Ha TY, et al. Application of intraoperative cine-portogram to detect spontaneous portosystemic collaterals missed by intraoperative doppler exam in adult living donor liver transplantation. *Liver Transpl.* 2007;13(9):1279–1284. PMID: 17763379 https://doi.org/10.1002/lt.21252

20. Rasmussen A, Hjortrup A, Kirke-gaard P. Intraoperative measurement of graft blood flow – a necessity in liver transplantation.

Transpl Int. 1997;10(1):74–77. PMID: 9002157 https://doi.org/10.1007/BF02044347

21. Novruzbekov MS, Olisov OD. Vascular complications after orthotopic liver transplantation. *Transplantologiya. The Russian Journal of Transplantation.* 2017;9(1):35–50. (In Russ.). https://doi.org/10.23873/2074-0506-2017-9-1-35-50

22. Lee M, Kim SK, Chung YE, Choi JY, Park MS, Lim JS, et al. Portal venous perfusion steal causing graft dysfunction after orthotopic liver transplantation: serial imaging findings in a successfully treated patient. *Ultrasonography.* 2016;35(1):78–82. PMID: 25827474 https://doi.org/10.14366/usg.15012

23. Awad N, Horrow MM, Parsikia A, Brady P, Zaki R, Fishman M, et al. Perioperative management of spontaneous splenorenal shunts in orthotopic liver transplant patients. *Exp Clin Transplant.* 2012;10(5):475–481. PMID: 22891944 https://doi.org/10.6002/ect.2011.0201

24. Kim H, Yoon KC, Lee K-W, Yi N-J, Lee HW, Choi YR, et al. Tips and pitfalls in direct ligation of large spontaneous splenorenal shunt during liver transplantation. *Liver Transpl.* 2017;23(7):899-906. PMID: 28481004 https://doi.org/10.1002/lt.24783

25. Castillo-Suescun F, Oniscu GC, Hidalgo E. Hemodynamic consequences of spontaneous splenorenal shunts in deceased donor liver transplantation. *Liver Transpl.* 2011;17(8):891–895. PMID: 21425432 https://doi.org/10.1002/lt.22304

26. Slater RR, Jabbour N, Abbass AA, Patil V, Hundley J, Kazimi M, et al. Left renal vein ligation: a technique to mitigate low portal flow from splenic vein siphon during liver transplantation. *Am J Transplant.* 2011;11(8):1743–1747. PMID: 21668639 https://doi.org/10.1111/j.1600-6143.2011.03578.x

27. Samson RH, Lepore MR Jr, Showalter DP, Nair DG, Lanoue JB. Long- term safety of left renal vein division and ligation to expedite complex abdominal aortic surgery. *J Vasc Surg.* 2009;50(3):500–504. PMID: 19595540 https://doi.org/10.1016/j.jvs.2009.04.041

28. Lee SG, Moon DB, Ahn CS, Kim KH, Hwang S, Park KM, et al. Ligation of left renal vein for large spontaneous splenorenal shunt to prevent portal flow steal in adult living donor liver transplantation. *Transpl Int.* 2007;20(1):45–50. PMID: 17181652 https://doi.org/10.1111/j.1432-2277.2006.00392.x

29. Reddi MS, Rela M. Portosystemic collaterals in living donor liver transplantation: what is all the fuss about? *Liver Transpl.* 2017;23(4):537–544. PMID: 28073180 https://doi.org/10.1002/lt.24719

30. Matake K, Tajima T, Yoshimitsu K, Irie H, Aibe H, Nishie A, et al. Hepatic encephalopathy from dual splenorenal shunts treated with balloon-occluded retrograde transvenous obliteration by using a double-balloon technique. *J Vasc Interv Radiol.* 2007;18(11):1436–1440. PMID: 18003996 https://doi.org/10.1016/j.jvir.2007.07.024

31. Al hajjaj A, Bonatti H, Krishna M, Dickson R, McKinney JM, Nguyen J, et al. Percutaneous transfemoral embolization of a spontaneous splenorenal shunt presenting with ischemic graft dysfunction 18 months post-transplant. *Transpl Intl.* 2008;21(8):816-819. PMID: 18498312 https://doi.org/10.1111/j.1432-2277.2008.00691.x

32. Kim B, Kim KW, Song G-W, Lee S-G. Portal flow steal after liver transplantation. *Clin Mol Hepatol.* 2015;21(3):314-317. PMID: 26523275 https://doi.org/10.3350/cmh.2015.21.3.314

33. Gupta A, Klintmalm GB, Kim PT. Ligating coronary vein varices: An effective treatment of "coronary vein steal" to increase portal flow in liver transplantation. *Liver Transpl.* 2016;22(7):1037–1039. PMID: 27028766 https://doi.org/10.1002/lt.24448

Information about the authors

Dmitriy L. Tsoy, Physician of the Department of Abdominal Oncology and Surgery, Sverdlovsk Regional Clinical Hospital No. 1; Medical Assistant at the Department of Surgery, Coloproctology and Endoscopy, Ural State Medical University, tsoy.transpl@gmail.com

35%, development of the concept of a scientific article and its design, obtaining data for analysis, analysis of the obtained data; writing the text of the manuscript; review of publications on the topic of the article

Mikhail I. Prudkov, Prof., Dr. Sci. (Med.), Science Director of the Surgical Clinic, Sverdlovsk Regional Clinical Hospital No. 1; Head of the Department of Surgery, Coloprocology, and Endoscopy, Ural State Medical University, https://orcid.org/0000-0003-2512-2760

20%, development of the concept of a scientific article and its design, obtaining data for analysis, analysis of the received data; writing the text of the manuscript

Oleg G. Orlov, Cand. Sci. (Med.), Head of the Department of Abdominal Oncology and Surgery, Sverdlovsk Regional Clinical Hospital No. 1

15%, development of the concept of a scientific article and its design, obtaining data for analysis, analysis of the obtained data;

Elena N. Bessonova, Dr. Sci. (Med.), Head of the Gastroenterology Department, Sverdlovsk Regional Clinical Hospital No. 1; Associate Professor of the Internal Medicine Department, Faculty of the Advanced Training and Professional Retraining, Ural State Medical University, https://orcid.org/0000-0002-4223-3473

10%, obtaining data for analysis, analysis of the obtained data

Ilya G. Leshchenko, Head of the Organ Donation Department, Sverdlovsk Regional Clinical Hospital No. 1

5%, obtaining data for analysis

Lev V. Kardopoltsev, Head of the Department for X-ray Surgical Methods of Diagnosis and Treatment, Sverdlovsk Regional Clinical Hospital No. 1

5%, obtaining data for analysis

Artem N. Ageev, Cand. Sci. (Med.), Physician of the Diagnostic Radiology Department, Sverdlovsk Regional Clinical Hospital No. 1

5%, obtaining data for analysis

Svyatoslav I. Solodushkin, Cand. Sci. (Phys.-Math.), Associate Professor of the Computational Mathematics and Computer Science Department, Ural Federal University

5%, obtaining data for analysis

The article was received on February 1, 2022;
Approved after reviewing March 3, 2022;
Accepted for publication March 30, 2022