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Single-center experience of intraoperative ligation of the splenic artery for prevention of splenic artery steal syndrome in patients after living donor liver transplant

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Abstract

Background. *Living donor liver transplant is an effective method of treatment in patients with different types of end-stage liver diseases. Unfortunately, patients undergoing such a complex treatment sometimes develop various vascular complications. Splenic artery steal syndrome has emerged as a cause of graft ischemia in living donor liver transplant recipients and may lead to high liver enzyme levels, cholestasis, hepatic artery thrombosis, and even a graft loss in some severe cases.*

Objective. *Evaluation of the first results in the experience of our center with a routine intraoperative ligation of the splenic artery during the procedure of right lobe living donor liver transplantation in adult recipients for the prevention of the steal syndrome development in the postoperative period.*

Material and methods. *Living donor liver transplant recipients with known hepatic arterial flow impairment were retrospectively studied. Patients were allocated into groups with regard whether the splenic artery had been ligated or not during the transplant procedure. Arterial complications were reviewed in both groups.*

Results. *None of 30 patients with ligated splenic artery developed splenic artery steal syndrome after living donor liver transplant. splenic artery steal syndrome occurred in 60% patients with non-ligated splenic artery. Surgical technique of performing arterial anastomosis was not related to the splenic artery steal syndrome development ($p<0.01$). There was no local ischemic necrosis noted in the spleen in patients with the ligated splenic artery.*

Conclusion. *Based on the analysis of our own experience and literature data, the splenic artery ligation appears to be an effective and safe method for preventing a splenic artery steal syndrome in patients following right lobe liver transplantation, with a minimal risk of ischemic complications for the spleen. However, further studies with larger sample sizes are needed to obtain more reliable results. Ultrasound examination and endovascular intervention are the primary tools for an early detection of abnormalities and rapid restoration of arterial blood flow in the hepatic artery of the graft.*

Keywords: living donor liver transplant, vascular complications, splenic artery steal syndrome, doppler ultrasound, endovascular management

Conflict of interests Authors declare no conflict of interest

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AS, anastomotic stricture
CCI, comprehensive complication index,
EV, esophageal varix
GRWR, graft-to-recipient weight ratio
HA, hepatic artery
LGAS, liver graft artery stenosis
LGAT, liver graft artery thrombosis
LMWH, low molecular weight heparin
MELD, model for end stage liver disease
MOF, multiple organ failure
MSCT, multislice spiral computed tomography
PGNF, primary graft non-function
PVS, portal vein stenosis
PVT, portal vein thrombosis
SA, splenic artery

Introduction

Living donor liver transplantation is an effective treatment for patients with various types of end-stage liver diseases. Early vascular complications, especially arterial ones, can lead to graft loss, especially if they are not diagnosed and treated in time. Liver graft artery thrombosis (LGAT) [1–4] and liver graft artery stenosis (LGAS) [4–5] may require revascularization or, in some cases, retransplantation [4–8]. At the same time, splenic artery (SA) steal syndrome is another cause of graft ischemia in liver recipients and may have the same negative effect. Steal syndrome can be characterized as a decreased blood flow into the hepatic artery (HA) in the absence of LGAT

and LGAS, which is associated with an increased blood flow into the enlarged SA. A complex combination of factors, including the graft artery hypoperfusion and portal hyperperfusion, can lead to the development of steal syndrome [9]. Several studies have reported successful treatment of steal syndrome and functional graft recovery using SA embolization [4, 10–11]. Other studies have noted the importance of detecting the enlarged SA in patients with cirrhosis and its endovascular embolization prior to transplantation to prevent the risk of developing steal syndrome in the early postoperative period [12–13]. Also, according to world literature, various transplant centers have reported successful prevention of steal syndrome using the routine intraoperative ligation of the splenic artery [14–17], which formed the basis of our study.

The objective was to evaluate the first results of our center's experience with routine intraoperative ligation of the splenic artery during the procedure of living related donor transplantation of the liver right lobe to adult recipients for the prevention of the steal syndrome development in the postoperative period.

Material and methods

The program of living related donor liver transplantation in the Republic of Uzbekistan on the base of the Hepatobiliary Surgery Department at V. Vakhidov Republican Specialized Scientific and Practical Medical Center for Surgery (Tashkent) started in October 2021. The surgical process (donor and recipient stages), as well as postoperative patient management, have been supervised by two experienced transplantologists.

A retrospective review and analysis were performed on prospectively collected information from our database of transplants performed from

October 2021 to November 2023. The mean follow-up period was 7 months (range 1–25 months). Patients with ligated or non-ligated SA at the time of liver transplantation were assessed and compared.

Patients. During the study period, we performed 35 orthotopic living related donor transplantations of the liver right lobe in adult patients. Among the recipients, there were 23 men and 12 women. The mean age was 41 (22–56) years, with a mean MELD score of 18 (10–30). In 32 cases, the donors were genetic relatives of the recipients. The familial relation degree between donors and recipients was distributed as follows: 9 donors were sons, 9 were brothers, 6 were sisters, 6 were cousins, 1 was a father, 1 nephew, and 1 aunt. Also, according to the laws of the Republic of Uzbekistan, spouses of recipients can be considered organ donors, provided that they have been married for more than 3 years. In view of this, 2 wives of recipients were approved as donors.

The main indication for transplantation was liver cirrhosis as a result of the following diseases: viral hepatitis B+D (30 cases), viral hepatitis C (3 cases), autoimmune hepatitis (1 case) and toxic hepatitis (1 case). All patients were diagnosed with portal hypertension and its complications, including esophageal varices (EVs) (in 100% of cases), bleeding EVs (7 cases), splenomegaly (100% of cases), cytopenia (100% of cases). Ligation esophageal varices was performed in 9 patients to prevent bleeding. In 3 patients, SA embolization was performed before liver transplantation. Two patients had grade 3-4 portal vein thrombosis according to Yerdell classification.

Indications for the splenic artery ligation and intraoperative technical charcteristics. Before liver transplantation, all patients underwent multislice spiral computed tomography (MSCT) with intravenous contrast enhancement, where, among other things, we assessed the SA and HA

diameters and, based on the difference in arterial diameters, determined the need for the SA ligation during transplantation. We determined the indications for the SA ligation according to the following criteria: if the diameter of the SA exceeded the diameter of the HA by 50% or more (Fig. 1), then in this case the SA was ligated. The SA ligation was performed at the level of the spleen hilum or the origin of the celiac trunk. In one case, we ligated the splenic artery and its collaterals after the SA embolization that had been preformed 7 years before transplantation, and, according to MSCT, the spleen had a new clearly marked collateral arterial blood supply. The gastroduodenal artery was also ligated in all cases. To prevent arterial hypoperfusion and reduce portal hyperperfusion [4, 18, 19], we tried to use grafts with a graft-to-recipient weight ratio (GRWR) greater than 0.9%.

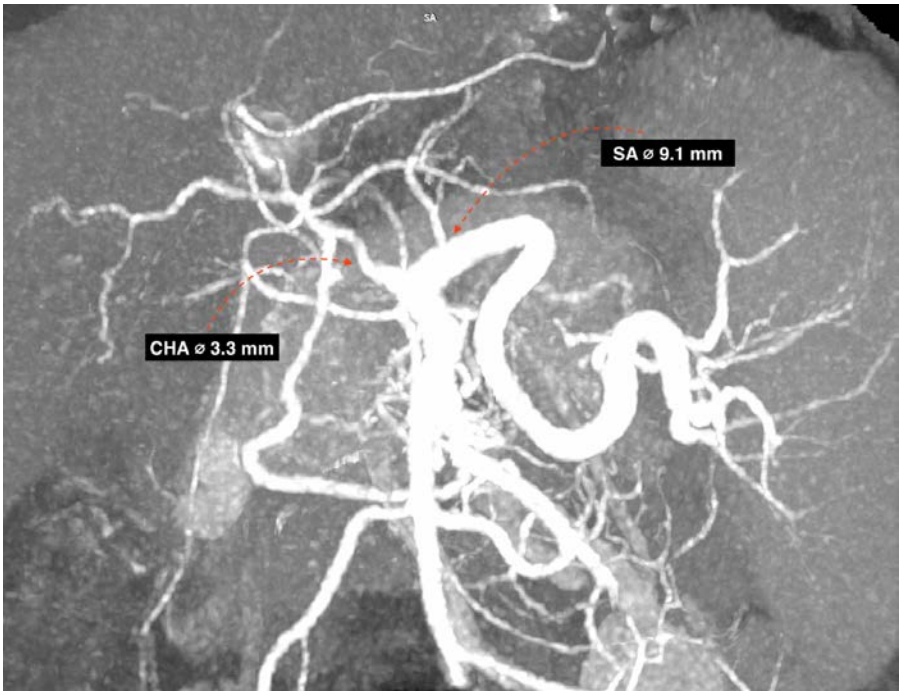

Fig. 1. Multislice spiral computed tomography with contrast enhancement. The difference in diameters of the splenic and common hepatic arteries exceeds 100%

CHA, common hepatic artery; SA, splenic artery

When forming arterial anastomoses, we used various surgical techniques. Thus, when the diameter of the donor HA was less than 2.5 mm or when there was a large discrepancy between the diameters of the donor and recipient arteries, we used separate interrupted sutures with Prolene 7/0 suture (using binocular optics with a magnification of 3.5 times).

When the diameter of the donor's obvoluted HA was more than 2.5 mm, a blanket suture with Prolene 7/0 was used. All anastomoses were formed with the recipient's general HA; but in two cases SA was used due to severe damage to the intima of the general HA. We routinely used intraoperative Doppler ultrasound (US) to monitor arterial inflow after arterial anastomosis had been formed, as well as after biliary reconstruction and final hemostasis.

Postoperative monitoring and differential diagnosis of steal syndrome. All patients received comprehensive thromboprophylaxis to reduce the risk of vascular complications. Routine prevention of postoperative arterial complications included the administration of alprostadil (prostaglandin E1) after arterial reperfusion. Further, starting from the first postoperative day, low molecular weight heparin (LMWH) and low doses of aspirin were administered starting from the 4th postoperative day. Alprostadil was discontinued 7 days after surgery. Treatment with LMWH was continued for 2 weeks after transplantation. Patients received aspirin for 3 months after surgery. In cases of significant coagulopathy, signs of bleeding, or platelet count lower than 50×10^9 /L, thromboprophylaxis was completely or partially discontinued until the corresponding complication was controled. We also provided intravenous fluid volume support under daily fluid balance monitoring.

Ultrasound monitoring was performed regularly for the first 7 days after transplantation. For routine ultrasound monitoring, GE Logiq P6 (General Electric, USA) and Mindray DC-40 (Mindray Medical International Limited, China) ultrasound systems were used with standard units of C6-2 convection sensors. The first postoperative monitoring of arterial blood flow using ultrasound was performed after transporting the patient to the Intensive Care Unit. Follow-up examinations were performed every 6 hours for the first week after surgery. After a week, ultrasound monitoring was performed once a day. In complicated cases, the period of ultrasound monitoring could last more than a week [4, 20]. Our protocol for monitoring arterial blood flow and control of arterial complications is demonstrated in Fig. 2.

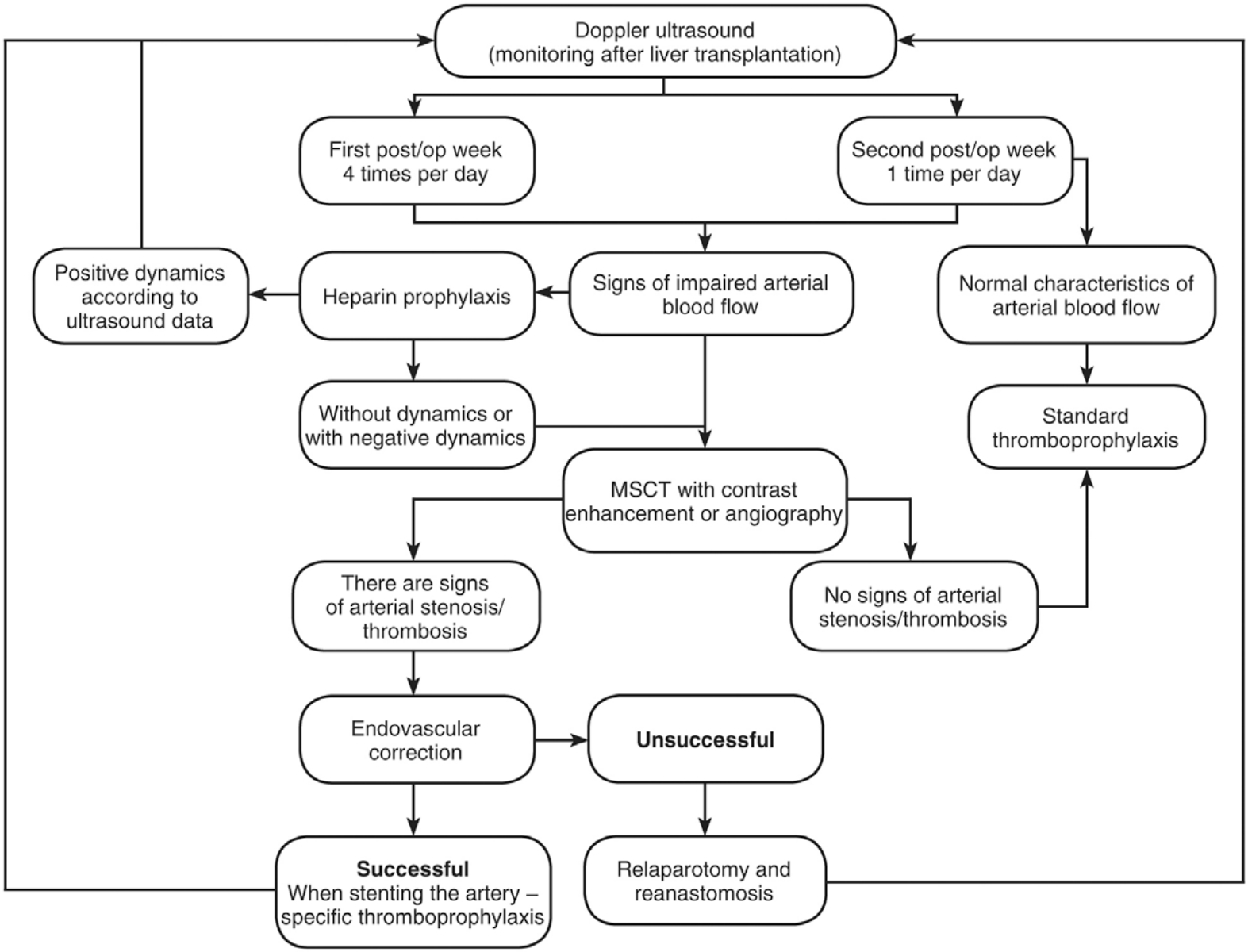

Fig. 2. Protocol for monitoring the arterial blood flow and the control for arterial complications

USE, Ultrasound examination; p/o, postoperative

The following dopplerography findings were considered significant: difficulty in visualizing the artery, changes in the resistance index (RI) with its increase to more than 0.85 or decrease to less than 0.5, as well as a decrease in peak arterial blood flow velocity to less than 15 cm/sec. In such cases, a permanent heparin infusion was started (a bolus dose of 80 U/kg and a maintenance dose of 18 U/kg/hour) with partial monitoring of thromboplastin time every 6 hours [21]. In cases where hepatic arterial flow could not be visualized by using ultrasound, an urgent contrast-enhanced MSCT was performed or the patient was urgently taken to the endovascular Operating Room for diagnostic angiography. After the impaired arterial blood supply to the graft had been confirmed, an immediate restoration of blood flow was performed, since in contrast-enhanced MSCT, or angiography, a final differential diagnosis of steal syndrome can be made to distinguish it from other arterial complications.

Diagnostic celiacography was performed using a 5-Fr CB1 5F, KA2 4-5F catheter (Merit Medical, USA) through a 25 cm sheath introducer 6Fr, 7Fr (Terumo, Terumo Cardioglass Corp., Japan). Steal syndrome was diagnosed in case of the following angiographic findings: absence of HA stenosis and thrombosis; a significant delay in filling the intrahepatic arterial branches with the contrast fluid compared to the rate of filling of the splenic arterial branches; the contrast fluid was quickly evacuated into the dilated SA. In case of steal syndrome, the SA coil embolization was performed. After the coil placement in the SA, the decrease in the blood flow was monitored for 5 minutes and, in absent angioreduction, an additional coil was placed until the effect was achieved. For embolization we used AZUR™ CX Peripheral Coil System coils (Terumo Cardioglass Corp., Japan). HA stenosis on angiography was defined as stenosis greater than 60% of the lumen diameter. The measurements of the vessel diameter and stenosis were made after

administering 200 μg of nitroglycerin and 50 Units/kg of heparin. Before revascularization, heparin was administered intravenously (50 mg/kg). Stenoses and thromboses were carefully passed through using a soft hydrophilic Prowaterflex 0.014" string (Asahi Intecc Co., Japan), Fielder 0.014" (Asahi Intecc Co., Japan) and PT2 LS 0.014" (Boston Scientific, USA) strings and performed balloon plastic repair and(or) stenting.

Statistical analysis. Continuous variables are presented as medians and ranges. Categorical variables are expressed as numbers and percentages. Patient survival rates were calculated using the Kaplan–Meier method. Differences in variables with a p-value < 0.05 were considered statistically significant. Statistical processing was carried out using Microsoft software Excel (USA), Orange 3 (Slovenia), IBM SPSS 26 (USA).

Results

In 30 cases, the SA diameter exceeded recipient's HA diameter by 50% or more. The mean HA diameter was 4.2 mm (2.8–6.0 mm), and the mean SA diameter was 8.8 mm (5.2–10.3 mm). The median difference in SA and HA diameters was 95% (4–241%). The median graft to recipient weight ratio (GRWR) was 1.1 (0.7–2.0).

Among all 35 patients, the SA was ligated in all cases where the SA exceeded the diameter of the HA by 50% or more, namely in 30 (85.7%) (patient characteristics and arterial complications are summarized in Tables 1 and 2). Of these, SA ligation was performed at the hilum of the spleen in 3 cases and at the level of the celiac trunk in 27 cases. In one case, we ligated the SA after coil embolization, since embolization had been performed 7 years before liver transplantation and, according to MSCT, the spleen had a new collateral arterial blood supply.

After liver transplantation, 7 (20%) of 35 patients had an impaired arterial blood flow. None of the 30 patients with a ligated splenic artery

developed steal syndrome after liver transplantation. Of the 7 patients with arterial complications, one patient (14.4%) developed graft arterial thrombosis, 3 patients (42.9%) had arterial stenosis, and another 3 patients had a steal syndrome (42.9%). In one of three patients with the steal syndrome, in whom SA embolization had been performed before transplantation, the SA was not ligated during transplantation, and the steal syndrome developed on the 7th postoperative day. The surgical technique of making the arterial anastomosis was not related to the development of steal syndrome ($p>0.5$). In patients with the ligated SA, no local ischemic necrosis was observed in the spleen.

Table 1. Clinical and demographic data of patients

Parameters	Value (n=35)
Age, years	41 (22–56)
Gender, n (%)	
Men	23 (65.7%)
Women	12 (34.3%)
Indications for transplantation, n (%)	
Viral hepatitis B+D	30 (85.7%)
Viral hepatitis C	3 (8.5%)
Autoimmune hepatitis	1 (2.9%)
Toxic hepatitis	1 (2.9%)
MELD score	18 (10–30)
GRWR, %	1.1 (0.7–2.0)
HA diameter, mm	4.2 (2.8–6.0)
SA diameter, mm	8.8 (5.2–10.1)
Difference between SA and HA diameters, %	95 (4–241)
SA ligation, n (%)	30 (85.7)
Ligation at the hilum of the spleen	3
Ligation at the level of the celiac trunk	27
Type of arterial anastomosis	
Interrupted suture	18
Blanket suture	17
Follow-up period, months	7 (1–25)

Notes: MELD, Model for End Stage Liver Disease; GRWR, graft to recipient weight ratio

Table 2. Arterial complications

Total complications, n	**7 of 35 (20%)**
Type of complication, n (%)	
LGAT	1 (14.4%)
LGAS	3 (42.9%)
Steal syndrome	3 (42.9%)
LGAS during SA ligation in the hilum of the spleen	3 of 3 (100%)
LGAS during SA ligation at the celiac trunk	–
Steal syndrome after SA ligation	–
Steal syndrome without SA ligation	3 of 5 (60%)
Post-operative day of complication development (range)	
LGAT	7 (7)
LGAS	3 (3)
Steal syndrome	4 (0–7)

Treatment of arterial complications. A summary of treatment methods is presented in Table. 3. In all cases of the developed arterial blood flow impairments, the selective celiacography was performed. The patient with arterial thrombosis underwent balloon angioplasty with HA stenting. All patients with LGAS underwent balloon angioplasty without stenting. Patients with the steal syndrome underwent SA embolization using coils. In one patient with a steal syndrome, the arterial anastomosis of the graft was damaged during selective angiography, so an emergency relaparotomy was performed to stop bleeding from the anastomosis, followed by the arterial artery ligation. During the follow-up period, no repeated episodes of decreased arterial blood supply were seen.

Table 3. Treatment of arterial complications

Treatment option	**LGAT**	**LGAS**	**Steal syndrome**
Open surgery, n			
SA ligation			1
Endovascular correction			
Balloon angioplasty		3	
Stenting	1		
SA embolization			2

Biliary complications occurred in 4 patients with arterial complications: one patient with HA thrombosis, two with the steal syndrome, and one with graft artery stenosis (57.1%). In all cases, a bile leakage was observed, and no biliary strictures occurred (Table 4). In one patient with biliodigestive anastomosis, puncture drainage of bile extravasation was performed under ultrasound guidance. The second patient with biliobiliary anastomosis had a stent placed using endoscopic retrograde cholangiography (ERCP), and thus the bile leak was stopped. In the remaining patients, bile leakage developed while the safety drainage tubes were in place and closed spontaneously, without any intervention. For comparison, in patients without arterial complications, bile leakage occurred in 8 cases (25.8%, p=0.039) in the early postoperative period. Also, two patients developed late bile duct strictures: one had an anastomotic stricture of the biliobiliary anastomosis 18 months after transplantation and one had an anastomotic stricture of the biliodigestive anastomosis 12 months after transplantation (Table 4).

Table 4. Biliary complications in patients with arterial complications

Types of complications	Values, n (%)
LGAT	
Bile leak	1 (2.9%)
Anastomotic stricture	–
LGAS	
Bile leak	1 (2.9%)
Anastomotic stricture	–
Steal syndrome	
Bile leak	2 (5.8%)
Anastomotic stricture	–
No vascular complications	
Bile leakage	8 (22.9%)
Anastomotic stricture (late)	2 (5.8%)

A comparison of the incidence of arterial complications in the groups is given in Table. 5. Thus, in patients with ligated SA, vascular complications included thrombosis of the graft artery in one case (3.3%), stenosis of the graft artery in three cases (10%), and thrombosis of the portal vein in 1 (3.3%) case. Bile leakage developed in 10 patients, and one was diagnosed with a late anastomotic biliary stricture. In patients with non-ligated SA, the splenic artery steal syndrome developed in 60% of cases. No other arterial complications were seen. Among biliary complications, the bile leakage was observed in 2 patients (40%), and a late anastomotic stricture was observed in one (20%).

Table 5. Comparison of complication rates in two groups of patients

Complication	Ligated SA, n=30	Non-ligated SA, n=5	p
Steal syndrome	0	3 (60%)	<0.01
Graft artery thrombosis	1 (3.3%)	0	1
Graft artery stenosis	3 (10%)	0	1
Portal vein thrombosis	1 (3.3%)	0	1
Abscess (necrosis) of the spleen	0	0	2
Bile leak	10 (33.3%)	2 (40%)	0.1
Anastomotic stricture	1 (3.3%)	1 (20%)	0.105

During the follow-up period, 3 patients who had arterial complications died. In all cases, the cause of death was not related to arterial disorders. One patient with LGAS (with ligated SA) developed COVID-19-associated pneumonia 2 months after liver transplantation and died from its complications. One patient with the steal syndrome (with non-ligated SA) died of aspiration at home one month after discharge. Another patient with the steal syndrome (with non-ligated SA) died from ovarian apoplexy (sepsis) that was undiagnosed at a local out-patient medical facility. In-hospital mortality in patients after liver

transplantation was 11.5% (n=4). The causes of death were a graft dysfunction secondary to acute portal vein thrombosis (1 patient), a primary graft non-function (1 patient), and sepsis (2 patients). Overall survival and survival rates in groups with arterial complications are presented in Fig. 3 and 4.

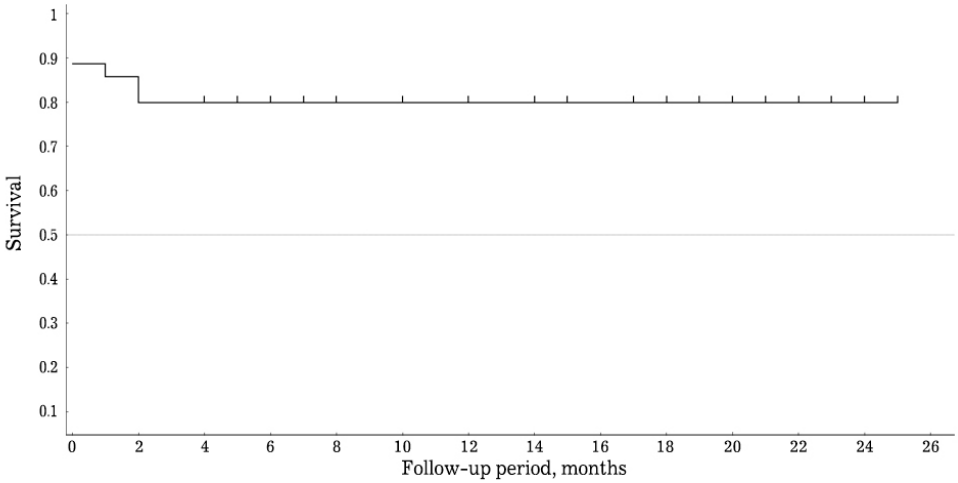

Fig. 3. Overall survival of patients after liver transplantation

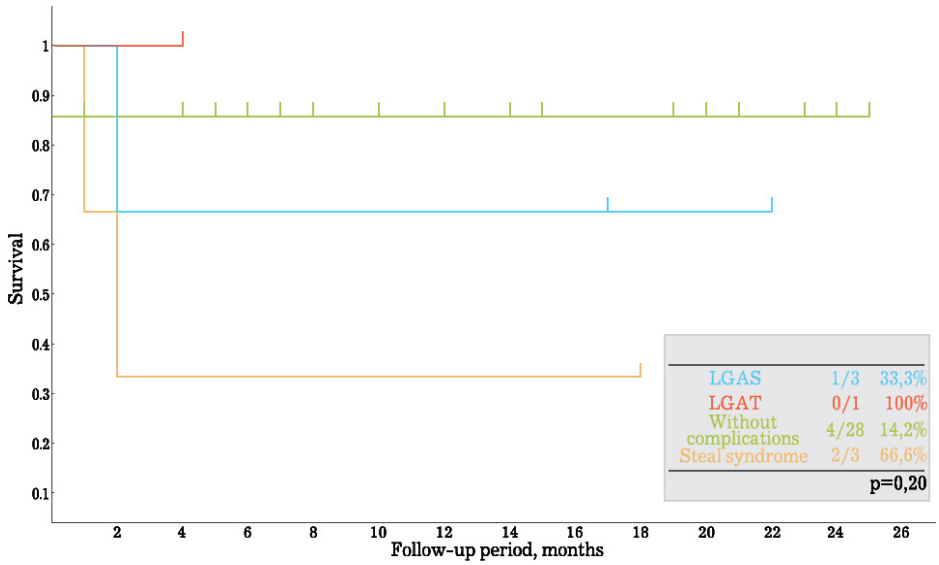

Fig. 4. Survival of patients in groups of vascular complications

LGAT, liver graft artery thrombosis; LGAS, liver graft artery stenosis

As seen from Fig. 3 and 4, the overall survival was 80% at 25 months of follow-up. The survival rate was 66.7% in the group of patients with liver graft artery stenosis, and 33.3% in the group of patients with

the steal syndrome. No deaths were observed among patients with liver graft arterial thrombosis. The survival rate of patients without arterial complications was 85.7%.

Discussion

Adequate arterial inflow is one of the key factors determining the liver graft function [22, 23]. In living donor liver transplant recipients, vascular complications develop more frequently due to a complex vascular reconstruction, a small vessel diameter, and the vessel diameter mismatch [24–26]. The surgical technique, intraoperative and postoperative ultrasound monitoring, and appropriate postoperative thromboprophylaxis are critical to prevent occlusive and non-occlusive arterial complications [4, 12, 22]. The SA steal syndrome is a rare but severe complication after related liver transplantation. It is characterized by hypoperfusion of the hepatic artery due to concurrent shunting of the blood flow into the splenic artery from the celiac trunk. The steal syndrome can cause elevated blood levels of liver enzymes, cholestasis, hepatic artery thrombosis, and even a graft loss in some severe cases. However, the steal syndrome and its prevention are often lacking due attention [4, 27, 28].

A SA diameter exceeding 5 mm and/or 1.5 times exceeding the HA diameter may be a risk factor for the development of a steal syndrome [14]. In our approach, we defined the risk factor for the development of this complication as a difference in the diameters of the SA and HA of 50% or more. All patients in our study did not have a SA diameter less than 5.2 mm. We believe that it is necessary to routinely ligate the SA in all cases where preoperative examination reveals risk factors for the development of the steal syndrome, since neither our study, nor other studies have reported the steal syndrome development after the SA

ligation. We did not notice significant complications from the spleen after it, either. When ligating the SA in the area of the splenic hilum, the graft artery stenosis occurred in all of our patients, but we believe that that was a statistical inaccuracy due to a small sample of such patients. In addition, we believe that the SA ligation in the area of the splenic hilum may be ineffective, since only one of the collaterals only will be ligated rather than the entire blood supplying vessel. Also, the development of steal syndrome is not affected by the surgical technique of arterial anastomosis ($p>0.5$).

Also, when comparing the groups of patients (see Table 5), we obtained statistically significant differences in the results between SA ligation and non-ligated SA ($p<0.01$) in relation to the development of the steal syndrome. We attribute this to several factors that are described in the literature. Thus, none of the patients with a ligated SA developed the steal syndrome. A significant contribution to the steal syndrome development is made by splenomegaly and enlarged splenic vessels amid with portal hypertension in patients with liver cirrhosis [4, 14, 19]. Also, the ratio of graft weight to recipient weight in living related donor transplantations is often lower compared to that in transplantations from posthumous donors, which may be another risk factor for the steal syndrome development [4, 18]. With SA ligation, all of the above risk factors for the steal syndrome development are leveled, which is confirmed by our results and the results of other studies [14–17]. At the same time, the incidence of other arterial complications did not differ statistically significantly with regard to SA ligation. The same applies to biliary complications.

Based on our experience, we determine not only the risk of the small-for-size syndrome and portal hyperperfusion syndrome, but also the development of the steal syndrome with a GRWR of less than 1.0% [4, 18, 19]; so we also consider an adequate donor selection to be a priority

task, especially in recipients with severe portal hypertension. We define ultrasound monitoring as the method of choice for timely diagnosis and identifying indications to an intervention when vascular complications are detected. Each surgeon on duty in our department performs Doppler ultrasound and, if visualization difficulties arise, reports this to the operating surgeon to determine further tactics. In our opinion, a qualified ultrasound diagnostic specialist can suspect the development of a steal syndrome and make a differential diagnosis only based on ultrasound signs, namely a decrease in the arterial blood flow velocity and difficulties in visualizing the arteries (Fig. 5), without waiting for clinical manifestations. It is very important to begin treatment immediately after the diagnosis has been confirmed [29, 30].

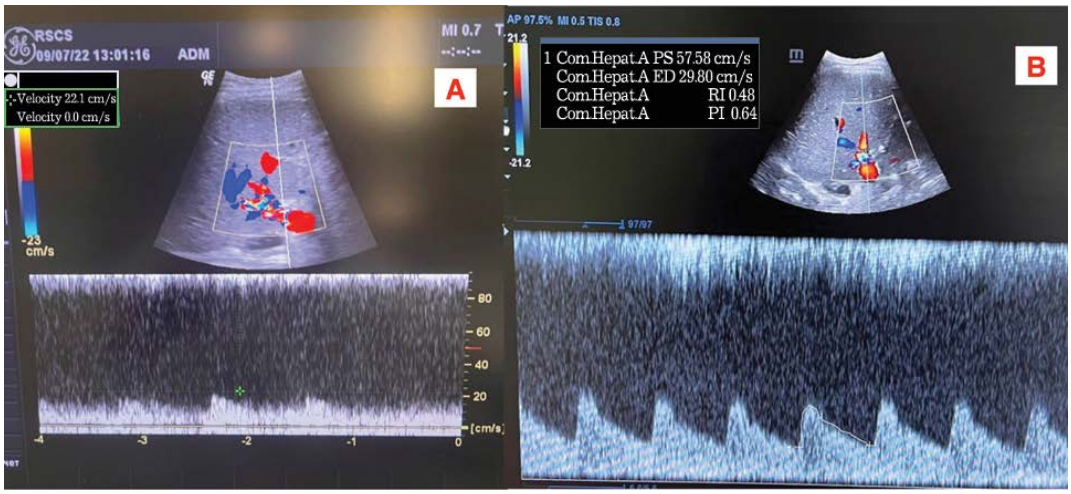

Fig. 5. Doppler ultrasonogram. A. A decreased resistance index, decreased visualization of the hepatic artery in a patient with the steal syndrome in the postoperative period. B. Arterial flow characteristics of the same patient after splenic artery embolization

We also compared the results of our work in SA ligation for the prevention of the steal syndrome after liver transplantation with the results of similar works by foreign authors; the comparison is presented

in Table 6. We obtained the results similar to the study by J.Y. Song et al. [14]. Meanwhile, in the studies of other authors, the percentage of the steal syndrome development in unligated SA was lower [15–17]. At the same time, the study of M. Wojcicki et al. demonstrated that the mean pressure in the HA system in the group of patients with ligated SA is significantly higher than in that without the SA ligation; so, according to the authors, an increase in the HA system pressure may reduce the risk of the steal syndrome occurrence [15]. We also believe that in our study we obtained a higher percentage of the steal syndrome in patients with non-ligated SA due to the fact that we performed only related donor transplantations where the risk of arterial complications is generally higher, the diameter of the graft vessels is smaller, and the risk of portal hyperperfusion is higher, and, in addition, a lower GRWR coefficient makes it contribution (when compared to cadaveric grafts).

Table 6. Compared results of the splenic artery ligation to prevent the development of steal syndrome in various transplant centers

Author	Cases, n	Ligated SA, n	Non-ligated SA, n	Steal syndrome in patients with ligated SA, n	Steal syndrome in patients with non-ligated SA, n (%)	Splenic ischemia, n (%)
J.Y. Song et al [14]	43	28	15	0	5 (33.3%)	1 (6.7%)
M. Wojcicki et al [15]	99	7	92	0	1 (1.1%)	0
M.T. Mogl et al [16]	504	98	406	0	26 (6.4%)	2 (2%)
N.C. Nussler et al [17]	1171	97	1153	0	44 (3.8%)	1 (1%)
Our study	35	30	5	0	3 (60%)	0

Conclusion

Based on the analysis of our own experience and analysis of literature data, the splenic artery ligation appears to be an effective and safe method for the prevention of the splenic artery steal syndrome in patients after living related donor transplantation of the liver right lobe

with a minimal risk of ischemic complications for the spleen; however, further studies with larger sample size and comparison groups are required for obtaining more reliable results. The main tools for an early detection of impairments and a rapid restoration of blood flow through the liver graft artery are the ultrasound examination and endovascular intervention, respectively.

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