



Graft revascularization after a hepatic artery rupture inflicted by a stent

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Abstract

Introduction. *Graft revascularization in deficient arterial blood flow is usually performed by X-ray endovascular stenting procedure and/or by reconstruction of vascular anastomosis. The most serious complication of catheter intervention is a hepatic artery rupture and the critical ischemia of the liver graft.*

Objective. *To discuss the successful treatment of a hepatic artery rupture which occurred during X-ray endovascular revascularization of the liver graft.*

Case Report. *A 48-year-old female patient with advanced alveococcosis underwent orthotopic cadaveric liver transplantation. After restoring the blood flow, it was found to be of low-velocity with RI 0.4 in the common*

hepatic artery. The gastrointestinal duodenal and splenic arteries were ligated. On the second day, a relaparotomy was performed because of intraabdominal bleeding originated from the parenchyma of the 7th–8th segments. Bleeding was arrested. After 18 hours, a selective angiography revealed stenosis up to 90% in the native hepatic artery. After balloon predilatation, stenting was performed, which accidentally caused the artery rupture. The further treatment included relaparotomy and reanastomosing. The patient was discharged from hospital on Day 19 after transplantation.

Conclusion. Low velocity blood flow in the hepatic artery did not meet an adequate level even after the ligation of the gastrointestinal duodenal and splenic arteries. We performed the reconstruction of anastomosis. Another possible approach could be a catheter revascularization. The decision should be made considering the specific disorders of regional blood flow, their origin, and the vascular anatomy. The rupture of the artery in the reported case was caused by disproportionate diameters of the stent and vessel.

Keywords: hepatic artery, transplant, stenosis, X-ray endovascular stenting, rupture, reanastomosis

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ALT, alanine aminotransferase

AST, aspartate aminotransferase

BBT, blood biochemistry test

CBC, complete blood count

GDA, gastroduodenal artery

HA, hepatic artery

INR, international normalized ratio
MSCT, multislice spiral computed tomography
RI, resistance index
SA, splenic artery
USDG, Doppler ultrasound imaging

Introduction

Stenosis of the graft hepatic artery (HA) occurs in 4–10% of cases and, without timely correction, leads to the loss of the liver graft [1, 2]. It is diagnosed by duplex ultrasound scanning in the presence of the following findings: a decrease in the resistance index (RI) <0.5 , an increase in the peak blood flow velocity >400 cm/s or a decrease <20 cm/s, as well as a typical blood flow pattern (“tardus parvus”) [2, 3]. Prevention of vascular complications, their timely diagnosis and rational treatment tactics improve early and long-term results [4].

L. E. Goldsmith et al. described 106 cases of a catheter graft revascularization for HA stenosis, in 8 of which complications developed: an intima dissection (5) and arterial rupture (3). Open surgery was not required; complications were controlled by stenting [3]. N. Rostambeigi et al. conducted a meta-analysis of 147 balloon angioplasties and 116 HA stentings after liver transplantation; complications were recorded in 16% and 19%, respectively [5].

D.I. Jurlevich et al. performed 46 endovascular interventions for stenosis of the graft hepatic artery, including its occlusion (10). In one case, the procedure was complicated by a vessel rupture. It was possible to restore blood flow and stop bleeding by implanting a stent [6].

In the period from 2019 to 2023, in the Irkutsk Regional Clinical Hospital, 8 patients (10.9%) of 73 underwent endovascular intervention for insufficient arterial blood flow of the liver graft (Table 1). Complications occurred in 1 case (12.5%).

Table 1. Endovascular interventions in arterial ischemia of the graft

Complication of transplantation	Number, n	Correction method	Result
Anastomotic stenosis	4	4 stenting + 2 occlusions of the gastroduodenal artery	Recovery in 3 cases Arterial rupture in 1
Stenosis + steal syndrome	1	Stenting + occlusion of the splenic artery	Recovery
Artery thrombosis	1	Thrombosuction + stenting	Recovery
Left hepatic artery stenosis	1	Stenting	Recovery
Intima dissection (intraoperatively)	1	Stenting	Recovery

The objective was to discuss a successful treatment of hepatic artery rupture occurred during x-ray endovascular liver graft revascularization.

Case Report

The female patient aged 48 years had the diagnosis of liver alveococcosis, obstructive jaundice, moderate liver failure. Concomitant pathology included type 2 diabetes mellitus, hypertension disease; chronic heart failure with satisfactory ejection fraction (52%), stage 1, functional class I; unspecified cytomegalovirus infection in the active phase; encephalopathy of combined origin, compensation; moderate cognitive impairments.

Since January 2023, jaundice rapidly increased, a loss of body weight was noted (16 kg); the body mass index was 25 kg/m². She was admitted at hospital on March 29, 2023, and was included on the waiting list for liver transplantation. The condition was severe.

Laboratory test results. Complete blood count (CBC): leukocytes $9.22 \times 10^9/\text{L}$, erythrocytes $4.37 \times 10^{12}/\text{L}$, hemoglobin 125 g/L, platelets

472×10⁹/L. Biochemical blood test (BAC): total bilirubin 328.3 µmol/L, direct bilirubin 233.8 µmol/L, alanine aminotransferase (ALT) 72 IU/L, aspartate aminotransferase (AST) 95 IU/L, alkaline phosphatase 814 IU/L, cholesterol 14.2 mmol/L. Enzyme immunoassay: positive for a-HBeAg (IgG), a-HBcorAg (IgM, IgG); negative for HbSAg, a-HCVIgM, IgG. Coagulogram: international normalized ratio (INR) 1.03, activated prothrombin time (APTT) 34.7 s, prothrombin time (PTT) 10.8 s, fibrinogen 8.02.

Ultrasound examination suggested a focal mass of increased echogenicity of 8.9×5.3×6.8 cm in size in the right lobe of the liver, heterogeneous structure with a liquid component; blood flow was not visualizable. The segmental bile ducts of the right lobe were dilated to 3–4 mm.

Magnetic resonance imaging showed a mass formation of 50×145×116 mm with small cysts in segments 2, 4–8, liquid inclusion of 30×35 mm; compression of the right posterior and anterior sectoral ducts, lobar, 6th, 7th, and 4th segmental ducts, and the left lobar duct; dilation of segmental and subsegmental ducts up to 6 mm, that of left lobar ducts up to 10 mm; common bile duct defect up to 34 mm long.

Multislice spiral computed tomography (MSCT) suggested a hypodensity mass formation of 160×154×153 mm with calcification in the right lobe of the liver; without contrast accumulation. Arterial anatomy without specific features. Stenosis of the inferior vena cava to 6 mm over the length of 24 mm, that of the right hepatic vena cava to 4.6 mm, the middle vena cava to 6 mm, the left vena cava to 6.7 mm.

On April, 13, the patient underwent surgery: hepatectomy with the resection of the inferior vena cava, orthotopic cadaveric liver transplantation (Fig. 1).



Fig. 1. Macroscopic specimen. The right lobe of the liver. Subtotal lesion

Arterial anatomy of the graft: HIATT 3. The right HA was anastomosed with the gastroduodenal artery (GDA) (Fig. 2). The total graft HA was up to 5 mm, the native HA was up to 6 mm, the end-to-end anastomosis with prolene 6/0 suture.

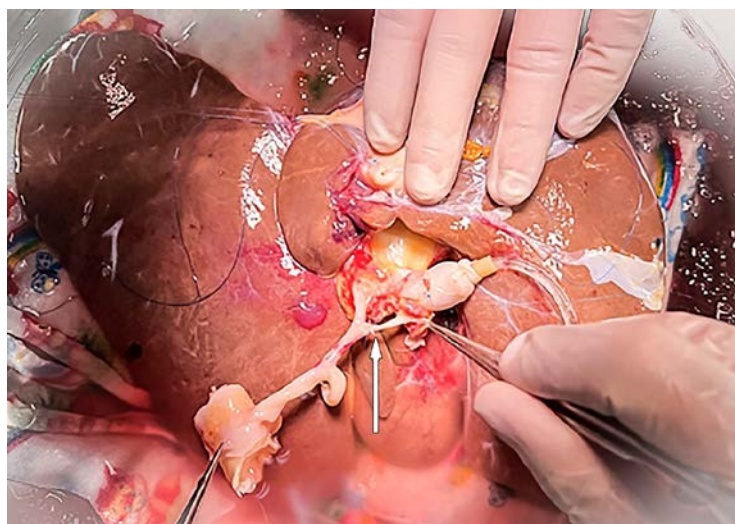


Fig. 2. Intraoperative photo. Preparation of the graft (back-table). Anastomosis between the right hepatic and gastroduodenal arteries (indicated by arrow)

Intraoperative Doppler ultrasonography (USDG) after restoring venous and arterial blood flow demonstrated its low velocity in the common HA, RI 0.41. Owing to the ligation of GDA and splenic artery (SA), the velocity parameters improved slightly. Meanwhile, the deformation of the HA persisted due to the GDA compression. A hepatico-hepatic anastomosis was formed. The surgery duration was 4 hours 35 minutes, cold ischemia time was 2 hours 20 minutes, warm ischemia time was 40 minutes; the blood loss made 1000 mL.

Sodium enoxaparin 0.4 ml twice a day subcutaneously was administered. CBC showed the following: red blood cells $3.26 \times 10^{12}/L$ hemoglobin 90 g/L. Biochemistry: ALT 328 IU/L, AST 548 IU/L, total bilirubin 307 $\mu\text{mol}/L$; lactate 3.2 mmol/L; INR 1.4.

After 24 hours the parameters were as follows: red blood cells $3.67 \times 10^{12}/L$, hemoglobin 111 g/L; total bilirubin 183 $\mu\text{mol}/L$; transaminases: ALT 716 IU/L, AST 1238 IU/L; lactate 2.9 mmol/L; INR 1.1.

On the 2nd day, anemia progressed and transaminase activities increased.

Doppler ultrasonography: blood flow velocity in the portal vein was 65 cm/s, peak in the HA was 90 cm/s, RI was 0.48. MSCT suggested the following: liver with defects along the diaphragmatic surface (S 7-8), there were contents up to 64 mm wide there and also in the hilum (Fig. 3). Short stenosis of HA up to 70%.

Conclusion: a graft rupture; intra-abdominal bleeding; hemodynamically significant HA stenosis.



Fig. 3. Multislice spiral computed tomography angiogram. Rupture of the 7th-8th segments of the liver. Massive hematoma under the right hemidiaphragm (1)

On April 15, 2023 relaparotomy was performed. There were up to 1000 ml of clots in the abdominal cavity. Rupture of the 7th-8th segments of the liver. HA pulsation was clearly detected.

The hematoma was removed. Damage to the Glissonian capsule of the 7th-8th segments was detected. The bleeding did not continue. Cleansing, drainage of the subdiaphragmatic and subhepatic spaces.

Transaminase activity increased, lactate was 1.6 mmol/L, INR 1.5 (Fig. 4). Ticagrelor, 180 mg, was prescribed.

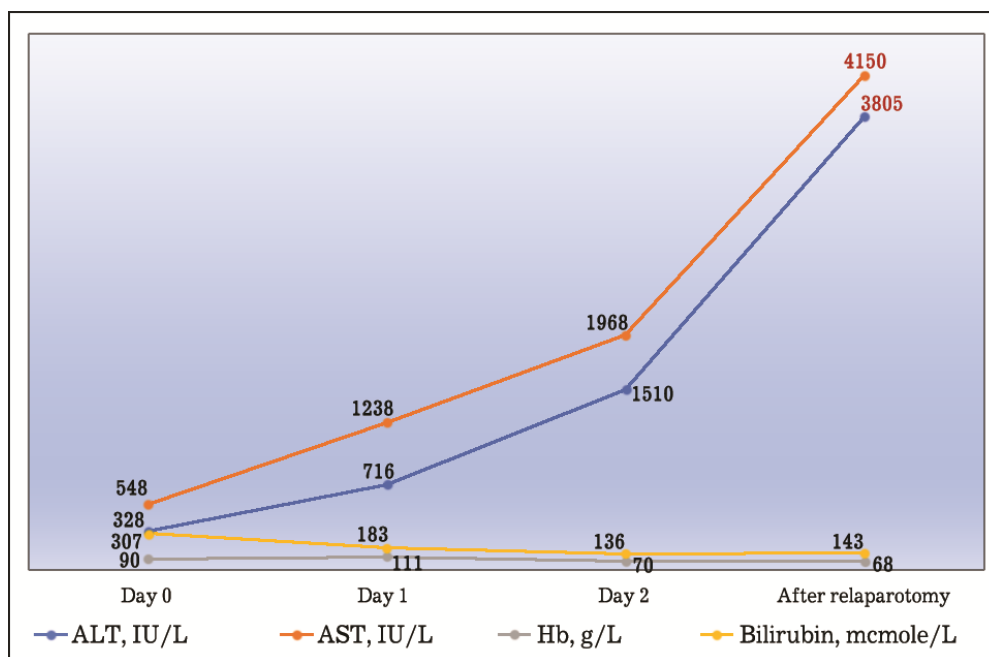


Fig. 4. Postoperative changes in laboratory parameters over time

Selective angiography on April 16 showed the following: stenosis of the native HA up to 90% (Fig. 5). Peripheral blood flow was preserved. Predilation of HA with a Blade balloon (NPK EVIPRO, Russia) (3.25–20), Hippocampus stent (Invatec S.p.A., Italy) (7.0–20) was installed at a pressure of 8 atm.



Fig. 5. Selective angiogram. Anastomotic stenosis

The control angiogram showed massive extravasation (Fig. 6). The balloon was inflated in the common HA, bleeding did not continue.



Fig. 6. Selective angiogram of the hepatic artery. Escape of contrasted blood into the abdominal cavity (indicated by arrows)

On April, 16, relaparotomy was performed. There was up to 650 ml of fresh blood in the abdominal cavity. Stream bleeding continued. Resection of the graft stented native HA (Fig. 7), reanastomosis.



Fig. 7. Macroscopic specimen. Segment of the hepatic artery with a wall defect (1); stent (indicated by arrows)

After surgery, the arterial blood flow of the liver graft normalized, and transaminases quickly decreased. Doppler ultrasonography On April 16, 2023 showed the flow velocity in the portal vein being 40 cm/s, peak velocity in the artery being 40 cm/s; RI was 0.6.

The patient was discharged from hospital on May 2, 2023 (19th day after transplantation). CBC was as follows: red blood cells $3.42 \times 10^{12}/L$, hemoglobin 109 g/L. Biochemistry: bilirubin 46 $\mu\text{mol}/L$, ALT 32 IU/L, AST 17 IU/L; INR 1.5. Doppler ultrasound showed blood flow velocity in the portal vein being 48 cm/s, peak velocity in the artery being 95 cm/s; RI was 0.67.

Discussion

Anastomosis of the liver graft and recipient vessels is a key stage of surgery. Tactical and technical inaccuracies, such as anastomotic stenosis, intima dissection, kinking, cause graft ischemia. Any doubt about the sufficiency of blood flow (at USDG) is a reason for revision and reconstruction of the anastomosis.

In the Case Report under discussion, there was a low-velocity blood flow in the common HA (RI 0.4). After the GDA and SA ligation, the parameters did not reach optimal values, which was an indication basis for reanastomosis that was not made.

The indication for emergency laparotomy on day 2 was bleeding from the 7th–8th segments of the graft. A possible cause was damage to the parenchyma during explantation, with a subcapsular hematoma that emptied into the abdominal cavity.

The probable cause of HA rupture during endovascular X-ray intervention was inappropriate stent dimensions for HA diameter and a high pressure (8 atmospheres) against the background of intima dissection with a balloon. L.E. Goldsmith et al. and J.P. Vairavamurthy et

al. identified the following risks of endovascular intervention: vessel tortuosity (kinking) and re-transplantation [3, 7]. A significant bending of the vessel also occurred in the case under discussion.

The decision not to reconstruct the anastomosis and to perform endovascular revascularization after surgery was controversial (rather incorrect as the results showed). Ischemia of the liver graft continued for another day, and transaminase activity increased. A successful resection of the stented vessel was performed and the anastomosis was made. Stenosis was the result of HA deformation caused by the fixed GDA. Reconstruction of arteria was indicated in the first operation.

Endovascular correction of arterial rupture, as described by D.I. Yurlevich and L.E. Goldsmith [3, 6], in the presented Case Report was unpromising due to the misfit between the stent diameter and the artery lumen, especially since profuse intra-abdominal bleeding had developed.

Conclusion

In case of low-velocity blood flow in the common hepatic artery of the graft (RI 0.4), which does not reach optimal values after ligation of the gastroduodenal and splenic arteries, the reconstruction of the anastomosis is indicated.

Reanastomosis should have been performed after relaparotomy for liver graft rupture. During endovascular revascularization, the diameter of the stent should fit to the artery lumen. With a rigid stricture, an excessive increase in pressure (> 4 atmospheres) may cause the vessel rupture, and the reconstruction of the anastomosis has to have been performed urgently.

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