Vascular complications in liver transplantation: a pictorial review

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Authors: C. E. B#la#a\textsuperscript{1}, S. Bivol\textsuperscript{2}, N. Leo\textsuperscript{2}, N. Dahbi\textsuperscript{2}, Y. Ajavon\textsuperscript{2}, S. AWAD\textsuperscript{2}, A. Elmaleh\textsuperscript{2}, Z. Dhina-Louison\textsuperscript{2}, M. Lewin\textsuperscript{2}, \textsuperscript{1}Craiova/RO, \textsuperscript{2}Villejuif/FR
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Learning objectives

To review the spectrum of vascular complications of liver transplantation
To illustrate such complications in a variety of anastomotic techniques
To focus on the treatment options available
Background

The indications of liver transplantation have been constantly expanding beyond acute or chronic end-stage disease to also cover malignancy such as hepatocellular carcinoma. Often occurring in the early postoperative period, the procedure’s vascular complications lack specific clinical manifestations and include arterial thrombosis or stenosis, arterial pseudoaneurysms followed in a decreasing order of frequency by portal, inferior vena cava or hepatic vein thrombosis or stenosis.
Findings and procedure details

We retrospectively analyzed the doppler ultrasound, CT and angiographic imaging aspects in a series of patients having vascular complications after liver transplantation for various conditions during 2008 to 2013.

The screening and follow-up protocol was based on a combination of daily Doppler duplex ultrasonography throughout the first week and a systematic CT angiography on the seventh day posttransplantation, followed by weekly ultrasounds during the first month. Most vascular complications develop within the first 3 months and should be suspected based on liver function alteration.

Considered the most frequent vascular complication, arterial thrombosis has a reported incidence of 2-12% and is generally suspected based on the absence of intrahepatic arterial flow and confirmed either by CTA or conventional angiography[1]. Known risk factors include significant caliber disparity ( ), previous stenotic lesions of the celiac axis or its branches (Fig. 2 on page 6), the interposition of a graft, multiple arterial anastomoses, acute rejection and prolonged cold ischemia time[2]. Patients present with altered liver function, cholangitis, biliary leakage or rejection.

Its most feared consequence is biliary necrosis and abscess formation, as the main blood supply to the biliary tree is arterial. (Fig. 3 on page 7 and Fig. 4 on page 7)

As previously reported in various studies[1,2], arterial stenoses were also generally diagnosed as early complications in our series, usually at or in close proximity to the anastomotic site (Fig. 5 on page 7, Fig. 6 on page 8, Fig. 7 on page 9, Fig. 8 on page 10), their prevalence seeming to be higher in living donor liver transplantation (Fig. 9 on page 11, Fig. 10 on page 12, Fig. 11 on page 13, Fig. 12 on page 14)[3]. Their diagnosis was based on the association between a value of the resistive index inferior to 0.5 and an acceleration time of over 0.08s[4].

Although surgical revision is sometimes necessary, the preferred treatment options are interventional consisting in balloon angioplasty and stenting, sometimes requiring repeated successful procedures. (Fig. 13 on page 15, Fig. 14 on page 16, Fig. 15 on page 17, Fig. 16 on page 18, Fig. 17 on page 19)[5].

Arterioportal fistulas are frequent findings related to early percutaneous biopsies to rule out rejection. (Fig. 18 on page 20 and Fig. 19 on page 21)

Pseudoaneurysms may present an intra- or extrahepatic location. Although considered rare, pseudoaneurysms should be feared due to their risk of rupture and hemorrhage or
thrombosis. Treatment options include resection, coil embolization or stent placement. ([Fig. 20 on page 22 and Fig. 21 on page 23 ] [1,2,5])

One of the rarer complications proper to our team’s experience was represented by a posttherapeutic arterial dissection occurring during an attempted angioplasty for a stenosis ([Fig. 22 on page 24 and Fig. 25 ]).

Anastomotic leakages are also considered amongst the potential vascular complications and may lead to arterial bleeding ([Fig. 24 on page 26 ]) or more often venous hemorrhage ([Fig. 25 on page 27 ]).

While portal vein complications are much less common, they should be constantly searched owing to the risk of graft loss. There was only one portal thrombosis in our series, atypically located in an intrahepatic segment ([Fig. 26 on page 28]). Portal venous stenosis was seldom encountered and related to caliber differences between the donor and the receiver, history of prior thrombosis and coagulopathies. It should be distinguished from external compression and is characterized by vessel narrowing, elevated peak anastomotic velocities or anastomotic to preanastomotic velocity ratios along with portal hypertension manifestations ([Fig. 27 on page 29, Fig. 28 on page 30, Fig. 29 on page 31, Fig. 30 on page 32 ]) [4]. Treatment options include endoluminal angioplasty, with stenting being reserved for cases resistant to dilatation.

Since venous complications are also highly infrequent, we only encountered one right hepatic venous stenosis ([Fig. 31 on page 32 ]).

There were no inferior vena cava stenoses or thromboses in the series analyzed.
Fig. 1: Retransplantation in a patient suffering from Rendu Osler disease displaying an important arterial caliber disproportion and a partial thrombosis of the proper hepatic artery of the receiver as well as a plicature of the donor proper hepatic artery

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Fig. 2: Male patient having benefitted from a transplant two years and a half previously for hepatocellular carcinoma MIP reconstructions demonstrating a complete thrombosis of the common hepatic artery stent with collateral revascularization of the intrahepatic arterial branches via the gastroduodenal arcade.

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Fig. 3: Female patient transplanted 18 years previously for an autoimmune cirrhosis MIP reconstructions presenting a complete thrombosis of the proper hepatic artery accompanied by a partial thrombosis of the common hepatic artery as well as multiple abscesses.

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Fig. 4: Same patient as in Fig.3 Portal phase CT scan illustrating parietal biliary enhancement and multiple abscesses compatible with ischemic cholangitis.

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**Fig. 5:** Patient suffering from alcoholic cirrhosis-stenosis-related decreased arterial resistance index estimated at 0.36 at the level of the right branch of the proper hepatic artery

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Fig. 6: Same patient as in Fig. 5- the arterial resistance index was normal at the level of the hepatic pedicle, estimated at 0.56

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Fig. 7: Same patient as in Fig.5 and 6- stenosis-related decreased arterial resistance index evaluated at 0.41 at the level of the left branch of the proper hepatic artery

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**Fig. 8:** Same patient as in Fig. 5, 6 and 7 suffering from alcoholic cirrhosis-MIP reconstructions demonstrating an early 6 mm long stenosis of the proper hepatic artery

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Fig. 9: Male patient suffering from alcoholic cirrhosis and two HCC nodules having benefitted from a right split graft presenting with a late 5mm long stenosis of the proper hepatic artery

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Fig. 10: Digital subtraction angiography aspect of the stenosis before angioplasty in the same patient as in Fig.9

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**Fig. 11:** Same patient as in Fig.9 and 10 Digital subtraction angiography aspect of the stenosis after angioplasty and stenting

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**Fig. 12:** Male recipient of a right split graft donated by the son developing an early severe stenosis of the proper hepatic artery

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Fig. 13: 15 year old suffering from biliary atresia and secondary biliary cirrhosis, having also received a right split liver, showing a low arterial resistance index with a value of 0.37 at the level of the right hepatic branch caused by a stenosis

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Fig. 14: Same patient as in Fig.13 Aspects after balloon angioplasty and stenting

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Fig. 15: Digital subtraction angiography in the same patient as in Fig.13 and 14. Aspects after successful balloon angioplasty and stenting

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Fig. 16: Digital subtraction angiography in a 14 year-old patient 2 months posttransplantation with a severe stenosis of the initial proper hepatic artery immediately after the emergence of the gastroduodenal artery.

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Fig. 17: Successful stent placement in the patient shown in Fig. 16. The gastroduodenal branch was spared by the stent due to the associated presence of an arcuate ligament. The patient subsequently developed another late intrastent stenosis.

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Fig. 18: Arterioportal fistula between the right proper hepatic artery branch (white arrow) and the anterior sectorial right portal branch (black arrow)

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Fig. 19: Perfusion anomalies related to the presence of the arterio-portal fistula

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Fig. 20: 18/8 mm juxtaanastomotic arterial pseudoaneurysm in a patient having benefitted from a double liver and renal transplantation

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**Fig. 21:** 25/11mm pseudoaneurysm in close contact to the right hepatic branch

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Fig. 22: Digital subtraction angiography of a severe initial proper hepatic arterial stenosis aspect prior to a failed angioplasty attempt

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Fig. 23: Hepatic artery thrombosis and aneurysm formation after failed angioplasty attempt

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Fig. 24: Digital subtraction angiography images of an early posttransplant arterial leakage originating in a branch stemming from the juxtaanastomotic proper hepatic artery (white arrows). This small branch was subsequently embolized with the aid of Bead Block microparticles and an Azur coil (not shown).

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Fig. 25: Active venous extravasation in close contact with the inferior vena cava (black arrows), leading to a subcapsular segment VI hematoma and requiring reintervention in a patient having received his son's right liver.

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**Fig. 26:** Thrombosis of the proximal part of the right hepatic branch in a male recipient transplanted for alcoholic liver cirrhosis presenting with elevated liver enzymes

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Fig. 27: Portal anastomotic stenosis with poststenotic dilatation

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Fig. 28: Portal anastomotic stenosis with poststenotic dilatation. Mean circulatory speed in the proximal segment is estimated at 58 cm/s

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**Fig. 29:** Portal anastomotic stenosis with poststenotic dilatation. Mean circulatory speed in the distal non-dilated segment was evaluated at approximately 30 cm/s

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**Fig. 30:** 13 year-old female biliary atresia recipient demonstrating an anastomotic portal stenosis with proximal dilatation, altered liver function and signs of portal hypertension

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**Fig. 31:** MRI: Portal phase postgadolinium injection—one of the very rare cases of a stenosis of the right hepatic vein secondary to an adjacent collection in a patient transplanted three years previously for acute alcoholic hepatitis

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Conclusion

Conclusions

Vascular complications continue to represent the second most important cause of graft failure and should therefore be rapidly identified in order to prevent graft loss and retransplantation. Imaging plays a vital role in the early recognition of this diagnosis and in the therapeutic management.
References


5. James C. Andrews Vascular Complications Following Liver