Role of interventional radiology in the treatment of complications following liver transplantations: Results of our experience

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Purpose

Liver transplantation is widely accepted as the gold-standard treatment of advanced chronic liver disease, acute liver failure, and of some metabolic liver disorders. Today, transplant patients have a survival rate of approximately 75%, nevertheless, significant complications still occur [1,2]. Furthermore, in the most recent surgical techniques, such as split liver transplant, living donor transplantation or auxiliary transplantation, the incidence of vascular and biliary complications increases with the surgical complexity [3]. The time period associated with the highest risk of mortality is the first year after transplantation particularly in the first 3 months due to graft dysfunction, technical problems due to vascular and/or biliary complications and infections. In a context of organ shortage, interventional radiology has become first line approach in management of vascular and biliary complications, in order to avoid re-transplantation or to provide a bridge until a suitable donor is available.

In this study we report our experience, to evaluate the efficacy and safety of percutaneous treatment of vascular and biliary complications following liver transplantation.
Methods and Materials

From December 2002 to June 2009 111 Patients underwent 114 liver transplantations (OLT). During this period, other 2 Patients transplanted in another Center were referred to ours for the onset of some complications: they decided to continue follow up in our Center, so that we considered them in this work.

Ultrasonography (US), including Doppler analysis, was performed in all patients as the primary Imaging modality in the immediate post-operative period, as well as in the subsequent follow up. Cross-sectional Imaging with CT or MRI and/or cholangiography were required to further evaluate any suspected complications whenever detected increase in blood chemistry indices of biliary stasis (serum bilirubin, AF, gGT, GTP-GOT), development of a septic state (pyrexia, leukocytosis), altered findings on abdominal examination, leakage of bile from surgical drains, recurrent cholangitis or abnormal findings on abdominal US examination. Angiography was performed only in case in which a vascular complication was highly suspected, for definite diagnosis and endovascular treatment.

During the follow up complications were observed in 32/113 Patients. Among them, 21 Patients developed vascular and/or biliary complications (Tables 1 and 2), while other 11 Patients developed complications (1 pulmonary embolia, 6 fluid collections, 4 persistent ascites). We focused on vascular and biliary complications, as they are related to a higher risk of morbility and mortality and susceptible to radiological treatment.

Exclusion Criteria

4/21 (19%) Patients affected by vascular and/or biliary complications were not included in this study: 1/22 underwent re-transplantation immediately after diagnosis of acute portal vein thrombosis. In 3/21 a mild anastomotic biliary stenosis was radiologically demonstrated, but Patients were asymptomatic with normal serum enzymes, so that a conservative approach was preferred.

Inclusion Criteria

Our study included 17/21 (81%) Patients (9 men, 8 women; mean age: 57 years; age range 39-72) who were referred to our interventional radiologic unit with a clinical and Imaging picture of vascular and/or biliary complications.

Each case had been previously discussed in a multidisciplinary surgical-radiologic meeting. Percutaneous procedures were attempted in alternative to surgical repair, to avoid a precocious re-transplantation or to provide a bridge until a suitable donor is available.

Arterial complications treatment
We always follow the same standard protocol to diagnose any hepatic artery lesion. Through a trans-femoral percutaneous arterial access, after percutaneous local injection of anesthesia (lidocaine 2%), we place a 5F introducer sheath and a 5F J curve catheter for selective angiography of the superior mesenteric artery and the celiac trunk first, with a 30-ml bolus injection of contrast medium at 5 ml/s. Delayed images are taken in the venous phase to study the porto-systemic veins. The same 5F J Curve catheter and a 0.035 J hydrophilic guide wire are used for superselective catheterization of the common hepatic artery, with a 12-30 ml bolus injection of contrast medium at 3-5 ml/s to better evaluate the extent of stenosis or of the thrombus in distal branches. Once a hepatic artery stenosis or occlusion is demonstrated, a 3F microcatheter ad a 0.016 guide wire are advanced through the 5F catheter, in order to cross through the lesion and consequently attempt PTA or stenting placement. Balloon and stents derived from the cardiological field allow to treat even complex lesions such as dissection, occlusion or stenoses occurring in very tortuous vessels (Figure 1).

Portal vein complications treatment

We follow a percutaneous approach with a direct percutaneous puncture of a portal branch in the liver or, in alternative, a transjugular/transfemoral venous approach. The right percutaneous approach is preferred. A 5F introducer sheath and a 5F vertebral catheter are advanced through the portal branch and selective catheterization of the portal vein is performed, with a 12-30 ml bolus injection of contrast medium at 3-5 ml/s to better evaluate the extent of portal lesion and to demonstrated any varices responsible of possible persistent porto-systemic shunt. We cross the portal lesion with a 0.035 J guide wire and PTA is performed with a balloon inflated on the stenotic site, after pressure measurement across the stenosis. If portography is satisfactory after the procedure or pressure gradient is below 5 mm/Hg, the procedure is finished (Figure 2). If there is a recoil or if the pressure gradient is higher than 5 mm/Hg, a stent is implanted across the stenosis. If a significant port-systemic shunt is demonstrated, a 5F catheter is advanced into the varices and coils embolization is performed. The procedure is complete when there is non evidence of flow into collaterals. Once treatment is complete, the intraparenchymal tract is embolized with coils.

Biliary stenoses treatment

All procedures are performed after local percutaneous injection of anesthesia (lidocaine 2) and intravenous administration of hyoscine N-butylbromide (60 mg) (Buscopan, Boehringer Ingelheim, Italy) given to relax the smooth muscle of the biliary Ducts. In some selective patients we recur to a monitored anesthesia care, with mild intravenous sedation (diazepam and fentanil). We use a multistep approach (Figure 3): a 7 Fr external biliary drainage catheter or internal-external catheter is inserted into the biliary tree after cholangiography obtained either using the surgical T-tube or by percutaneous transhepatic access. After 3-5 days, various guidewires and different types of 5 Fr catheters (Angled Taper or Cobra or Shepherd Hook, Terumo, Tokyo, Japan)
are used and a balloon dilatation catheter placed across the stenosis and inflated (10-30 sec in the same treatment) until the stenosis disappeared. Balloons 4-8 mm in diameter are used for dilatation of strictures extending into secondary bile ducts and a 8-18 mm balloon is used for the dilatation of the perianastomotic stenoses. An internal-external biliary drainage catheter (8-10 Fr) is inserted for a period ranging from 3 to 90 days after bilioplasty. Cholangiography are routinely performed to assess the result of the stricture dilatation. Secondary bilioplasties, if considered necessary, are performed at regular intervals of 20-30 days. Removable stent placement is proposed in case of recurrent stenosis of common hepatic duct. Percutaneous treatment is concluded after patency assessment of the bile ducts and removal of the biliary drainage catheter.

Both technical and clinical success of radiological treatments were evaluated. Technical success was considered as feasibility of procedure, while clinical success as freedom from disease at least at 6 months follow up. In case of biliary strictures, as complete treatment often require several months, clinical success was considered as a significant improvement in clinical and laboratory assessment associated to partial improvement in biliary morphology and during follow-up. All Patients also underwent US Doppler and/or MR to study vascular and biliary morphology after treatment.
Table 1. Vascular complications

<table>
<thead>
<tr>
<th>Complications</th>
<th>On-set during follow up</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Post-operative (within 30 days)</td>
<td>Late</td>
</tr>
<tr>
<td>Portal stenosis</td>
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<td>1</td>
</tr>
<tr>
<td>Hepatic artery stenosis</td>
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</tr>
<tr>
<td>Persistent portal-systemic shunt</td>
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<td>1</td>
</tr>
<tr>
<td>Portal thrombosis</td>
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<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

* Patient underwent liver re-transplantation

**Fig. 0:** Table 1: Vascular Complications

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**Table 2. Biliary complications**

<table>
<thead>
<tr>
<th>Complications</th>
<th>On-set during follow up</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Post-operative (within 30 days)</td>
<td>Late</td>
</tr>
<tr>
<td>Anastomotic stenosis</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Anastomotic leakage</td>
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<td>-</td>
</tr>
<tr>
<td>ITBL</td>
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<tr>
<td>Ischemic strictures</td>
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<td>2</td>
</tr>
<tr>
<td>Immunological stenosis</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>5</td>
<td>9</td>
</tr>
</tbody>
</table>

* 3 of them were mild stenoses without any clinical and laboratory evidence: no treatment was required.

**Fig. 0:** Table 2: Biliary complications

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Fig. 0: Figure 1: Stenosis of hepatic artery: diagnosis (a-b) and angiographical control after treatment (c-d). Superselective catheterism of hepatic common artery in two different projections (a-b) highlights a severe perianastomotic stenosis of hepatic artery. A microcatheter is advanced to cross through the arterial stenosis and PTA is performed with a balloon derived from the cardiological field. After treatment (c- d) no significant stenosis is demonstrated.

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Fig. 0: Figure 2: Portal stenosis. a) Portography before treatment confirms the diagnosis of severe anastomotic stenosis. b) We proceed to PTA, inflating balloon at the stenosis site for 60-80 seconds. At the end of procedure a residual stenosis of 45% is demonstrated (c). As pressure gradient was less than 5 mmHg, the procedure was considered concluded.

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**Fig. 0:** Figure 3: Percutaneous treatment of ITBL secondary to immunological causes: multiples procedures in recurrent disease. At percutaneous first choloangiography multiple extensive intrahepatic, hilar and perianastomotic strictures, are without a significant dilatasion of biliary intrahepatic ducts. The type as well as the site of lesions are typical signs of ITBL. b-f) Multiple treatments are performed during 21 months of follow up, as restenosis is quite common. Multiple bilioplasties are firstly attempted at the perianostomotic site and at the biliary confluence, obtaining only a partial resolution of strictures (b). So, a removable stent is placed to treat the perianastomotic stenosis (c). After 8 months, the stent is removed and cholangiography demonstrates a good morphological result with a normal caliber of choledocus. Anyway, recurrent stenoses are still seen at the origin of left hepatic duct, at its confluence with the right one and in the proximal choledocus (d). So another bilioplasty is performed (e). At last cholangiography, 21 months after the first treatment, a significant improvement in biliary tree is highlighted: there is only a recurrent tight stenosis into the III segmental duct.

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Results

6/17 (35.3%) Patients were treated for vascular complications: 1 hepatic artery stenosis, 2 portal vein stenoses and 3 persistent porto-systemic shunts. Detailed endovascular treatments are resumed in Table 3.

It is worth noticing that in a Patient treated for porto-systemic shunt a concomitant portal stenosis was demonstrated at portography (Figure 4): US Color Doppler misdiagnosed it as a portal thrombosis, as no flow had been demonstrated in portal vein.

Portal stenosis was treated only by PTA in a Patient with a 70% stenosis (Figure 2), while in a Patient affected by subocclusive stenosis (80-90% of stenosis), stenting was necessary to achieve permanent patency.

Endovascular procedure was feasible in all Patients (technical success 100%).

At 6 month follow up in a Patient a recurrent portal stenosis was demostrated at Imaging (Figure 5). Overall clinical success was obtained in 5/6 Patients (83.3%).

11/17 (64.7%) Patients underwent treatment for biliary complications: 8 ischaemic type biliary stenoses (ITBL), 2 anastomotic strictures and 1 peri-anastomotic leakage. In 3/8 Patients with ITBL, an hepatic artery complete occlusion was demonstrated by angiography: in 2 out of 3 several tiny collateral branches for the liver had been already developed, so that we decided not to proceide to endovascular. In the other Patient biliary lesions were demonstrated in the follow up despite successful surgical revascularization of the acute arterial thrombosis (Figure 6).

Detailed endovascular treatments are resumed in Table 4.

Percutanous approach was feasible in 10/11 Patients (technical success 91%). In a Patient with ITBL strictures percutaneous biliary access was failed twice, due to multiple peripheral biliary serpiginous stenoses; even after achievement of a percutanous access bilioplasty was extremely difficult and precocious restenosis was observed, despite several treatments. Biliary perianastomotic leakage was successfully treated placing a percutaneous endoprotesis. Both anastomotic biliary stenoses were treated by bilioplasty followed by the placement of a percutaneous removable stent at 3 months follow up (Figure 7).

ITBL required multiple bilioplasties (mean 6: range 4-10 percutaneous bilioplasties) to achieve a good clinical and morphological result, due to high incidence of recurrences. As a consequence, all these Patients had a longer follow up (at least 1 year).

At 6 months follow up, all recurrences were observed in Patients with severe ITBL: 1 had to undergo liver re-transplantation, while 2 of them died for septycal and
systemic complications (Clinical success 63.6%). Multiple percutaneous approaches were performed to achieve this result.
### Table 3. Vascular complications

#### Endovascular treatment

<table>
<thead>
<tr>
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<th>Portal stenosis</th>
<th>Persistent portal-systemic shunt</th>
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<td>PTA</td>
<td>1</td>
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<td>-</td>
<td>2</td>
</tr>
<tr>
<td>PTA + stenting</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Embolization</td>
<td>-</td>
<td>-</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>PTA + stenting + coil embolization</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>

**Recurrences (at 6 months)**

<table>
<thead>
<tr>
<th></th>
<th>0/1</th>
<th>1/2</th>
<th>0/3</th>
<th>1/6</th>
</tr>
</thead>
</table>

**Fig. 0:** Table 3: Endovascular treatment of vascular complications

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Fig. 0: Figure 4: Porto-systemic shunt and portal anastomotic stenosis: diagnosis and treatment. a-b) Through left percutaneous access intrahepatic right portal vein is selectively catheterized (a). 5F catheter is advanced into the portal vein at the hylum, a huge and a huge collateral venous cardio-frenical branche is highlighted, responsible of a porto-systemic shunt (b). No flow is demonstrated into the portal vein at the hylum, suggestive of a stenosis associated to hemodynamic portal liver hypoperfusion. c) After a partial embolization with coils of venous collateral, portal anastomotic stenosis is confirmed. d-f) After complete embolization of varices, balloon PTA of portal stenosis (d) is performed, obtaining a residual stenosis of more than 25% (e), so that a stent has to be placed. At the end of procedure no residual portal stenosis is demonstrated, as well as no flow into the collateral venous branches (f)

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Fig. 0: Figure 5. Recurrence of portal stenosis at Imaging follow up at 6 months (same Patients figure 2) At Color Doppler US Imaging there is evidence of turbulence in the flow at the anastomotic site and a significant increase in vascular velocity at this point, highly suggestive of re-stenosis. b-d) RM coronal TSE T2 (b) and post-gadolinium (c) images, as well as MIP reconstructions (d) better demonstrate the site and the extent of re-stenosis of 80%.

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Fig. 0: Figure 6: Ischemic ITBL strictures after surgical revascularization of acute hepatic artery thrombosis. a-b) Angiography 24 hours after transplantation demonstrates acute hepatic artery thrombosis, immediately after its origin. Despite successful surgical revascularization, the Patient developed during the first month of follow up diffused intrahepatic biliary lesions, characterized by irregular and serpiginous dilatation of intrahepatic ducts, multiple stenoses, more or less associated to sludge and desquamation debris into their lumen. CWRM (c) and cholangiography (d) are comparable in demonstration of the disease. e-f) A multistep percutaneous approach is performed in this Patient in the next 2 years of follow up: comparison between the first treatment (20/04/07) and last cholangiography through a naso-biliary drainage (03/04/09) demonstrates almost complete regularization of biliary tree is demonstrated.

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### Table 4. Biliary complications

#### Percutaneous treatment

<table>
<thead>
<tr>
<th></th>
<th>ITBL</th>
<th>Anastomotic stenosis</th>
<th>Leakage</th>
<th>Total</th>
</tr>
</thead>
<tbody>
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<td></td>
<td>Ischaemic strictures</td>
<td>Immunological stenoses</td>
<td></td>
<td></td>
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<tr>
<td>Bilioplasty</td>
<td>3</td>
<td>3</td>
<td>-</td>
<td>6</td>
</tr>
<tr>
<td>Endoprotesis</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Removable stent</td>
<td>-</td>
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<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Technical failure</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>11</td>
</tr>
</tbody>
</table>

|                  | 0/3  | 3/5                  | 0/2     | 0/1   | 3/11 |
| Clinical unsUCCESS (at 6 months) |       |                      |         |       |

**Fig. 0:** Table 4: Radiological treatment of biliary complications

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Fig. 0: Figure 7: Treatment of anastomotic biliary stenosis. a) At first cholangiography intrahepatic duct are enlarged and they immediately stopped at the proximal common biliary duct, while the recipient choledocus has a normal caliber. After bilioplasty, a removable stent is placed (b). After its removal, at 6 months follow up, anastomotic stenosis is disappeared without any residual stenosis.

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Conclusion

Discussion

Despite improvements in organ preservation technology, refinements in procurement and surgical techniques, and advances in immunosuppressive strategies, vascular and biliary tract complications remain a significant cause of morbidity and mortality after liver transplant. Di Martini et al. reported in a review of over 4000 transplantations actuarial 1-, 5-, and 10-year survival rates were 79, 67, and 57%, respectively [4]. So, early detection of post-operative complications is essential for graft and patient survival. Clinical signs of complications often are non-specific, so that diagnosis is frequently are based on imaging findings [5].

Vascular complications, with an overall incidence of 9%, are a primary diagnostic consideration in a liver transplant recipient [5]. In our series the incidence of vascular complications was a little better (6%) than that reported in Literature. They commonly occur early in the post-transplant period, although significant complications may be clinically silent [2].

The patency of hepatic arterial vessels are of crucial importance after liver transplantation as the biliary tree is fed only by the hepatic artery. Arterial complications can be divided into early arterial thrombosis occurring in the first month after transplantation, late arterial thrombosis occurring after this delay, hepatic artery stenosis, and steal syndrome from the splenic or gastroduodenal artery [3]. In literature it is reported a rate of hepatic artery thrombosis of 6.8%, one-third in the first month after transplantation and two-thirds after this delay [6]. A similar amount of hepatic artery stenosis is reported [7]. Angiography is the gold standard for diagnosis arterial complications, but CT and MR angiography are increasingly used in these circumstances. Doppler US in our experience was the primary screening modality: every suspicion was confirmed with CT and/or angiography. Endovascular fibrinolytic therapy followed by balloon angioplasty or stent placement is reported in literature for treatment of hepatic artery thrombosis [2,3,8], but we have no experience of it. We diagnosed acute hepatic artery thrombosis only in a Patient and he undergo immediate surgical revascularization (Figure 6 a-b).

Treatment of hepatic artery stenosis is more debated because a higher number of patients are asymptomatic at the moment of the diagnosis. Arguments for the treatment are numerous: it has been reported that stenosis, if not treated, may evolve to hepatic thrombosis and be related to a significant increase of biliary complications, known to reduce life and graft expectancy [7]. Furthermore, there is evidence of clinical improvement and blood chemistry indices amelioration after stenosis correction [3,9,10]. In our experience we performed a successful ballon angioplasty, without any recurrence at 6 months follow up (Figure 1). We think it should be the first choice treatment: stent
placement should be considered only in case of a residual stenosis of more than 20-25% or of an intimal dissection, due the high risk of intra-stent occlusion.

Hepatic artery steal syndrome from the splenic or gastro-duodenal artery is a more recently studied entity. It is defined by a decreased perfusion in the hepatic artery due to a diverted blood flow either in a large splenic or gastroduodenal artery. While it is found in 5.9% of patients, its role in the liver ischemia is still debated. Furthermore, its treatment remains complex, with a high rate of complications after splenic ligation or embolization [11].

Portal vein complications are relatively rare and include thrombosis and stenosis. Portal vein thrombosis occurs in about 1%-2% of cases. Portal vein stenosis has a reported incidence of 1% after liver transplantation [5]. They are usually secondary to surgical techniques, vessel length and caliber discrepancies, previous portal vein surgery or previous thrombosis, increased downstream resistance due to a suprahepatic stricture of the inferior vena cava, decreased portal inflow, and hypercoagulable states. Patients may be asymptomatic or develop portal venous hypertension, hepatic failure and ascites. US Color Doppler, CT and MR angiography provide excellent depiction of filling defects and focal narrowing of the portal vein (Figure 5). Transhepatic or transjugular portography should be performed only in case of treatment, which includes balloon angioplasty, thrombolysis and stenting.

Persistent porto-systemic shunt is usually considered as a "minor" complication. It is often under-diagnosed or misdiagnosed at US Color Doppler, but it can lead to a significant liver portal hypoperfusion if not treated. According to the results of our experience embolization of collateral varices is an effective procedure: results are seen immediately after embolization, with a sudden increase in portal perfusion.

Overall results in endovascular treatment of vascular complications in our experience are extremely promising. All procedures were performed without any complications, and only a Patient experienced a recurrent portal stenosis, even if he was completely clinically asymptomatic. We think a high rate of technical and clinical success can be achieved only with a precocious diagnosis and treatment. According to our experience, radiological treatment should be performed whenever there is a clinical or Imaging suspicion of vascular complications. Surgical revascularization or re-transplant should be considered as a second-line choice, in case of endovascular failure: as a matter of fact, graft loss is a serious problem, because of the complexity of the surgical procedures and the shortage of livers available for transplantation [8].

Biliary complications remain a common source of morbidity and mortality after liver transplantation, so that it is defined the technical "Achilles heel". Despite a dramatically reduction of such complications related to improvements in organ selection, retrieval, preservation and implantation techniques, they still occur in 10-30% following liver transplant, resulting in a mortality rate up of 10% of cases [12].
Biliary leaks and strictures are the most common biliary complications, but sphincter of Oddi dysfunction, hemobilia, biliary obstruction from stones, sludge or casts have been reported too. The vast majority appear within the first 3 months. Approximately half of them are involving only the anastomotic site and consist of bile leaks and anastomotic strictures [3]. Virtually, all these early complications have a technical origin related to an ischemic necrosis of the end of the bile duct or a technically unsatisfactory anastomosis. Stenosis are usually treated with percutaneous drainage and balloon biliopasty followed by long-term drainage, either endoscopic or percutaneous, with frequent changes of drains or stents to avoid silent occlusions. A long term success rate of more than 50-70% has been reported in literature for such strictures [12], which was confirmed in our experience. Management of leaks requires antibiotherapy, bilioma drainage, and biliary drainage. Biliary leaks related to a T tube or to a cystic duct are managed successfully from a percutaneous or endoscopic approach in most cases, as in our experience in which a percutaneous endoprothesis was placed. When larger anastomotic leaks related to biliary necrosis at the anastomotic site occur, non-operative management can fail and a surgical approach may be required [3].

Late biliary complications may appear months or years after transplantation. They are due to arterial insufficiency or immunological reactions which cause ischemic type lesions; they have a much worse prognosis as they are typically chronic problems that tends to persist or recur after temporizing measures [3]. Because the mechanism (ischemic, cytomegalovirus infection, long cold ischemia exceeding 12 h) causes diffuse lesions of the biliary tree, extensive intrahepatic, or hilar lesions may be found in that context. ITBL occur in 2-20% of Patients and localized proximal to anastomotic site, showing destroyed intra-hepatic as well as extrahepatic bile ducts more or less associated to sludge and desquamation debris in the lumen of bile ducts [3,12] . Stenosis and debris and sludge removal can be managed percutaneously; extensive use of antibiotics are required during manipulations, because of the risk of biliary infection and septic shock. Radiological treatment is a challenge: recurrence of stenosis is observed in more than 66% of cases, so that multiple percutaneous approaches have to be performed. As it is almost impossible to achieve a complete resolution of radiological pattern of disease, it should be considered a clinical success to avoid liver transplantation and to obtain satisfactory clinical results ( improvement in laboratory biliary indices, significant reduction in number of cholangitis episodes). In our experience percutaneous management of biliary complications reported an overall good clinical success thanks to a multi-step treatment approach. It is worth noticing that better results were achieved in Patients with ITBL due to arterial insufficiency, rather than in those with immunological ITBL: in two Patients we demonstrated an almost complete regularization of biliary tree after percutaneous treatment (Figure 6), while in the other Patient re-transplantation was avoided. All recurrences at follow up were observed in patients with immunological ITBL strictures (Figure 3), suggesting that in such cases percutaneous treatment should be considered palliative.
So, according to our experience and like Righi et al. [13], we believe that percutaneous treatment of biliary complications is the most appropriate choice in many cases, as it is less traumatic than surgery and better accepted by this population of patients. Reoperation and retransplantation should be needed, in the presence of massive biliary leakage or severe and extensive biliary strictures, especially when no improvement is seen after multiple approaches.

Conclusion

Although decrease in mortality and morbidity from liver transplantation, significant post-transplantation complications are still seen. Radiologists play a key role in the post-operative and follow up care of transplant recipients, as a precocious diagnosis and treatment is essential to increase clinical success in treatment. Endovascular treatment of vascular complications has a high clinical and technical success rate and it should be considered as a first-line treatment choice. Percutaneous management of biliary complications has good clinical success but it is required a multi-step treatment approach to achieve better results.
References


