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The Application of Interventional Radiology in Living-Donor Liver Transplantation

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Owing to improvements in surgical techniques and medical care, living-donor liver transplantation has become an established treatment modality in patients with end-stage liver disease. However, various vascular or non-vascular complications may occur during or after transplantation. Herein, we review how interventional radiologic techniques can be used to treat these complications.

Keywords: *Living donor liver transplantation; Portal vein complications; Hepatic vein complications; Hepatic arterial complications; Biliary complications*

INTRODUCTION

Living-donor liver transplantation (LDLT) has become a worthwhile alternative to deceased-donor liver transplantation (DDLT) in Asian countries because of the shortage of deceased organ donations [1]. LDLT has several advantages over DDLT, including a short waiting time, good quality graft, and short ischemic time [1]. However, it does have several disadvantages, such as a small liver graft volume and technical complexity. Compared with those in DDLT, the donor graft vessels and bile ducts in LDLT are smaller and their stumps are shorter. In addition, multiple bile duct openings are not uncommon in LDLT [2].Therefore, various post-transplant vascular and biliary complications are common despite improvements in surgical techniques. Advances in the field of interventional radiology have facilitated the treatment of many of these complications by interventional radiologic techniques. Herein, we describe how interventional radiologic techniques can be used in the treatment of post-transplant complications.

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Portal Vein Complications

Portal Vein Stenosis

Portal vein stenosis (PVS), a complication of the portal vein (PV), is rare in adult DDLT cases; however, it may occur in 2–3% of adult LDLT cases [3,4] and 8–14% of pediatric LDLT cases [5-8]. Technical factors, such as a tight suture line, discrepancy in PV size, tension or torsion of the PV, a redundant PV, or the use of a bypass graft, can contribute to early PVS [9,10]. Furthermore, in the later period, intimal hyperplasia or fibrosis around the anastomosis may induce PVS [11]. Although such complications have traditionally been treated with surgery, interventional techniques, including balloon angioplasty and stent placement, have recently been accepted as the initial treatment of choice [4-6,9].

Interventional procedures for the PV can be performed via percutaneous transhepatic, transsplenic, transjugular, and inferior or superior mesenteric vein access. The access choice depends on variables including preoperative PV status, interval from LDLT, coagulation profile, location and degree of the stenosis, status of the intrahepatic PV and splenic vein, and the operator's preference.

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Transhepatic access is the most commonly used approach [3-5]. However, this route has several drawbacks, such as risk of liver damage, bleeding, and access difficulty in patients with a collapsed PV. Liver damage associated with transhepatic access can lead to deterioration of the liver function in LDLT recipients due to the small liver graft volume. Therefore, transhepatic access should be performed carefully by targeting the peripheral PV branches under ultrasonography and/or fluoroscopy. Transsplenic access is a useful alternative with limited risk of liver damage, especially in patients in the early post-transplant period or in those with a collapsed PV [4,12]. This mode of access is also useful for dual LDLT with bilateral PVS (Fig. 1). However, bleeding complications from the spleen remain a concern [12]. Transjugular access is theoretically suitable for patients with coagulopathy or massive ascites; however, this approach is technically more challenging [13]. PV access via the mesenteric vein is another option to avoid liver damage, but it requires laparotomy. Therefore, this approach is frequently indicated during LDLT or in the early post-transplant period (Fig. 2) [4,5,14-16]. Cannulation of a PVS via the transhepatic access is not difficult with a conventional technique using a 0.035-inch guidewire. If it is unsuccessful, the transsplenic or intraoperative transmesenteric access may be useful [5,17].

"Leave nothing behind" is the main advantage of balloon angioplasty, and this technique is frequently performed prior to stent placement, especially in children. Balloons with the same or about 10–20% larger diameter than that of the pre-stenotic extrahepatic PV are used.

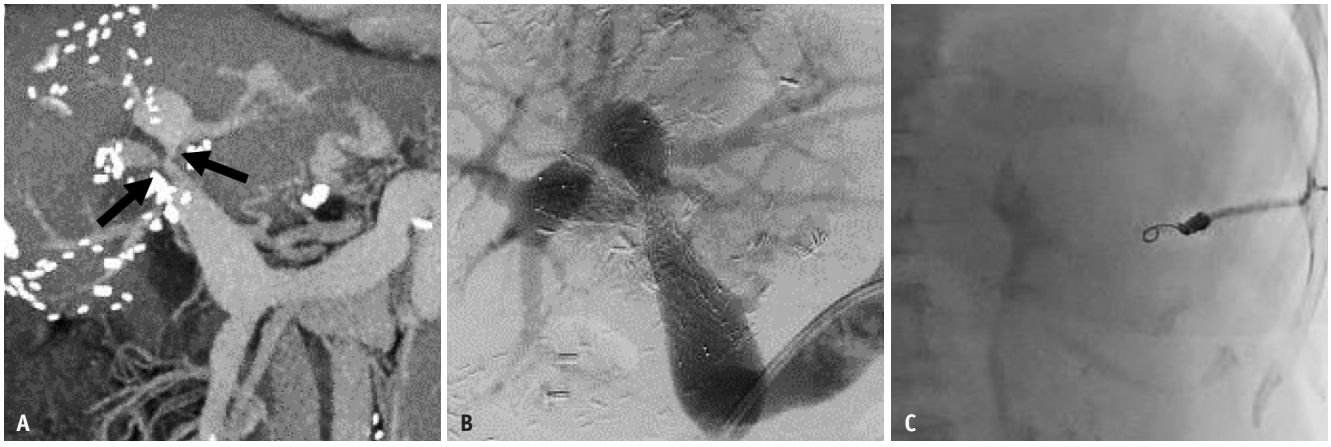

Fig. 1. A 66-year-old woman with stenosis in the main PV bifurcation 12 months after dual left lobes living-donor liver transplantation.
A. CT shows stenosis (arrows) in the main PV bifurcation. **B.** Dual stents are placed in the right- and left-sided PVs via transsplenic access. **C.** Transsplenic route is embolized with coils followed by glue. PV = portal vein

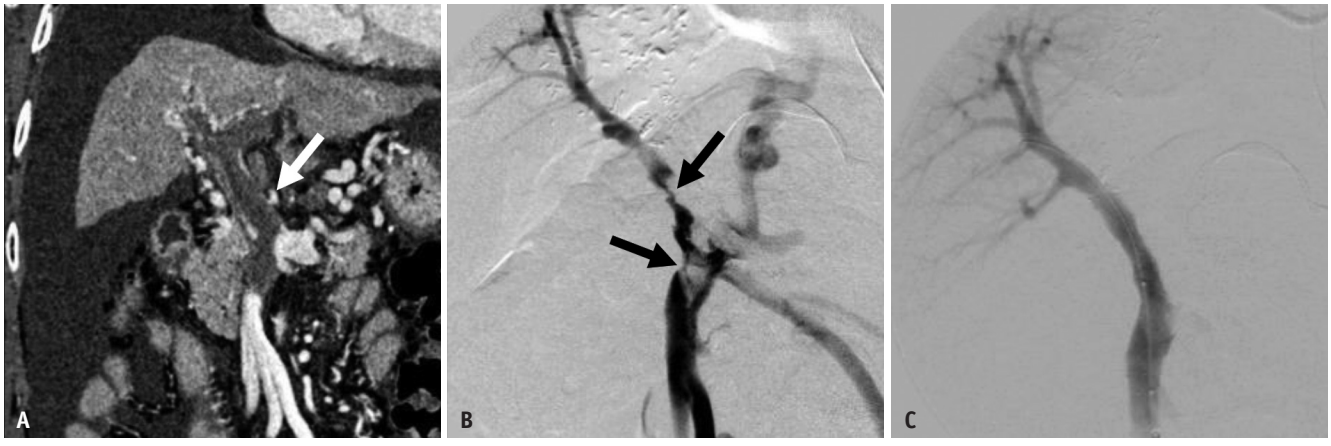

Fig. 2. A 67-year-old man undergoing PV stent placement during living-donor liver transplantation.
A. A pre-transplant CT shows extensive main PV thrombosis (arrow). **B.** Venogram via the superior mesenteric vein after PV thrombectomy and end-to-end PV anastomosis shows diffuse stenosis of the main PV with the remaining thrombi (arrows) in the proximal superior mesenteric vein and main PV. **C.** Post-stenting venogram shows normalized PV with a disappeared coronary varix. PV = portal vein

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However, balloon angioplasty is limited by a high incidence of elastic recoiling or restenosis. The reported recurrence rate with balloon angioplasty in pediatric LDLT ranges from 21–42% [5,8,11,15].

Stent placement is indicated to treat elastic recoiling or recurrence after balloon angioplasty. Primary stent placement is often used during LDLT or in the early (< 1 month) post-transplant period [9,14,18,19]. Stent placement is often performed as the first-line treatment in cases with PV occlusion because cannulation of a PV occlusion can be unsuccessful or very difficult to perform. PV occlusion is a well-known major cause of technical failure of endovascular treatments [4,20,21]. Stent placement generally has a higher technical success rate and primary patency rate than that of balloon angioplasty. Kim et al. [3] reported a 10-year primary patency rate of 100% in 8 adults. Kim et al. [14] reported a technical success rate of 98% and a 2-year primary patency rate of 88% in 36 patients. In pediatrics, several studies have reported 85–100% stent patency rates for various follow-up periods (28–118 months) [15,19,21].

However, stents have several potential drawbacks, including in-stent restenosis, thrombosis, stent fractures, interference with PV anastomosis during re-transplantation, and functional stenosis of the stent in children as they grow [12,15,21].

Therefore, it is important to choose a stent of the most appropriate type, diameter, and length. In adults, a self-expandable stent with the same or a 10–20% larger diameter than that of the pre-stenotic PV is frequently used. Stents with appropriate lengths to cover the stenosis with minimal angulation relative to the PV should be selected. In pediatrics, a balloon-expanding stent ≥ 7–8-mm diameter and a short length is preferred because this type of stent can be positioned accurately, thus minimizing the difficulties of re-transplantation [22,23]. It can also be re-expanded or overdilated in the future if functional stenosis of the fixed stent occurs [24,25]. However, several studies have reported that self-expandable stents ≥ 7–8-mm diameter are also effective in treating pediatric PVS, exhibiting excellent long-term patency [4,5,15,21].

After the procedure, the percutaneous transhepatic or transsplenic route can be embolized using coils, gel-foam, or glue to prevent bleeding. In patients with coagulopathy or those in the early post-transplant period, embolization with coils followed by glue may prove useful (Fig. 1) [4,12]. Post-procedural antiplatelet therapy for ≥ 3 months is recommended following endovascular treatments of PV [11,14]; however, there is no consensus regarding peri- and post-procedural anticoagulation or antiplatelet therapy [26].

Portal Vein Thrombosis

Portal vein thrombosis (PVT) is a rare but serious complication, especially in the early post-transplant period. It usually occurs in the extrahepatic PV and may extend into the intrahepatic PV. Risk factors include pre-transplant PVT, small PV diameter, splenectomy, use of interposition grafts for PV reconstruction, large portosystemic collateral vessels, and PVS [27,28].

Surgical thrombectomy is the initial treatment for early post-transplant PVT because of its clinical urgency and the risk of bleeding during thrombolysis [27-29]. A combination of surgical thrombectomy and endovascular treatments through the mesenteric vein may be useful for further management of the underlying PVS and the remaining PVT or competitive portosystemic collateral veins, which are the usual causes of PVT [14,30].

There is no consensus regarding endovascular treatment of PVT, including that of the access route, thrombolytic agent, dose, and duration. Urokinase has been used as a thrombolytic agent, but recently, tissue plasminogen activators (tPA) have been used instead. Takatsuki et al. [31] reported successful recanalization in all 5 late-onset post-LDLT PVT by continuous systemic intravenous infusion of tPA (Actilyase, Boehringer Ingelheim) at a dose of 0.25 mg/kg/d for 10 days. However, the direct PV approach is preferred because most cases of PVT have underlying PV anastomotic abnormalities such as anastomotic stenosis or kinking.

Small series and case reports have described various combinations of endovascular techniques, including catheter-directed thrombolysis, aspiration or mechanical thrombectomy, mechanical fragmentation, balloon angioplasty, stent placement, and embolization of portosystemic collateral veins [32-38]. Lorenz et al. [33] reported a case of successful recanalization using overnight thrombolysis with tPA at a dose of 0.5 mg/h, followed by mechanical thrombectomy and balloon angioplasty through the transhepatic and transjugular access. Koo et al. [39] reported complete thrombolysis in 10 out of 13 patients (77%) by percutaneous transhepatic and/or transjugular thrombolysis, in which tPA was administered as an initial bolus of 4 mg followed by additional aliquots of up to 20 mg. The tPA was then left to dwell within the

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thrombi for > 10 minutes, and then adjunctive mechanical thrombectomy was applied. Continuous thrombolysis at a rate of 1 mg/h (for patients weighing > 33 kg) or 0.02 mg/kg/h (for patients weighing < 33 kg) was also performed if needed. Thrombolysis via transjugular access may have a lower risk of bleeding than other accesses. Additionally, transjugular intrahepatic portosystemic shunt can be performed through this approach. However, the puncture of a thrombosed PV may be technically challenging, and the management of intrahepatic PVT may be difficult. In such cases, transhepatic or transsplenic access may be useful. Aspiration or mechanical thrombectomy using various devices can reduce the total dose of the thrombolytic agent, bleeding, and procedural time (Fig. 3) [33,37,38]. The potential drawbacks of this procedure are the risks of vessel injury or distal embolization and high cost.

Persistent Portosystemic Collateral Veins

Some of the portosystemic collateral veins disappear after LDLT, but they may be continuous because of the partial liver graft in LDLT. The diversion of the PV flow through these veins may cause hypoperfusion-induced graft failure or PVT [40,41]. There is general agreement that hepatofugal collateral veins should be closed to avoid PV stealing [41]. These veins can be ligated during LDLT; however, endovascular embolization is an effective alternative. An intraoperative approach during LDLT has several advantages over the percutaneous approach, including prompt PV flow evaluation, early embolization before liver dysfunction, and less injury to the liver graft [17,42]. Various embolic agents, including coils, glue, or an Amplatzer vascular plug, can be used for embolization. Balloon-occluded or plug-assisted retrograde transvenous obliteration is also an effective alternative to manage PV stealing via a gastrorenal or splenorenal shunt [43,44].

Hepatic Vein or Inferior Vena Cava Complications

Hepatic vein (HV) stenosis is not uncommon in LDLT, and the reported incidences range from 5–11% [8,45-47]. It is associated with discrepancies in the HV size, a tight suture line, twisting or kinking, and multiple anastomoses in the early post-transplant period [47,48]. Late-onset stenosis is frequently due to peri-anastomotic fibrosis, but it may also be associated with compression due to liver graft edema or regeneration [47,48].

Endovascular treatments are typically performed through the transjugular or transfemoral access. Usually, the cannulation of a target HV by a conventional technique is not difficult. If unsuccessful, a rendezvous technique may be useful (Fig. 4) [49]. In this technique, an occluded HV can be cannulated antegradely via the intrahepatic veno-venous collaterals from other preserved HVs. Following this, a guidewire can be snared in the inferior vena cava (IVC) via another transjugular access. Although transhepatic access may be a useful alternative, it is associated with risks of liver graft damage and bleeding [50,51].

Balloon angioplasty is the first choice for the treatment of HV stenosis and presents a high technical success rate. However, elastic recoiling or recurrence is common, and some patients may require multiple procedures or stent placement [46,50,52,53]. The rate of recurrence with balloon angioplasty in pediatric LDLT ranges from 33–47% [8,46,50,54]. Stent placement is an effective modality for managing elastic recoiling or recurrent stenosis after

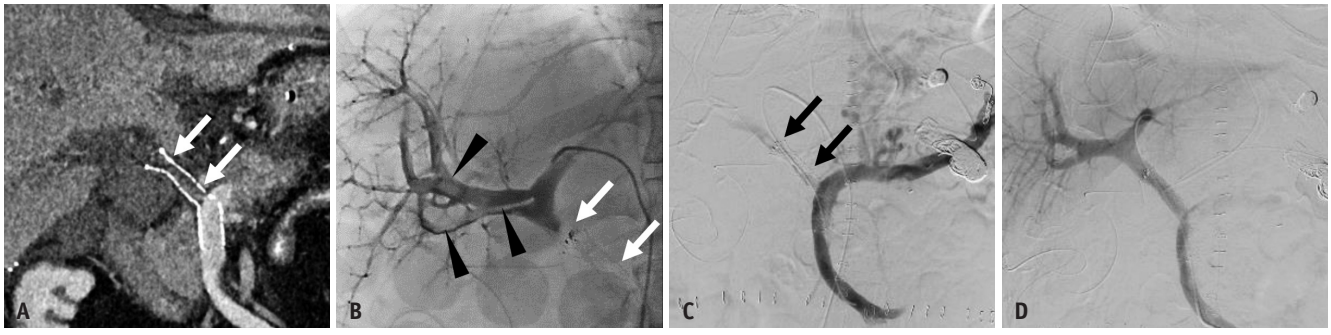

Fig. 3. A 51-year-old man with main and right PV thrombosis 12 days after deceased-donor liver transplantation and intraoperative PV stent placement.
A. CT shows thrombosis in the stent-placed main PV (arrows) and right PV. **B, C.** Venogram via left PV shows diffuse partial thrombosis (arrowheads) in the right PV and thrombotic occlusion (arrows) of the stent-placed main PV. **D.** Completion venogram after mechanical thrombectomy using the Angiojet® thrombectomy catheter shows restored PV flow. PV = portal vein

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balloon angioplasty. Primary stent placement is also useful in the early post-transplant period because HV stenosis in this period is frequently caused by kinking of a redundant HV, twisting by a displaced liver graft, or extrinsic compression due to liver graft edema or regeneration, which are resistant to balloon angioplasty alone [47,55,56]. In addition, there is a risk of disruption of a fresh anastomosis during balloon angioplasty. For stent placement, the length of a stent protruding toward the IVC should be as short as possible to avoid potential IVC stenosis or interference with re-transplantation in the future.

The patency of HV stents is acceptable. Chu et al. [56] reported a 5-year primary patency rate of 94% in 15 patients. Ko et al. [47] reported a 5-year primary patency rate of 72% in 108 patients, and they found that stent diameter was an independent factor associated with stent patency. Jang et al. [55] reported similar outcomes.

There is no consensus regarding whether short HVs (i.e., the right inferior HV or the middle HV tributaries) should be revascularized during right lobe LDLT, because intrahepatic venous collaterals may relieve hepatic congestion in the territory of the ligated or stenotic HVs [57]. However, several authors assumed that revascularization of these HVs might be necessary to avoid small-for-size graft syndrome and to facilitate the restoration of liver function [47,58,59].

In pediatrics, several studies have demonstrated favorable outcomes using balloon-expanding [53,60] or self-expandable stents [46,52]. However, stent placement in pediatric cases should be approached very carefully because of the uncertain long-term patency (Fig. 5).

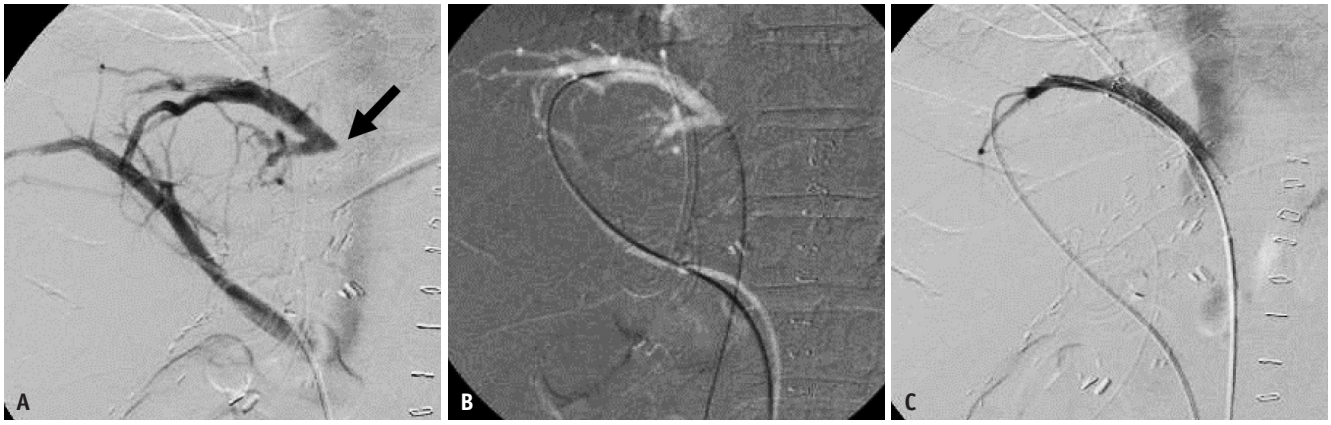

Fig. 4. A 58-year-old man undergoing stent placement using a rendezvous technique 3 days after modified right lobe living-donor liver transplantation.
A. Venogram of the right inferior HV following failed cannulation of the right HV shows the right HV via intrahepatic veno-venous connections but with an occluded proximal right HV (arrow). **B.** The occlusion is passed through the intrahepatic connection using a guidewire from the inferior HV, and the wire is grasped with a snare in the inferior vena cava. **C.** Post-stenting venogram shows brisk HV outflow. HV = hepatic vein

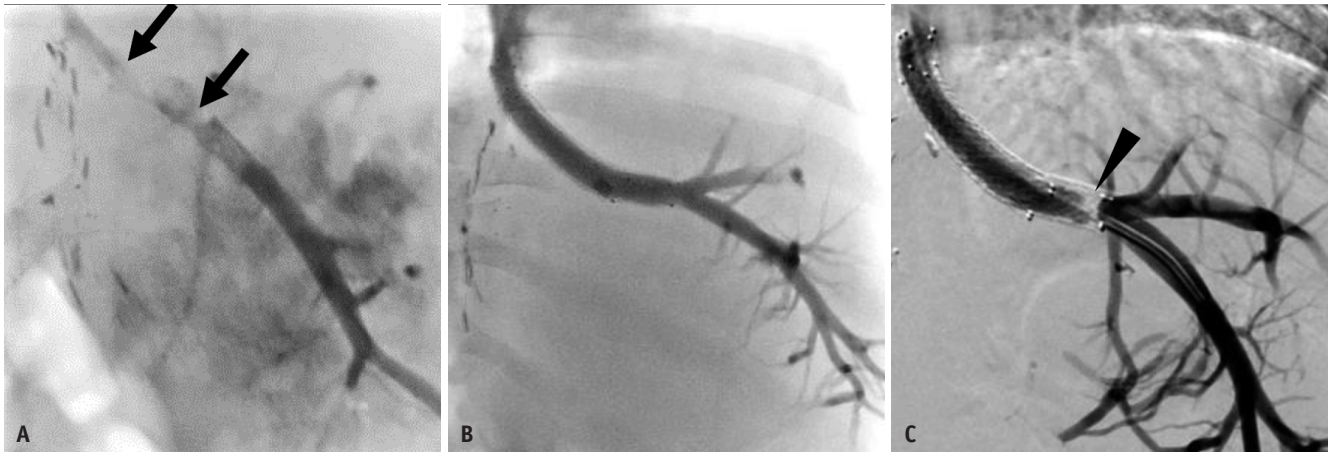

Fig. 5. A 24-month-old girl with HV thrombosis 9 months after lateral segment living-donor liver transplantation.
A. Venogram shows thrombotic occlusion (arrows) of the left HV. **B.** HV outflow is restored after thrombolysis and placement of two stents. **C.** Two-year follow-up venogram shows in-stent restenosis (arrowhead). The patient underwent 12 sessions of repeat balloon angioplasty and/or mechanical atherectomy due to restenosis during a 12-year follow-up. HV = hepatic vein

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Stenosis or occlusion of the IVC is a rare complication. Balloon angioplasty is the first choice in the treatment of IVC stenosis; however, stent placement is frequently necessary because of the inherent IVC elasticity and elastic recoil [45,48,61,62]. IVC stents need to have a high radial strength, barbs to prevent stent migration, large stent diameter, and wide interstices to minimize interference with the HV outflow [62]. Careful evaluation of HV outflow following IVC stent placement is required because of the risk of HV outflow disturbance caused by an IVC stent [61,62].

Hepatic Artery Complications

Hepatic Artery Stenosis

Hepatic artery (HA) stenosis is a potentially devastating complication occurring in the early post-transplant period. Patients with this complication may present with graft dysfunction or biliary complications [48,63-65]. HA stenosis frequently occurs at the anastomosis, and it could be caused by the operative technique, acute cellular rejection, intimal dissection, previous transarterial chemoembolization, or microvascular injury [27]. The most effective treatment is surgical revascularization or re-transplantation; however, repeat surgery can be very difficult because of the presence of severe fibrosis or inflammation around the HA or the lack of an adequate artery for reconstruction. Therefore, endovascular techniques, including balloon angioplasty and stent placement, have emerged as less invasive alternatives to surgical treatments [63-68]. Splenic and/or gastroduodenal artery embolization can also be performed to increase HA flow. Some investigators prefer stent placement to balloon angioplasty because of the former's superior primary patency [66-68]. However, in a meta-analysis of 257 patients in 26 articles, both balloon angioplasty (n = 147) and stent placement (n = 116) showed similar results with respect to procedural success (89% vs. 98%), complications (16% vs. 19%), arterial patency (76% vs. 68%), survival (80% vs. 82%), and re-transplantation (20% vs. 24%) (*p* value was non-significant) [63].

Most of the previous data were from DDLT, as LDLT cases were rare [6,64,69]. In LDLT, the diameters of the HA branches are usually smaller than that in DDLT. Moreover, the HA course is frequently tortuous; hence, negotiation of the stenosis and advancing a balloon catheter or a stent assembly should be performed carefully (Fig. 6). HA dissection or rupture may occur during endovascular treatments, and it is necessary to prepare covered stents for such a complication. Kodama et al. [64] reported encouraging results of balloon angioplasty (2.0–4.5 mm in diameter) in 18 LDLT patients. In their study, the technical success, recurrence, and complication rates were 94%, 33%, and 7%, respectively. There are insufficient data regarding stent patency in LDLT, and stent placement should be reserved for cases with a large HA diameter and straight course. Although there is no consensus, anticoagulation or antiplatelet therapy is frequently administered during or after endovascular treatments [70].

Hepatic Artery Thrombosis

HA thrombosis is the most common cause of graft loss in the early post-transplant period and ischemic biliary complications in the late period [48,71]. Urgent re-

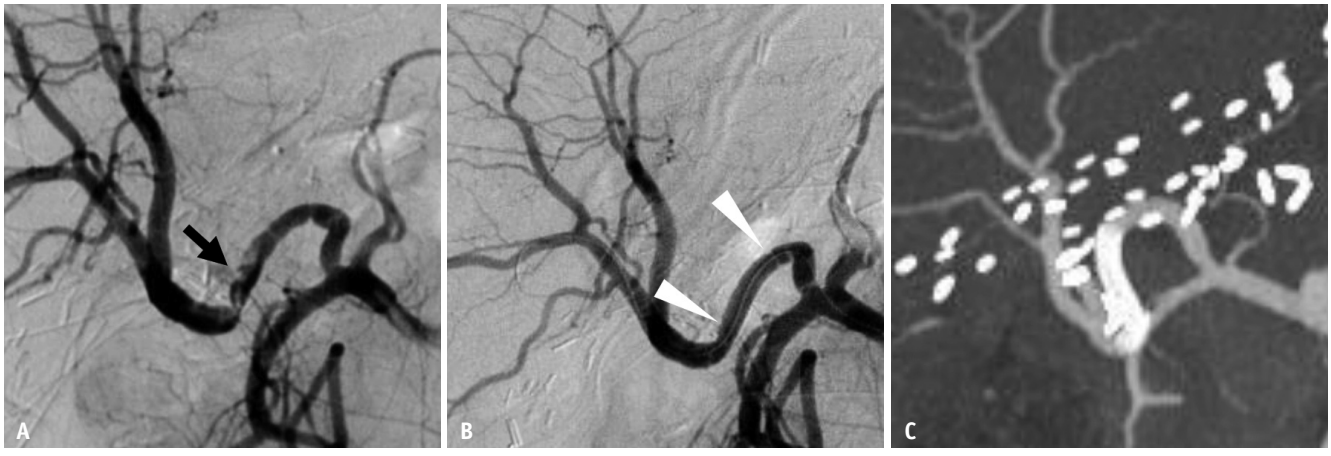

Fig. 6. A 54-year-old woman with a HA dissection 2 days after living-donor liver transplantation.
A. Arteriogram shows a dissection (arrow) around the HA anastomosis. **B.** Arteriogram after a coronary stent placement (4 x 25 mm, NIR stent) (arrowheads) shows normalization of the HA. **C.** CT obtained 7 years after stent placement still shows patent HA flow. HA = hepatic artery

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transplantation or surgical revascularization has been the first choice of therapy in the early post-transplant period [70-72]. However, it presents limitations of scarcity of available liver grafts and morbidity related to repeated surgeries. Endovascular treatments, including catheter-directed thrombolysis, balloon angioplasty, and stent placement, or a combination of these, have been attempted in recent years as a definitive treatment or as a bridge to re-transplantation [70-75].

There is no consensus regarding the therapeutic window for thrombolysis, thrombolytic agent, dosage, method of delivery, duration of therapy, or adjunct use of heparin. Saad et al. [76] suggested a therapeutic window of 1 week to 3 months after transplantation. In comparison, Lee et al. [73] reported that endovascular treatments could be an alternative treatment for HA thrombosis within 24 hours after LDLT. Urokinase has been used in most previous studies, but it is no longer available, and tPA should be used instead. Continuous intra-arterial infusion of tPA (Alteplase; Genentech) for treating post-transplant HA thrombosis has been reported with doses ranging from 0.25–2.0 mg/h. Most studies reporting endovascular treatments for HA thrombosis have small sample sizes. In a review of 69 patients (63 DDLT cases) included in 16 studies, thrombolysis was successful in 47 (68%) patients, and 29 (62%) of the 47 patients underwent additional balloon angioplasty and/or stent placement [72]. Bleeding (20%) was the most common complication, of which 3 had fatal outcomes. In comparison, Kogut et al. [75] reported disappointing outcomes. In their study of 26 patients, the recanalization and major complication rates were 46% and 42%, respectively. There were no statistical differences between patients with successful and unsuccessful recanalization with respect to the survival rates, need for surgical revascularization, re-transplantation rates, ischemic biliary complications, or procedural complications. In LDLT, Lee et al. [73] performed drug-eluting stent placement and/or intra-arterial thrombolysis with a bolus dose of 100000–300000 IU urokinase followed by continuous infusion at a rate of 30000 IU/h for several hours. They reported a technical success rate of 100% (10 patients), but 2 of them required re-transplantation due to anastomotic bleeding complications and 7 experienced biliary strictures (BS). In comparison, Choi et al. [71] reported a technical success rate of 45% (5 of 11 patients), and graft failure and biliary complications occurred in each one patients despite successful recanalization. Therefore, endovascular treatments for HA thrombosis should be selected carefully based on the possibility of liver function recovery, availability of re-transplantation, interventional expertise, and the operator's preferences.

Hepatic Artery Anastomotic Pseudoaneurysm

HA anastomotic pseudoaneurysm is associated with surgical techniques or localized infection. The source of infection is frequently associated with subhepatic fluid collection related to a bile leak or small bowel perforation [77,78]. Rupture of a pseudoaneurysm can be devastating; therefore, urgent surgical or endovascular treatments are required. In the early post-transplant period, surgical therapy is the first choice because maintenance of HA flow and management of coexisting infection are important.

Endovascular treatments, including covered stent placement or embolization, can be performed if surgical therapy is not indicated [65,77-79]. Embolization is effective in achieving hemostasis in patients with a ruptured pseudoaneurysm. However, selective embolization of a pseudoaneurysm while preserving HA flow is almost impossible. In addition, subsequent HA occlusion may lead to graft failure or biliary complications. This procedure is used as a bridge method for surgical revision or re-transplantation. Covered stent placement may be a good option to treat a pseudoaneurysm while preserving the HA inflow. However, the small diameter and tortuous course of the HA, especially in LDLT, makes stent placement challenging. Coexisting infection can also cause HA thrombosis or the recurrence of a pseudoaneurysm. Prolonged antibiotic treatment is necessary following covered stent placement.

Splenic Steal or Portal Hyperperfusion Syndrome

Splenic steal syndrome is characterized by diminished HA flow with predominant flow to the hypertrophied splenic artery [80]. Some authors recently proposed a "portal hyperperfusion theory" to explain the concomitant decrease in HA flow rather than direct siphoning of the HA flow. This diminished HA flow occurs both by direct compression of the sinusoids as well as through the HA buffer response, a regulatory mechanism of arterial vasoconstriction mediated by adenosine [81,82]. This syndrome may be a significant cause of graft ischemia after transplantation, and its most common presentation is elevated liver enzymes. Although there are no established objective diagnostic criteria for splenic steal syndrome, non-occlusive HA hypoperfusion

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with brisk splenic arterial flow on celiac arteriography in conjunction with its clinical manifestations and a high arterial resistive index (> 0.80) can be used to diagnose this syndrome [48,81,83].

Splenic arterial ligation, splenectomy, or a portocaval shunt can be applied to modulate PV flow if this syndrome is suspected during liver transplantation [80,84]. Splenic artery embolization is the predominant endovascular method to not only reduce the splenic artery flow but also increase HA flow and decrease portal hyperperfusion [83]. Embolization is usually performed in the proximal segment of the splenic artery to minimize the risk of splenic infarction, which can lead to abscess, sepsis, or splenic vein thrombosis.

Biliary Complications

Biliary Strictures

Biliary complications, including BS, leaks, stones, biloma, and cast syndrome, have decreased in recent years, but they are still the major cause of morbidity after LDLT. In a systemic review of 14359 patients in 61 studies, the incidence of BS was 12% and 19% among DDLT and LDLT recipients ($p < 0.001$), respectively [85]. BS can be classified as anastomotic or non-anastomotic stricture. Anastomotic strictures are localized to the anastomotic site and are short in length, whereas non-anastomotic strictures are usually multiple and located in the intra- and extrahepatic ducts. An anastomotic stricture is caused by ischemia or fibrosis following suboptimal biliary anastomosis or bile leak [85-87]. Non-anastomotic stricture is associated with HA thrombosis, immunogenicity (chronic rejection, ABO incompatibility, and autoimmune hepatitis), and microangiopathy [87].

Endoscopic therapy is the first choice for treating BS, but percutaneous treatments are frequently used in patients with choledochojejunal anastomosis [88,89] or in those with duct-to-duct anastomosis when an endoscopic approach has failed [90,91]. Percutaneous treatments for anastomotic BS generally consist of percutaneous transhepatic biliary drainage (PTBD), stricture cannulation, balloon dilation, serial exchanges of interpositioning of a drainage catheter up to 14–18 Fr in size, and catheter removal [88,89,92,93]. Two or more PTBDs are sometimes required, especially in right lobe LDLT because of the separated anterior and posterior bile ducts associated with a short donor extrahepatic bile duct and a high incidence of multiple ductal openings. Multiple ductal openings have been reported in 22–56% of living-donor liver grafts [2,94,95]. Ko et al. [88] reported that 2–3 PTBDs were required in 17 (20%) of 83 LDLT recipients to treat anastomotic BS.

Cannulation of the BS could be achieved with a conventional technique using a 0.035-inch guidewire. If cannulation fails while using this technique, a coaxial microcatheter/microwire technique or a snare (rendezvous) method via both percutaneous and endoscopic approaches could be useful [96]. In some cases of short segmental occlusion, cannulation can be possible with a blind puncture using a stiff segment of a guidewire, but it must be performed with caution due to the risk of vessel injury. Magnetic compression anastomosis is another option [86,97]. Balloon dilation is usually performed using conventional balloon catheters. A few reports have demonstrated the efficacy of cutting or drug-eluting balloon catheters for treating BS after liver transplantation [98,99]. However, further investigations are required to verify the efficacy of these balloons.

The reported technical and clinical success rates of percutaneous treatments for BS are acceptable (> 85% for both) [88-91]. However, percutaneous treatments have several drawbacks, including invasiveness, procedure-related complications (arterial injury and cholangitis), discomfort associated with maintaining a drainage catheter, high recurrence rates, and long treatment duration.

Various alternatives have been reported to overcome these drawbacks. Repeat endoscopy using a rendezvous technique following percutaneous cannulation of BS may be useful to improve a patient's quality of life [86,90,91]. Retrievable stents may be effective in reducing the treatment duration and recurrence rate (Fig. 7) [100,101]. The drawbacks of retrievable stents are the risks of stent migration and branched intrahepatic duct occlusion by the stent, especially in LDLT. A recently developed biodegradable stent could help to reduce the treatment duration while achieving favorable and prolonged clinical outcomes. Dopazo et al. [102] used a biodegradable biliary stent for treating 16 anastomotic strictures after liver transplantation, and reported technical and clinical success rates of 100% and 81%, respectively, during a median follow-up of 18 months.

Non-anastomotic strictures are difficult to treat with both endoscopic and percutaneous treatments because of their multiple biliary involvement and progressive nature [87,103]. Multiple PTBDs with prolonged interpositioning

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of internal and external drainage catheters are frequently required. Dual catheter placement via a single percutaneous access may reduce patient discomfort [93]. Frequent drainage catheter exchanges and/or endoscopy are also needed because of the repeated accumulation of biliary sludge or biliary casts. Non-anastomotic strictures are often exacerbated despite such management, and percutaneous treatments are often considered as a bridge method for re-transplantation [103].

Bile Leak

Bile leak is a potentially serious complication, and it is frequently associated with BS [85,86,104]. It usually occurs at the biliary anastomosis but could also occur at the liver cut surface or at the T-tube insertion site. Bile leakage from the liver cut surface may occur in LDLT or split DDLT, and a proportion of this type of leak may be derived from biliary radicles draining the caudate lobe [105]. In a systemic review, the incidence of bile leak was not significantly different between DDLT and LDLT (7.8% vs. 9.5%) [85].

Although endoscopic therapy with bile diversion is the first choice of treatment, percutaneous treatment is a valuable alternative for treating bile leaks resistant to or inaccessible by endoscopic methods (Fig. 8) [104]. Among

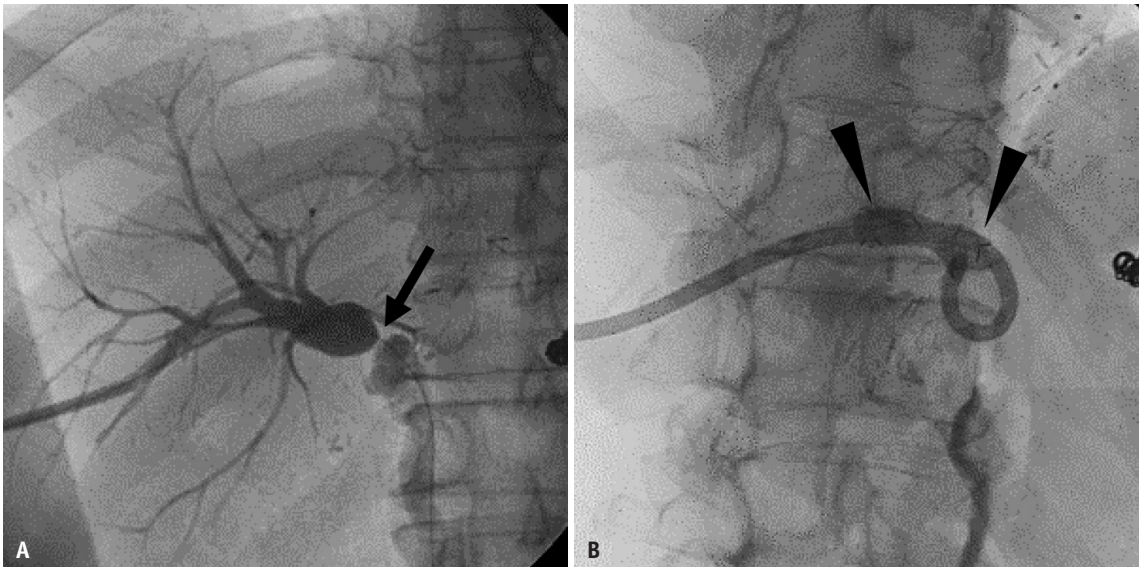

Fig. 7. A 62-year-old man with cholangitis 35 days after living-donor liver transplantation.
A. Percutaneous cholangiogram shows an anastomotic stricture (arrow). **B.** A retrievable covered stent (arrowheads) is placed across the stricture for 4 months. A 10 Fr biliary drainage catheter is positioned in the common bile duct through the stent to prevent stent migration.

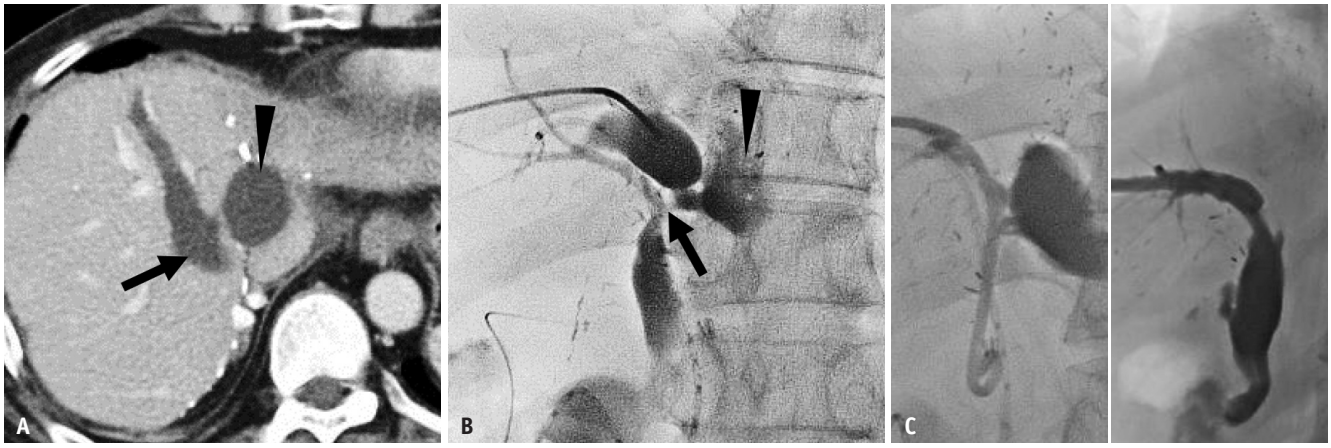

Fig. 8. A 52-year-old man with incidentally detected biliary abnormalities 3 months after dual left lobe living-donor liver transplantation.
A. CT shows bile duct dilation of the right-sided graft (arrow) and an extrahepatic biloma (arrowhead). **B.** Percutaneous cholangiogram after failed endoscopic cannulation of the dilated bile duct shows an anastomotic stricture (arrow) with bile leaks (arrowhead). **C.** A drainage catheter is positioned across the stricture and leak for 7 months.

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the 551 bile leaks reported in a systemic review, endoscopy, surgical revision, PTBD, and observation were performed in 38%, 28%, 10%, and 34% of the cases, respectively [85]. Percutaneous treatments for bile leaks are the same as those for BS, and perihepatic biloma drainage is frequently required. Initial PTBD of the decompressed bile ducts may be difficult, but it could be achieved by ultrasound guidance or opacification of peripheral bile ducts via a previously placed endoscopic nasobiliary catheter or puncture of the centrally located bile ducts [104,106]. de Jong et al. [106] reported a PTBD technical success rate of 91% in 63 patients who had decompressed bile ducts associated with postoperative bile leaks. However, the risk of HA injury is still one of the main drawbacks of PTBD performed for decompressed bile ducts, and an incidence of up to 11% has been reported [107]. Crossing an anastomotic bile leak is usually not difficult with conventional techniques. If that fails due to a major defect, a snare (rendezvous) method in the extraluminal space via both percutaneous and endoscopic approaches can be useful [107]. Balloon dilation of a coexisting BS after the initial PTBD can exacerbate bile leaks; therefore, it is recommended to perform balloon dilation only after the bile leaks have improved [104]. If the bile leaks persist after PTBD with internal biliary drainage, alternative methods may be needed, and the placement of retrievable stents may be useful [107]. The drawbacks of using retrievable stents in bile leaks are the same as those seen when used for BS. Embolization with glue, coils, plugs, or alcohol may be useful for managing bile leaks at the liver cut surface or cystic duct stump [107].

The outcomes of percutaneous treatments of bile leaks are acceptable, but a high incidence of BS following the treatment is a drawback. In a series of 23 LDLT patients with bile leaks, Kim et al. [104] reported technical and clinical success rates of 91% and 70%, respectively. However, repeat PTBDs were required due to BS in 43% of 14 patients showing clinical success.

SUMMARY

With significant improvements in the surgical techniques, post-transplant medical care, and interventional techniques, LDLT has become a definitive treatment for end-stage liver disease. Various vascular and non-vascular interventional techniques can play a pivotal role in improving liver graft and patient survival following LDLT. Although procedure-related complications and recurrences are still a concern, the outcomes of interventional treatments will continue to improve with the ongoing development of interventional instruments and multidisciplinary collaboration among clinicians.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

Author Contributions

Conceptualization: Gi-Young Ko, Kyu-Bo Sung. Data curation: Gi-Young Ko, Kyu-Bo Sung. Formal analysis: Gi-Young Ko, Kyu-Bo Sung. Investigation: all authors. Methodology: Gi-Young Ko, Kyu-Bo Sung. Project administration: Gi-Young Ko, Dong-Il Gwon. Resources: all authors. Supervision: Gi-Young Ko, Kyu-Bo Sung. Validation: Gi-Young Ko. Visualization: Gi-Young Ko, Dong-Il Gwon. Writing—original draft: Gi-Young Ko. Writing—review & editing: Gi-Young Ko, Kyu-Bo Sung.

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