



Surgical complications of liver transplantation

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Abstract: After the first successful human liver transplant in 1967 by Dr. Thomas Starzl at the University of Colorado Health Science Center, the techniques, medicine and technology continued to improve. Liver transplantation is a lifesaving surgery, however, it is not immune to complications. Patient co-morbidities, quality of the graft, surgical procedure and postoperative management all contribute to postoperative morbidity and mortality. Liver transplantation is a complex operation which involves the selection of the recipient, evaluation of the donor, the operation and the recovery. Every time a liver transplant occurs, the surgeons realize that they are not only responsible for one patient, but the lives of three patients. The donor, the recipient and the patient who is waiting to be the recipient. Every step from the organ retrieval, to implantation plays a role in the success of the organ transplant and recipient outcome. Complications can arise during and after liver transplantation, perioperative and surgical complications, immunologic and infectious disorders, and a variety of medical complications. This article covers a brief description of the operation, and the surgical complications of liver transplantation, focusing on surgical complications of the recipient.

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Hepatectomy

The recipient hepatectomy is performed in two ways. The classic technique was first described and performed by Starzl *et al.* (1). The piggy back technique was first described by Tzakis *et al.* in 1989 (2).

The classic hepatectomy involves dissecting out the supra and infra hepatic vena cava, mobilizing the inferior vena cava (IVC) off the retroperitoneum, achieving full vascular isolation and removing the intrahepatic portion of the IVC with the native liver (1). This method may cause hemodynamic instability and the patient may require venovenous bypass for stability prior to reconstruction of the suprahepatic and infrahepatic and restoring the patency of

the IVC. To assess if the patient can tolerate an interrupted venous flow without venovenous bypass, the suprahepatic IVC is cross clamped and the Pringle maneuver is applied prior to the division of the suprahepatic and infrahepatic IVC, and the hepatic artery and portal vein.

The hepatectomy in preparation for a Piggy-back liver transplant involves preservation of the vena cava by dissecting the diseased liver off of the IVC, as it involves anastomosis of recipient hepatic veins anastomosed to donor cava (2). There is less of a risk of hemodynamic instability as the IVC flow is maintained. This technique is also used for recipients of living related donor.

Retrospective studies have compared the classic technique without venovenous bypass and piggyback

technique, and there were no significant differences in post-operative rise in creatinine, decreases in intraoperative blood pressure, transfused packed red blood cells or survival rates between the groups. Warm ischemic time was approximately seven minutes longer in the classic group but still less than 52 minutes, which is an acceptable time for this phase. Therefore, the classic technique without venovenous bypass is just as safe and does not contribute to surgical complications significantly compared to Piggy back in cirrhotic livers when necessary or if the physician prefers this (3).

Cochrane study looked at trials comparing piggy-back with and without intestinal blood diversion, and piggy-back versus classic with venous diversion. Although the studies had high risk of systematic errors there was no significant difference in postoperative death, re-transplantation due to primary graft non-function, nor vascular complications.

The risk of any operation includes bleeding. Specifically, the risk of massive hemorrhage and transfusion are associated with increased risk of mortality and morbidity such as dialysis, and surgical site infection. Cleland *et al.* reviewed resuscitation and transfusion protocols and studies assessing the epidemiology of hemorrhage during liver transplantation such as portal hypertension and coagulopathy, phases of transplantation and the consequences of massive blood loss and transfusion. The studies are limited by outcomes following liver transplant and they were not able to demonstrate association nor causality between blood loss, transfusion requirements and morbidity and mortality. However, they identified pre-operative factors associated with massive transfusion include previous surgery, re-do transplantation, the aetiology and severity of liver disease. Intra-operatively the use of piggy-back technique and avoiding veno-veno bypass has been shown to reduced blood loss (4). Studies demonstrate that a classic transplant does not require veno-veno bypass any longer (5). To manage patients after massive hemorrhage and transfusion, temporary abdominal closures such as a vacuum-assisted closure (VAC) dressing have been used. Chan *et al.* compared and evaluated 34 liver transplant cases that underwent VAC closures for the management of massive intra-operative exsanguination and transfusion (6,7). They demonstrate that this was safe and there were improved short-term survival outcomes, but further evaluation is required to identify long-term morbidity and mortality. In addition, Komorowski *et al.* have recently published

that temporary abdominal closure and delayed biliary reconstruction due to manage these patients with improved outcomes (8).

Reconstruction

The anhepatic phase consists of reconstructing the IVC and portal vein. At all times, the liver is kept cool with ice packs and a slow cold crystalloid infusion.

The goal of the caval anastomosis is to decrease thrombogenesis, by optimizing laminar flow with a wide lumen, adequate flow, prevention of twisting and providing a smooth lumen. In both the classic and piggy back reconstruction, the recipient hepatic veins confluence is opened wide by dividing the tissues between right, middle and left hepatic veins to create a common inflow to IVC and decrease outflow obstruction (9). In a classic transplant, the bicaval reconstruction starts with a suprahepatic anastomosis followed by infrahepatic anastomosis. In a piggy back reconstruction, the donor cava is stapled closed and the recipient hepatic veins are joined together and anastomosed, to the donor cava, such that the liver hinges off the donor cava like opening the hood of a car.

Because there was some concern that venous outflow complications may be increased with piggy back technique versus end-to-end anastomosis (10), variations of the caval reconstruction were created prior to this publication to involve the following. They involved staple closure of the recipient hepatic veins and creation of lateral side-to-side IVC venovenostomy or end-to-end cIVC venovenostomy or a triangulated side-to-side IVC venovenous anastomosis (9,11-13). Regardless of the technique, the imbrication of intima technique is used to prevent leaks and to exclude potential thrombogenic adventitial surfaces.

Studies continue to demonstrate that there is no difference in the risk of thrombosis and narrow and liver dysfunction due to the choice of cava anastomosis (5).

In Piggyback, using the caval preservation technique, there is greater hemodynamic stability, less bleeding, decreased warm ischemic time, improved renal flow, better visualization, and with venous outflow modifications, there has been reduced venous (14) outflow complications. This avoids the need for venovenous bypass and risks of air embolism, nerve injury and wound infections. Splanchnic congestion can be avoided with temporary portal caval shunting (14) and by delaying the division of the portal vein (PV) until the last phase of the hepatectomy (15).

The criteria used to determine which technique to use

depends on surgeon preference, and donor and recipient factors. If the liver is large or caudate lobe is large, a classic caval resection may be easier, such as recipients with Budd Chiari and large polycystic livers. Patients undergoing retransplantation with extensive adhesions and collaterals may also pose a challenge and a classic technique may be the better option. If the hepatocellular carcinoma involves the IVC, the classic caval resection is chosen to achieve tumor negative margins. At the University of British Columbia, a retrospective review was completed to compare three caval reconstruction techniques in orthotopic liver transplantation. The piggy back technique was faster and used less cell saver return, blood and blood products, despite similar blood loss (5).

If there is a mismatched recipient and donor IVC size, as in pediatrics, reduced-size, split or living donor liver transplantation the piggy back technique must be used. In addition, if there is an outflow obstruction following a piggy back anastomosis, the modified piggy back with a side-to-side IVC venovenous anastomosis is an option (16).

As mentioned previously, there is no significant difference in the surgical complication rate between the two techniques (17-20) as long as the anastomosis is widely patent, imbricated and the warm ischemic time is limited. In summary, both techniques are important and may be necessary in order to adapt to the different situations.

Hepatic vein thrombosis or stenosis has an incidence of 1% to 5 % and this is due to intimal hyperplasia. The patients present with Budd Chiari, extremity edema, ascites and dyspnea. Angioplasty, stenting and surgical revision are ways to treat this.

Portal vein

As in the caval anastomoses, the goal is to prevent poor flow, thromboses and narrowing of the portal vein. The recipient and donor vessels are imbricated such that the endothelium of the donor and recipient vessels are touching to avoid thrombogenic surfaces and leaks. In addition, the anastomosis is created without redundancy to prevent kinks and twists. This can be achieved by releasing retractors, trimming off excess length, flushing out clots, and not fully tying down the knot to allow for the expansion of the anastomoses once blood flow is restored to avoid narrowing at the suture line.

There are early and late venous complications. Portal vein thrombosis or stenosis has an incidence of 2-3% (21). Early portal vein thrombosis can lead to liver insufficiency

and failure. If the portal vein thrombosis occurs late, depending on the collateral circulation, the patient can present with portal hypertension with varices and ascites, but liver failure is rare (21). The etiology of portal vein thrombosis is usually (22,23) poor technique causing venous redundancy, kinking and anastomotic stenosis. Other factors that contribute to an increased risk of portal vein thrombosis include prior surgery on the portal or splanchnic venous system, pre-transplant portal thrombosis requiring thrombectomy, a portal vein smaller than 5 mm in diameter, previous splenectomy, hypoplastic portal vein, large portosystemic collateral and the use of venous conduits for portal vein reconstruction (21). In live donor liver transplant recipients, small PV size, and liver graft position (21,24). Depending on the severity at time of diagnosis, the treatment involves possibly venoplasty or stenting, surgical revision or retransplantation (21).

The majority of recipients are patients with cirrhosis who are at risk of having chronic portal vein thromboses. This can be seen in 15% (25) of recipients. Most of these are non-occlusive and a simple eversion thrombectomy of the portal vein can re-establish flow in the recipient. However, if this is not successful, a venous conduit or venous jump graft from the proximal portal vein or proximal to the insertion of the middle colic vein to the superior mesenteric vein may be constructed with the donor iliac vein. If these are not possible, the splenic vein, inferior mesenteric vein or large collateral may be used as an inflow source. It is possible also to create a caval hemi-transposition and anastomose the donor PV to the infrahepatic IVC, then staple the recipient IVC. This is an unusual choice and outcomes are technically inferior (26,27). It is also possible to perfuse the liver by creation of a jump graft or conduit from an artery to the portal vein. This is called arterialization and is rarely done.

Hepatic artery

The hepatic artery anastomoses are created with branch patches on the donor and recipient ends to allow for a Carrel patch to optimize inflow and prevent stenosis of the anastomosis. In addition, attention to orientation and avoiding redundancy is critical to prevent twist and kinks, which may contribute to hepatic artery thrombosis (HAT). Variations in anatomy such as replaced or accessory arterial branches in the donor and recipient add complexity when deciding on the appropriate reconstruction. Usually, the donor celiac artery and splenic artery are connected and the

left gastric artery is ligated, and the recipient proper hepatic artery and gastroduodenal artery are connected to create the patch. If a replaced left or right artery in the donor is identified, the reconstruction is completed on the back table; such as a Busuttil patch.

If there is inadequate inflow from the common hepatic artery, another inflow source is used such as a dominant gastroduodenal artery or a replaced right hepatic artery. If these are not appropriate candidates, a jump graft, using the donor iliac artery, from the aorta to the donor artery can be created. Numerous studies demonstrate that arterial reconstruction of multiple vessels and increased time to arterial reperfusion are risk factors for HAT (28-30), therefore it is critical to prepare the reconstruction on the back table.

HAT is the most severe complication. The overall mortality rate for patient with early HAT is about 33%. HAT can occur early or late. The etiology of HAT is due to surgical and nonsurgical causes. Some surgical causes would include organ retrieval damage such as intimal tears and dissection, technical problems leading to anastomotic stenoses or kinking of the artery, and small (<3 mm diameter) or multiple arteries requiring reconstruction. Arterial conduits are associated with higher rates of HAT (31). Warner *et al.* examined 914 OLTs using univariable and multivariable analyses. They concluded that the main risk factors associated with early HAT (occurring within 1 month post-operation) are abnormal arterial anatomy in the graft requiring back table reconstruction and delayed arterial reperfusion. Abnormal arterial anatomy was associated with a fourfold increased risk of early HAT, and each additional 10 min delay in arterial reperfusion was associated with a 27% increase in the risk of early HAT (28).

Nonsurgical factors which increase the risk of HAT would include the following. Patients who are in procoagulant states due to a pre-existing hereditary coagulopathy such as anti-cardiolipin antibodies or factor V Leiden mutation, or liver disease, such as primary sclerosing cholangitis and human immunodeficiency virus. The use of drugs such as tranexamic acid, aprotinin, excessive intraoperative fresh frozen plasma, elevated hematocrit, and the presence of massive ascites also are contributing factors. Patients with fragile vessels have been associated with HAT including patients with alpha-1-antitrypsin deficiencies and familial amyloid polyneuropathy (30). Pediatric recipients (less than 3 years old and weighing less than 10 kg), small donor or recipient arteries, a split right liver graft, a neonatal donor liver, a cytomegalovirus negative recipient,

a long cold ischemia time, a large liver graft, small-for-size syndrome and ABO incompatibility (31,32). Patients who undergo repeat transplantation have a higher incidence of HAT. It can be diagnosed and treated with angiography and balloon angioplasty (33).

Arterial reconstruction causing HAT early, would present as graft dysfunction. Cholangitis, biliary strictures and patient without symptoms are late presentations of HAT and are due to severe rejection or prothrombotic state usually. The treatment ranges from nothing to retransplantation.

Late HAT is often asymptomatic as collateral blood vessels develop gradually, allowing the patient maintain graft function. Unfortunately, if late allograft dysfunction is detected through monitoring, graft salvage is usually not successful.

Hepatic artery aneurysm or pseudoaneurysm is rare and has an incidence of 0.27–3%, and they may present with nonspecific abdominal pain and or massive gastrointestinal bleeding, or no symptoms at all. They occur in the second or third post-transplant week after infection caused by biliary sepsis, intestinal perforation, anastomotic leak, or intrahepatic stenting, or technical failure. The treatment involves coil embolization, surgical excision or ligation (34).

Hepatic arterial stenosis has an incidence of 1% to 2%. This is due to fibrotic healing or surgical technique. If hepatic artery stenosis is diagnosed immediately postoperatively, revision of the anastomosis should be completed or possibly transplanted. The patients that present late can present with graft dysfunction. The treatment is angioplasty. Unfortunately, if a biliary complication results from this complication, retransplantation may be necessary. If diagnosed promptly, surgical hepatic artery revision is an option.

The use of a doppler ultrasound after surgery and daily for a week, keeping a low hematocrit, replacing coagulopathy proteins, and administration of chemical deep vein thrombosis (DVT) prophylaxis have been demonstrated to reduce the incidence of HAT. The use of microvascular surgical techniques may also decrease the incidence of HAT as well (31).

Bile duct

This anastomosis is created after the liver is perfused. The goal is to create an anastomosis that is well perfused, tension-free, and as widely patent as possible to decrease the risk of leaks and stenosis. To achieve an end-to-end choledochocholedochostomy the ends are trimmed to

healthy surfaces, and bleeders are controlled by ligation or low setting electrocautery to preserve as much duct perfusion as possible. If there is a minor size discrepancy, spatulating or partially closing the ends are ways to correct the discrepancy.

Stents are placed to facilitate flow of bile and maintain a patent sphincter of Oddi to prevent leaks, which can lead to strictures later on.

If there is a size discrepancy, difficulty connecting the two duct ends, poor condition or blood supply, a Roux-en-Y hepaticojejunostomy should be considered. Pre-existing duct diseases such as choledocholithiasis, biliary atresia, biliary cirrhosis or primary sclerosing cholangitis are also indications for Roux-en-Y. Unfortunately, this prevents access to the liver with standard ERCP after it is created. Because of this, some authors report the use of duct-to-duct anastomosis in patient with primary sclerosing cholangitis, and retransplantation cases who have healthy ducts.

Despite all the advances in transplant patient care and surgical techniques, biliary complications remain the most common postoperative technical complication with an estimated incidence of 15% or less in deceased donors, and up to 30% in living donor or split liver transplant. There are early and late complications, and there are anastomotic, and nonanastomotic biliary complications, such as stones, sludge and casts (35,36).

Anastomotic leaks are most due to technical failure or ischemic necrosis of the anastomosis. Acute HAT will cause vascular insufficiency and may lead to ischemia and necrosis of the liver and associated biliary tree (37).

Biliary strictures are the most frequent late biliary complication. These are caused by ischemia and reperfusion injury, poor technique, fibrotic healing and bile leak. The incidence of anastomotic biliary stricture is 0.6% to 17.6% (38). These can usually be treated with dilatation and stenting with ERCP or with a PTC drain or biliary reconstruction with a Roux-en-Y choledochojejunostomy.

The incidence of nonanastomotic biliary stricture is 5% to 10%, and this is usually due to HAT, immunogenic causes and prolonged cold-ischemia time. When biliary strictures occur, biliary stones, sludge and cast can develop due to poor flow or stasis. Patients with stones usually present with biliary obstruction causing cholangitis and graft dysfunction. There is a 5% incidence (35).

Risk factors which most correlates with increased risk of biliary complications are liver cold ischemia time, and donor and receiver age over 20 years (37).

Late surgical complications in pediatric recipients

are higher than in adults. Both biliary and vascular complications are higher as there is a common need for vascular reconstruction as split allografts or reduced size are used. Late anastomotic and nonanastomotic biliary strictures occur in 7% of pediatric recipients (36). Due to variable biliary anatomy of donor segments 2, 3 and 4, reconstruction is challenging and the stricture rates is 24%; 1/3rd of patients will have hepatic outflow or portal vein late stenosis. The rates of HAT has decreased due to microsurgical techniques and routine anticoagulation and antiplatelet therapy in pediatric recipients (36).

Extrahepatic complications

Transplant recipients are on immunosuppression medications that interfere with healing and angiogenesis immediately, therefore, it is not a surprise that herniation is the most common late general surgery complication after liver transplantation. Incisional hernias occur in 4% to 20% of patients (38). These hernias can be observed or repaired with prosthetic mesh. Right-sided diaphragmatic hernias have an incidence of less than 1%, and are observed in pediatric liver recipients. These hernias are surgically repaired as they are frequently associated with bowel obstruction. Factors that contribute to an incisional hernia includes patients with repeated surgery, postoperative wound infection and who are obese (38).

Anyone who gets an abdominal operation is at risk of intraperitoneal adhesions and bowel obstructions. The incidence is 1% to 2%. In addition, internal hernias, abdominal wall hernias, and neoplasms (post-transplant lymphoproliferative disorder) have been reported to cause bowel obstruction. Recipients who have had a Roux-en-Y choledochojejunostomy are at risk for internal hernia through the mesenteric defect of the Roux limb which can lead to life threatening bowel strangulation in the absence of bowel obstruction (38).

Most surgical complications occur in the first 90 days post-transplant. Late complications do occur and the management of these can range from observation, to interventional radiology procedures to re transplantation.

Although there are some minor variations in liver transplant technique, the classic versus the piggy-back (PB) technique have minor different outcomes. At our institution, we demonstrated that the PB technique was faster, and despite similar blood loss, the PB technique used less cell saver return, fresh frozen plasma and platelets. These findings demonstrate that different caval

reconstruction techniques are equally efficient and safe, and provide numerous options for difficult cases (5).

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