Hepatic Artery Interventions in the Transplant Patient

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Abstract

Arterial complications are some of the feared complications of hepatic transplantation. This stems from the fact that while hepatocytes derive most of their blood supply from the portal vasculature, bile ducts get their supply from the hepatic artery. Arterial insufficiency overtime causes biliary ischemia, necrosis, and often hepatic graft failure. Once suspected clinically, a prompt imaging work-up is usually performed. After confirmation of suspected hepatic arterial injury or insufficiency, quick intervention is usually required. Knowledge of the common hepatic artery complications, their imaging diagnosis, and different treatment modalities is critical for any provider who treats these patients. This review covers the imaging diagnosis and types of vascular injuries, and analyzes the different treatment options.

Keywords
- transplant
- hepatic artery
- intervention

Liver transplantation (LT) was pioneered in the 1960s, with initial limited success in adult patients.[1,2] Since then, numerous improvements have been made in both the surgical technique and supportive immunosuppression to bring this life-saving procedure to both adults and children with end-stage liver disease.[3] Today, the most common procedure is the orthotopic LT (OLT), in which the recipient's diseased liver is removed and a donor's liver is placed in the normal anatomical location.

While the success of this procedure has improved, it still has numerous complications. Many of these complications are related to the hepatic vascular structures (hepatic artery, portal vein, and hepatic veins). With recent advances in anticoagulation and endovascular technique, open surgical explorations are often not the first-line treatment for many of these ailments.[4]

Given this area of rapid change and improvement, it is important for providers to be familiar with the pathophysiology and current treatment algorithms. While many of the complications are related to venous stenosis or occlusion within the portal/hepatic veins or the inferior vena cava,[4] this review primarily focuses on the arterial anastomosis and associated complications. These include surgically related complications such as hemorrhage, pseudoaneurysm, arteriovenous fistula, and dissection (possibly leading to thrombosis), or more delayed, but equally urgent, findings such as hepatic artery stenosis (HAS) and thrombosis, which can be found in up to 9 to 13% of transplants.[5] While hepatocytes receive dual blood supply from both the hepatic artery and the portal vein, bile ducts are solely supplied by the hepatic artery and with arterial insufficiency leads to biliary necrosis, leakage, strictures, cholangitis, and possible graft failure.[6]

This review starts with a look at the types of surgical arterial reconstructions followed by analyzing the different noninvasive imaging techniques that are critical to the initial work-up as well as follow-up of hepatic arterial injury before discussing the different types of arterial injuries and their associated treatment options.

Types of Arterial Reconstructions

When evaluating the posttransplant patient, it is important to understand what type of hepatic arterial anastomosis was performed. This is frequently the most technically challenging aspect of the transplant procedure and requires knowledge of microvascular techniques.[7] In most cases, an end-to-end anastomosis between the donor hepatic artery and the celiac axis stem or common hepatic artery stem is performed.[3,5]

Although when circumstances arise that prohibit this portion of the procedure, an interposition conduit may be used. Often this is due to a donor hepatic artery that is too

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short or of unsuitable caliber or celiac stenosis. The conduit is usually performed using an end-to-side iliac graft to the abdominal aorta.[8] If this conduit cannot be performed, there are many reports on the use of other grafts from the donor gastroduodenal, splenic, right gastroepiploic, radial, and inferior mesenteric arteries.[9]

Noninvasive Imaging of Hepatic Transplant Graft Arteries

The primary imaging modality used for the follow-up of LT is ultrasound (US).[10] The normal US appearance should demonstrate a homogenous or minimally heterogenous pattern on gray-scale imaging without biliary duct dilation.[10] However, for the evaluation of the hepatic vasculature, particularly the hepatic artery, color and power Doppler are indispensable. The hepatic artery should demonstrate a low resistive waveform with rapid systolic peak and low resistive index between 0.5 and 0.7, with no reversal of diastolic flow.[10]

In the setting of hepatic artery compromise, the spectral waveform is helpful for further investigation. The absence of flow on Doppler and power color suggests thrombosis. A "syndrome of impending thrombosis" has been suggested occurring in the immediate posttransplant period, with progressive loss of diastolic flow leading to loss of all flow within a 10-day period, demonstrating the importance of follow-up imaging of abnormal US findings.[11] Other findings such as elevated velocities (>200 cm/s), aliasing, or parvus tardus waveforms are also suggestive of stenosis and warrant further evaluation; however, it should be noted that absolute velocity cutoffs have not been well established in the hepatic artery. High resistive indices (>0.8) are expected for the first 3 days after transplant and should really be considered abnormal after day 4.[12]

Overall, US is up to 92% sensitive for the diagnosis of HAS.[13] Of note, a new hypo/anechocic structure within the surgical bed should be evaluated with color Doppler for a pseudoaneurysm.[10] Recent reports have shown a possible benefit using microbubble contrast-enhanced US given its increased sensitivity (near 100%) and specificity (~70%).[14]

The computed tomography (CT) angiographic findings of abrupt cutoff of the hepatic artery are consistent with occlusion or high-grade stenosis. Luminal narrowing may suggest a lower grade moderate stenosis. Overall, CT has a high sensitivity (100%), specificity (89%), and diagnostic accuracy (93%) in vascular complications.[14] An enhancing outpouching suggests pseudoaneurysm formation. The additional advantage of CT is the ability to evaluate for multiple causes of graft dysfunction with a single examination. Compared with magnetic resonance angiography (MRA), CT is more readily available, which makes it an appropriate second-line imaging modality.

MRA of hepatic arterial disease has a similar appearance when compared with CT. However, one of the increased benefits of MRA compared with CT is biliary evaluation.[14] Biliary excreted MR contrast agents (gadobenate dimeglumine or gadoxetic acid) using T1-weighted and heavily T2-weighted noncontrast sequences can provide a more detailed evaluation of the biliary system looking for strictures and other sequelae of arterial insufficiency.[15,16]

Hepatic Artery Thrombosis

Hepatic artery thrombosis (HAT) is the most common arterial complication post–OLT, occurring in approximately 2 to 9% of grafts.[17,18] Unfortunately, it also carries a high mortality rate, which is reported in 20 to 60% of cases.[17] In one retrospective study, approximately one-third of all hepatic transplants were because of HAT in a previously transplanted patient.[18,19] HAT is described as "early" if it occurs within the first 30 days of post-transplantation and "late" after 30 days.

Early HAT (E-HAT) is usually seen in the setting of ABO blood type incompatibility, increased cold ischemic time during transplantation, cytomegalovirus (CMV) mismatch (seropositive donor with seronegative recipient), acute rejection, or surgical factors such as intimal injury during anastomosis.[6,17] A meta-analysis demonstrated an overall incidence of 2.9% in adults and 8.3% in children. This risk is approximately sixfold higher in patients who have been retransplanted.[20] Given its abrupt clinical presentation and severity (mortality is ~33.3%), it is important to have a low suspicion, and early diagnostic studies should be obtained.[6] In fact, it is often routinely screened for in the early postoperative period.[6,18]

E-HAT presents as right upper quadrant pain, fevers, ascites, and transaminitis.[17] This abrupt ischemia leads to profound damage to hepatocytes and bile ducts. In an immunosuppressed patient, this can lead to uncontrollable biliary sepsis and, in many cases, death.

If E-HAT is suspected, an urgent hepatic US with color Doppler vascular examination is the preferred first-line imaging modality. If the US is equivocal or further preprocedural planning is required, a CT angiogram should be performed.

The most effective treatment for HAT is an area of active discussion. Urgent retransplantation is usually considered the best option in E-HAT, but with the overall scarcity of donor livers, this is often not possible. If retransplantation is not possible, revascularization should be entertained. Traditionally, this has been done surgically, but there have been increased reports of endovascular management with intra-arterial thrombolysis, balloon angioplasty, and stent placement.[19,21]

Results from endovascular therapy have been mixed. Thrombolysis alone has been shown to be suboptimal by Kogut et al, with only 62% of E-HAT patients regaining hepatic arterial patency.[22] However, in a midterm evaluation by Lee et al, assisted primary patency of endovascular therapy using thrombolysis, angioplasty, and stents was 80% at 643.5 days.[21]

One of the challenges of endovascular management is the small hepatic artery dimensions and availability of suitable stents. Use of small-caliber drug-eluting coronary stents has been reported.[19,21]

The major complication of endovascular therapy is rupture given the manipulation and angioplasty of a newly created anastomosis. In E-HAT, bleeding rates of approximately 20 to 62% have been reported for patients receiving catheter-directed thrombolysis.[22,23] Balloon angioplasty should be avoided within

the early postoperative period (<2 weeks) given the risk of rupture.

Unlike E-HAT, late HAT (L-HAT) presents more insidiously, usually with biliary tract pathology (strictures, bilomas, hepatic abscesses). Often, L-HAT can present with elevated liver function tests in an otherwise asymptomatic patient.[24] A less severe presentation is possibly due to the arterial collateralization that is able to develop after transplantation.[25] This collateralization allows enough blood supply for the hepatocytes (with alternative supply from the portal vein) but insufficient supply for bile ducts with their single blood supply.

Approximately 20% of patients presenting with L-HAT will go on to develop eventual graft failure requiring a second transplant and, 33% have been reported to survive long term without revascularization or need for retransplantation.[24] Risk factors for L-HAT are less clear compared with that for E-HAT. The most common risk factors are CMV mismatch, female donor/male recipient, hepatitis C positive recipients, tobacco consumption, and retransplantation.[25]

As with E-HAT, in a patient with suspected L-HAT, an US should be obtained followed by CT and catheter angiography as needed. The first report of thrombolytic therapy for L-HAT was by Hidalgo et al in 1989, and since then, numerous studies have subsequently been performed, although no consensus has developed on when catheter-based thrombolysis may be effective.[23,26] However, after successful thrombolysis, evaluation of the underlying etiology of thrombosis should be performed. In case of kinking or stenosis, balloon angioplasty or stenting should be performed to prevent rethrombosis. Although it should be noted that many reports of stent placement (including drug-eluting stents) have not demonstrated long-term patency.[27–29]

Hepatic Artery Stenosis

HAS is defined as a transluminal diameter decrease of >50% on angiography. This decrease in flow can lead to graft ischemia. As previously noted, ischemia can eventually lead to biliary duct complications, though this causal relationship is not well defined in HAS.[30] HAS does have a relationship with eventual HAT, which can lead to biliary complications. Overall, HAS presents in approximately 2 to 13% of transplants.[25] The etiology of HAS is unclear, but many authors suggest a multifactorial etiology with

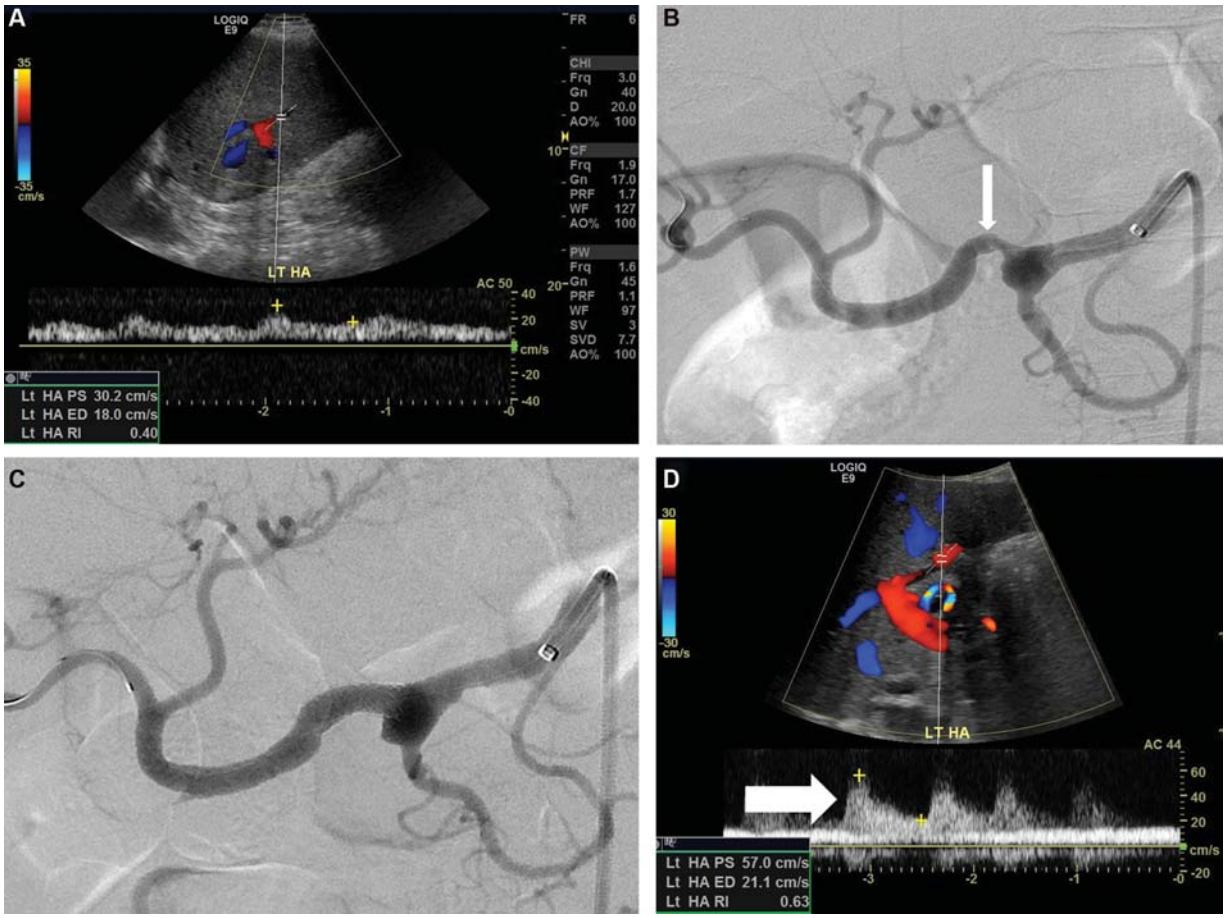

Fig. 1 (**A**) Hepatic arterial duplex ultrasound in a patient 20 days postorthotopic liver transplantation, with elevated liver enzymes demonstrating a parvus tardus waveform within the left hepatic artery. Additionally, there is a reduced resistive index measuring 0.4 (*white arrow*). (**B**) Oblique digital subtraction angiogram of the celiac artery showing a hemodynamically significant stenosis near the anastomosis of the common hepatic artery to the celiac artery (*white arrow*). (**C**) The patient was treated with balloon-expandable stent placement with resolution of the stenosis. (**D**) Postprocedure ultrasound highlighting the return of normal waveform to the left hepatic artery (*white arrow*). (These images are provided courtesy Andrew Lipnik, MD, and Ketan Shah, MD.)

perioperative vascular injury, hepatic arterial kinking or angulation, or microvascular injury (such as acute cellular rejection).[25,31]

If HAS is suspected, color Doppler US is the first line evaluation. The area of stenosis is most commonly at the arterial anastomosis.[31] A hepatic artery with a peak systolic velocity (PSV) > 200 cm/second is suggestive of HAS. It is often difficult to get a sonographic window of the arterial anastomosis. Therefore, the diagnosis can be suggested with secondary signs such as a low post anastomotic PSV (< 40 cm/second) or parvus tardus spectral waveform.[32] Studies have shown US having an 85% sensitivity and a negative predictive value of 100% for detecting HAS.[25]

Once HAS has been diagnosed, it is important to intervene to prevent transformation into HAT. Conventionally, this has been performed with surgical revision or transplantation. However, as techniques have improved, endovascular management has been suggested as a viable first-line therapy, though this is debated in the literature.[31] As with HAT, the mainline therapies are percutaneous transluminal angioplasty (PTA) and stent placement (►**Fig. 1**). A meta-analysis looking at 263 cases of HAS demonstrated no significant difference between the outcomes of PTA and stenting when considering rates of reintervention and retransplantation.[31] Given their relative similarities, other specific patient characteristics could be considered when choosing treatment modality.

For instance, PTA is considered contraindicated in the immediate postoperative period (<2 weeks) due to the fear of anastomotic rupture.[31] Shorter segment stenosis may benefit from PTA, whereas arterial kinking or long-segment occlusions may benefit from stenting, although this has not been evaluated in the literature.[31] Overall, the long-term outcomes from stenting remain unclear. Older reports demonstrate poor stent patency, with one study of 37 patients demonstrated a primary patency of 44% at 14.5 months.[33] Newer studies demonstrate improved primary stent patency of 78% and an assisted primary patency rate of 93% at 24 months.[34]

The main benefit of percutaneous therapy is the decreased rate of eventual HAT development. Saad et al describe a greater than threefold decrease in the progression of HAS to HAT in patients who underwent PTA.[33] Newer reports suggest that HAT can be avoided in >95% cases with endovascular management.[34]

Hepatic Artery Pseudoaneurysm and Rupture

Hepatic artery pseudoaneurysms (HAPs) are less likely to occur compared with thrombosis at a rate of 0.3 to 3%.[35,36] Their risk of life-threatening hemorrhage makes HAP one of the more important acute postsurgical complications. In a recent retrospective review, 69% cases of HAP presented by the 20th day postoperatively and 81% by the 35th day postoperatively (median: 13 days).[36] The most common presentation is acute blood loss anemia with hemorrhage

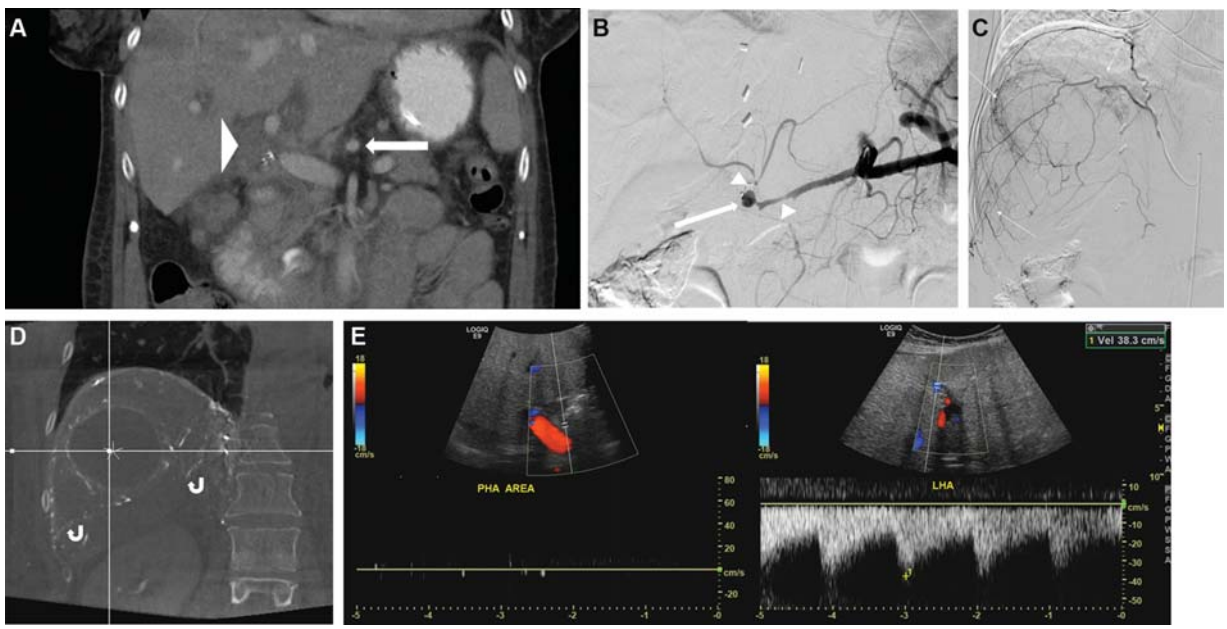

Fig. 2 (**A**) Coronal projection from a CTA (computed tomography angiography) in a patient 2 weeks posttransplantation with acute onset upper gastrointestinal hemorrhage and hemobilia. There is abrupt termination of the common hepatic artery near its origin from the celiac artery (*white arrow*). Additionally, there is ill-defined fluid density material near the porta hepatis (*arrowhead*). (**B**) Anteroposterior (AP) digital subtraction angiogram after catheterization of the celiac artery demonstrating irregularity of the common and proper hepatic arteries (*arrowheads*) with a 1.2-cm pseudoaneurysm (*white arrow*) from the proper hepatic artery. (**C**) AP digital subtraction angiogram of the left inferior phrenic artery demonstrates numerous collaterals (*arrows*) to the hepatic parenchyma. (**D**) Cone-beam CT confirms collateral development to the hepatic parenchyma from the left inferior phrenic artery (*curved arrows*); a large cyst from the donor liver is noted (*asterisk*). (**E**) After consultation with the transplant surgeon, the patient received surgical repair and reconstruction of the hepatic artery. Three months later, an ultrasound demonstrated thrombosis of the proper hepatic artery repair with enough flow through left hepatic artery, likely from the robust collateralization. The patient had no adverse sequalae.

into the peritoneum, retroperitoneum, or gastrointestinal tract (►**Fig. 2**).[37] Patients who present with rupture have a reported mortality rate greater than 50%.[36] Elevated liver function tests may also be seen with this condition, although is less specific.[38]

Based on location, pseudoaneurysms have been divided into intra- and extra-hepatic. Intrahepatic origin is usually due to percutaneous intervention such as a biopsy, whereas extra-hepatic aneurysms occur most frequently at the hepatic arterial anastomotic site. Extrahepatic pseudoaneurysms most often are mycotic in origin, from a localized postoperative infection (such as a biliary leak) or perforation from the hepaticojejunostomy.[38,39] Nonmycotic origins are usually related to technique during the creation of the hepatic arterial anastomosis.

In a patient with elevated liver enzymes and blood loss anemia post transplantation, after initial resuscitative efforts, multidetector CT angiography is an excellent first diagnostic test to perform given its potential superiority compared with US.[40] This can rapidly make the correct diagnosis with the ability to often discern the etiology of the pseudoaneurysm, which can potentially alter procedural planning. For instance, in the setting of a mycotic aneurysm, a stent-graft would not be an appropriate treatment. Increased morbidity is noted with delayed time to diagnosis; therefore, at our institution, it is usually appropriate to obtain a CT examination even with a low index of suspicion.[36]

A multidisciplinary discussion should take place between the transplant surgeon, hepatologist, and interventional radiologist prior to initiating any treatment as early hepatic arterial failure contributes to a high risk of transplant graft failure (►**Fig. 3**). There are numerous reports of surgical PSA excision and arterial repair using grafts.[36] New reports of endovascular techniques present a minimally invasive approach compared with a potentially morbid surgery in a recent postoperative patient.

Choice of intervention is usually decided based on the size, location, and neck of the PSA. In the setting of nonmycotic pseudoaneurysms, there has been reported success with stent-graft exclusion of the PSA.[41–43] Coil embolization may be an alternative therapy in these settings. Care should be taken as a pseudoaneurysm is a contained perforation, and coils may migrate or rupture the PSA.[44]

Further complication of intrahepatic pseudoaneurysms is the possibility of fistula formation from rupture into the portal vein or biliary ducts. Fistula formation into the portal vein is typically related to iatrogenic injury from liver biopsy and is reportedly much more frequent in biopsies performed during the first week of transplant.[45] Treatment algorithm is similar to pseudoaneurysms, with angiogram and embolization as effective minimally invasive first approaches.[46]

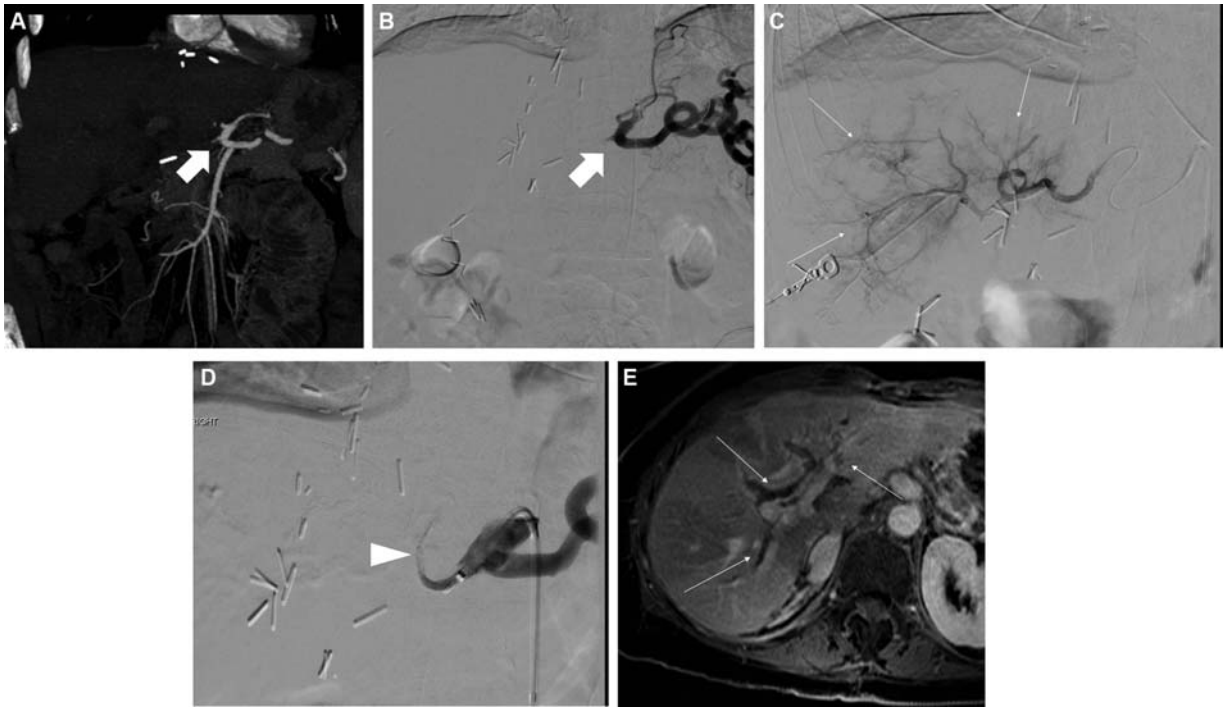

Fig. 3 A patient 4 weeks postorthotopic liver transplantation with abnormal liver function tests on outpatient follow-up. (**A**) Coronal Maximum intensity projection CTA (computed tomography angiography) demonstrating proximal occlusion of the common hepatic artery (*arrow*). (**B**) Anteroposterior (AP) digital subtraction angiogram with selection of the celiac artery confirming occlusion of the common hepatic artery near its anastomosis (*arrow*). (**C**) AP digital subtraction angiogram after 24 hours of catheter-based thrombolysis shows improved, but sluggish, flow through the proper hepatic artery, with little and irregular parenchymal staining (*arrows*). (**D**) After 48 hours of thrombolysis, the common hepatic artery remains thrombosed due to poor antegrade flow (*arrowhead*), and no stenotic lesion was seen. Conservative management was elected given patient stability. (**E**) Two months postprocedure, axial contrast-enhanced T1-weighted MRI (magnetic resonance imaging) demonstrated abnormal and irregularly dilated central bile ducts diagnostic of ischemic cholangiopathy due to arterial insufficiency (*arrows*). The patient was eventually lost to follow-up after moving out of state.

Hepatic Artery Dissection

Of the posttransplantation arterial pathologies, hepatic arterial dissection (HAD) is the least common and is confined to case reports and small series. Although rare, it remains important due to its quite severe sequelae. The etiology of HAD is usually related to technical factors such as surgical technique and subsequent posttransplant endovascular therapies (often related to treating HAS). A reported risk factor for endovascular dissection is hepatic arterial tortuosity, which was seen in 75% of patients with complications compared with 37.5% of patients without complication.[47]

Ultimately, HAD can result in HAT, which carries the risk of hepatic graft failure or subsequent biliary ischemia and related morbidities, as described previously. In one study evaluating endovascular therapy for HAS, five cases of hepatic artery dissection were reported in 106 interventions.[47] These were ultimately treated with bare-metal and drug-eluting stents. At 22 months follow–up, 50% had progressed to HAT compared with 1.4% of patients without complications, highlighting the critical nature of this condition.

Conclusion

Hepatic arterial injury remains one of the most severe common complications following OLT. While many of these conditions can eventually lead to graft failure, other complications such as biliary ischemia and subsequent sepsis are important when evaluating a patient with a suspected arterial injury. Historically, therapies have revolved around urgent surgical management, but as endovascular techniques have improved, many invasive and complex surgeries can be substituted with percutaneous procedures. This highlights the important interdisciplinary approach when treating posthepatic transplant patients.

Conflict of Interest
None.

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