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Understanding Local Hemodynamic Changes After Liver Transplant: Different Entities or Simply Different Sides to the Same Coin?

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Abstract. Liver transplantation is an extremely complex procedure performed in an extremely complex patient. With a successful technique and acceptable long-term survival, a new challenge arose: overcoming donor shortage. Thus, living donor liver transplant and other techniques were developed. Aiming for donor safety, many liver transplant units attempted to push the viable limits in terms of size, retrieving smaller and smaller grafts for adult recipients. With these smaller grafts came numerous problems, concepts, and definitions. The spotlight is now aimed at the mirage of hemodynamic changes derived from the recipients prior alterations. This article focuses on the numerous hemodynamic syndromes, their definitions, causes, and management and interconnection with each other. The aim is to aid the physician in their recognition and treatment to improve liver transplantation success.

INTRODUCTION

Liver transplantation (LT) is one of the major breakthroughs in the treatment of end-stage liver disease (ESLD) and has suffered several modifications and been the subject of many studies ever since it was first performed almost 60 y ago by Starzl.[1] To increase the donor pool and allow for living donor liver transplant (LDLT), smaller grafts, such as left liver grafts, were developed.

With these smaller grafts came numerous problems, concepts, and definitions. The spotlight is now aimed at the mirage of hemodynamic changes derived from the recipient's prior alterations (portal hypertension, portosystemic shunts, splenomegaly) and a healthy graft, previously unexposed to such changes. Different and not surprisingly confusing terms, such as "small for size," "small for flow," "big for size," "portal steal syndrome," "splenic steal syndrome," and "splenic artery steal syndrome," have overcome the literature and can transform posttransplant hemodynamics into a true headache for the new coming physician. Not infrequently, these entities intertwine with each other, presenting the transplant team with a complex scenario and decision making. The aim of this article is to present a complex case, in which the patient presented numerous hemodynamic syndromes (large-for-size, small-for-size, portal hyperflow, splenic artery steal syndrome [SASS]). This requires prior knowledge and understanding of these conditions. To do so, the current literature regarding these topics will be reviewed, focusing on definitions, causes, and management. It is to be duly noted that all of these scenarios are by themselves rare and by no means present as a frequent complication in most experienced transplant units. However, recognition of these as potential problems and complications is crucial in their prevention and treatment. We will mainly focus on the information pertinent to whole graft transplantation, as many of these conditions vary when discussing partial grafts.

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"SMALL FOR SIZE" OR "SMALL FOR FLOW"?

Scarcity of suitable donors has forced surgeons into LDLT. After the first successful pediatric LDLT in 1990[2] and the first adult-to-adult LDLT in 1994 by Hashikura et al,[3] its popularity has risen worldwide, especially in Asian countries. As expected, several reports regarding adequate graft size were published. Just as it happened with oncologic liver resections, remnant liver volume or graft size became the focus of several studies.

The question in LDLT classically focused on the "size of the graft" compared with the recipient. How much liver does the patient require, and how can surgeons make hepatectomy a safe procedure for donors? Several studies confirm a "safety threshold" under which graft vitality and patient survival are greatly compromised. A graft-weight-to-recipient-weight (GWRW) ratio of 0.8% and a standard liver volume (SLV) of 40% have been considered adequate limits for LDLT. With smaller grafts, a syndrome that included encephalopathy, intractable ascites, and sustained bilirubin and international normalized ratio impairments was reported. From these observations in small-sized grafts, the term "small-for-size syndrome" (SFSS) was cradled. Soejima et al[4] reference SFSS for the first time in 2003. In their definition, SFSS included total bilirubin (TBil) levels of >5 mg/dL at day 14, as well as 1 L of ascites at day 14 or 500 mL/d at day 28. Over the next 15 y, numerous groups have attempted to define SFSS. One of the most popular definitions is one proposed by Dahm et al[5] in 2005. In patients with GWRW <0.8%, SFS dysfunction was considered in those patients with 2 of 3 criteria for 3 consecutive days during the first postoperative week: international normalized ratio >2, TBil >100 μmol/L (5.84 mg/dL), or grade III/IV encephalopathy. However, SFSS is an exclusion-based diagnosis, and one must consider other causes, such as acute rejection, sepsis, and outflow impairments. The latter have little to no correlation with SFSS when discussing orthotopic LT (OLT) because outflow impairments are rare, and normal escape pathways are correctly preserved in an adequately sized liver. However, special attention must be set on them when retrieving right livers in LDLT,[6] in which normal outflow pathways such as the middle and inferior hepatic veins are sectioned, and size of the graft may be small to marginal in some patients. Outflow impairments are discussed later on.

Pathology analysis of SFSS specimens revealed ballooonization of hepatocytes, with hemorrhagic and ischemic areas, associated with cholestasis. These findings were coincident in animal experiments as well and gradually led to a change of paradigms: SFSS was less influenced by size than it was by portal hypertension and hyperflow. In 2013, Asencio et al[7] hypothesized that the hemodynamic changes that occurred after small graft transplantation were similar to those in extensive liver resection for oncological purposes. In these cases, portal hyperflow generates shear stress upon sinusoidal capillaries and Disse spaces, leading to ischemia and necrosis. Shear stress generates several microvascular changes, including overexpression of endothelin-1, a potent vasoconstrictor, and underexpression of antioxidant proteins such as heme oxygenase-1.[4,8] Figure 1 shows how these vascular changes affect the donor graft.

Several studies have proved the role of portal hemodynamics in SFSS. In 2011, Sainz-Barriga et al[9] published several measurements in 81 LT, including portal vein flow (PVF), portal vein pressure (PVP), and hepatic venous pressure gradient. They observed that there was no correlation between PVF and PVP as one might think but identified hepatic venous pressure gradient >15 mm Hg as a risk factor for poorer outcomes and a valuable tool for assessment of peritransplant hemodynamics. Sainz-Barriga et al[9] hypothesized a flow and pressure "window," which would dictate the necessity of portal venous pressure modulation. In their consideration, the PVF limit should be 4 times normal values, which means >360 mL/min/100 grams of graft.

In 2016, Uemura et al[10] achieved noninferior results in terms of graft and patient survival in grafts with GWRW between 0.6% and 0.8%. To do so, subjects receiving these grafts were submitted to concomitant PVP modulation. A combined analysis of portal pressure >15 mm Hg and a PVF >180 mL/min/100 grams were considered indications for inflow modulation. Inflow modulation techniques included splenic artery ligation (SAL), splenectomy, portal banding, and, in extreme cases, portocaval/mesocaval shunts. In 2003, Troisi et al[11] published results comparing use of SAL in 17 adult LDLT versus a control group with no SAL. Portal hemodynamics were significantly improved in the SAL group, although no differences in matters of complications were identified, most likely because of the small number of patients in the study. In the light of all of these findings, some authors support shifting away from SFSS and renaming it "small for flow Syndrome" (SFFS)[12] as suggested by Asencio et al.[7]

One may conclude that SFSS/SFFS is mainly a problem of flow, rather than size, as it may be present even in grafts that exceed 0.8% GWRW. When expected or confirmed, inflow modulation can aid in its treatment or prevention. Portal hyperflow/hypertension are not the only indications for inflow modulation techniques. It is well known that portal hyperflow has a directly inverse relationship with hepatic artery flow (HAF). This mechanism, known as "hepatic artery buffer response" (HABR), regulates inflow to the liver and

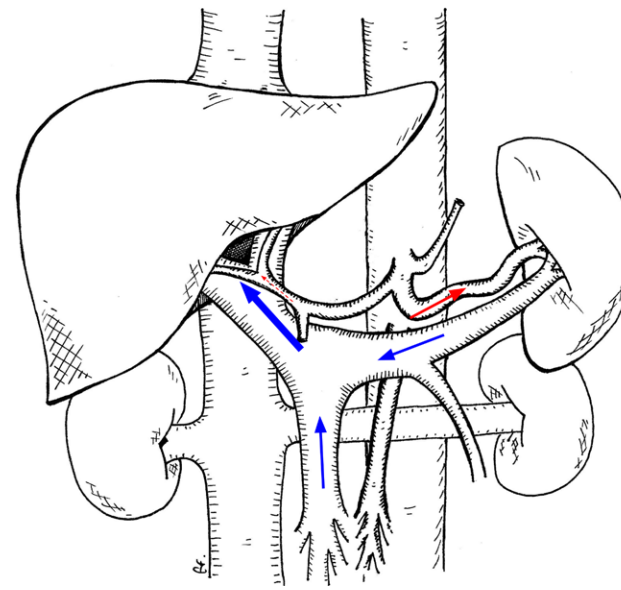

FIGURE 1. "Small for size" or rather "small for flow" syndrome. Previous portal hyperflow due to chronic cirrhosis leads to an increased portal flow to a healthy graft, consequently producing hepatocyte damage due to the excessive flow. Flow through the hepatic artery is diminshed because of the hepatic artery buffer response, and arterial flow is diverted to the spleen because of previous splenomegaly.

can be severely altered in LT context.[13,14] Understanding the relationship between arterial and portal flow is key to making decisions about inflow and outflow modulation. An example of how inflow modulation can affect arterial flow via HABR can be seen in the SASS, as will be discussed.

"SPLENIC STEAL SYNDROME" AND "SPLENIC ARTERY STEAL SYNDROME"

Hepatic artery complications are rare but potentially lethal threats to both graft and recipient. Hepatic artery thrombosis (HAT), for example, has a frequency of 3% to 8% in OLT but mortality rates as high as 50% to 80%, depending on the series.[15,16] Prevention and treatment of HAT has given place to many diagnostic and therapeutic tools, such as Doppler ultrasonography and digital angiography (DA). Ultrasonographic surveillance can evidence a decreased HAF and serves as the first line of detection of hemodynamic alterations in immediate posttransplantation scenarios.[17] A low HAF obeys multiple causes, as previously mentioned. HAT, hepatic artery stenosis, kinking, outflow impairments, and graft rejection are all possible causes of hepatic artery hypoperfusion. Another hemodynamic phenomenon, present in about 3% to 10% of OLT (depending on the series), is "splenic artery syndrome" or "SASS." Originally described by Manner et al[18] in 1991, SASS has been on the radar for >30 y. SASS refers to a clinical entity that presents itself usually in the first 60 d after LT. In its first references, the physiopathological mechanism was thought to be a "siphoning" of arterial blood flow toward the spleen.[19] In ESLD, portal hypertension leads to splenomegaly (>13 cm), with a subsequent increase in the splenic arterial bed and decrease in vascular resistance. This also leads to an enlarged splenic artery. All of these changes were assumed to be the cause of SASS. Its consequence is a diminished arterial flow into the liver, with manifestations varying from a discrete and persistent elevation of enzymes, biliary complications such as biliary leaks, or liver failure with compromise of graft survival. Figure 2 shows hemodynamic changes in SASS.

The diagnosis of this entity is primarily based on clinical suspicion and imaging findings. Doppler ultrasound (DUS) is a useful tool in the evaluation of LT hemodynamics.[20,21] SASS usually exhibits high-resistance hepatic artery waveforms, with low diastolic flows or even reverse diastolic flows. Resistance index (RI) is usually elevated (>0.8). However, RI can be elevated in various clinical conditions, including graft edema, rejection, outflow impairment, or infection.[22,23] SASS may be difficult to differentiate from HAT in many cases, thus usually forcing physicians to more invasive studies. DA is considered the gold standard for detection of SASS.[24] DA findings include sluggish flow in a patent HA (<50% stenosis) and delayed filling of hepatic arteries in comparison to the spleen. Another highly specific but less sensitive angiographic criterion proposed by Uflacker et al[25] is the "catching up" of the portal flow with the arterial flow to the liver. Simultaneous visualization of portal branches with hepatic artery branches suggests that the flow through the spleen and portal system is augmented and "caught up" with the flow directed through the hepatic artery. Some colleagues validly point out that SASS is an exclusion-based diagnosis, meaning that patients must not present HAT to be able to consider SASS as a possible cause. However, not everyone was convinced by the "arterial siphoning" theory. In 2008, Quintini et al[26] challenged this theory,

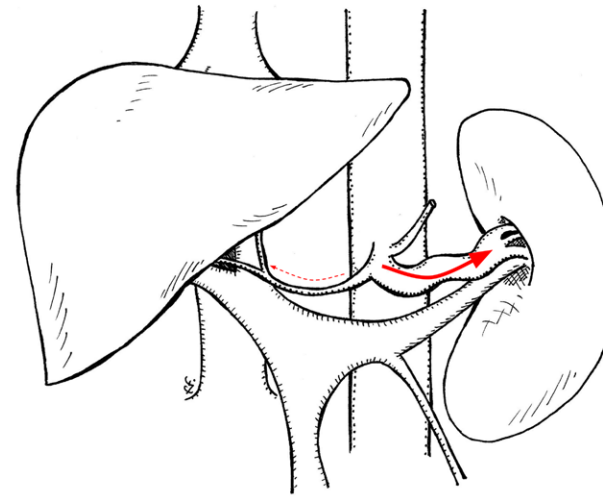

FIGURE 2. Splenic artery steal syndrome. An enlarged splenic artery can be seen, with the majority of the celiac blood flow going toward the spleen (red arrow). Hepatic artery flow can be seen diminished (interrupted red arrow) because of the flow diversion.

aiming the scope toward portal hypertension and hyperflow, as well as the HABR, as the cause of SASS. Intraoperative measurements of PVF, PVP, and HAF showed that HAF greatly varied with portal vein clamping. High PVF leads to high adenosine washout at the hepatic arterioles, depriving arterial flow of a potent vasodilator, therefore, increasing vascular arteriolar resistance. This was how they explained positive effects of portal inflow modulation upon HAF. However, HABR only partially accounts for all SASS cases. Several reports on normal PVF and PVP in SASS patients have led to questioning of theory of Quintini et al.[26] Saad[27] noted 2 situations that raise doubts about the HABR as the lead cause of SASS: The first one is a variant of SASS, gastroduodenal artery (GDA) steal syndrome. In these cases, embolization of the GDA is also the main treatment, but there should be little to no repercussions in portal hemodynamics. Several studies confirm amelioration of HAF when GDA is ligated. Another weakness to the theory put in evidence by Saad[27] is that SASS can be treated with aorto-hepatic conduits, as is shown in different reports.[19,22] Being an intrahepatic mechanism, HABR should be unchanged whether arterial blood supply comes from the celiac axis or directly from the aorta. Recent meta-analysis[28] failed to identify any strong indicators or predictors for SASS. The path to a better understanding and management of SASS seems to lie in intraoperative hemodynamic metrics and even real-time portography.

As previously mentioned, SASS is an exclusion-based diagnosis. Some of these diagnoses include arterial stenosis, kinking, or celiac axis stenosis.

Hepatic artery stenosis occurs in about 3% to 7%[29-31] of all liver transplants. It has been associated with HAT and long-term biliary strictures.[32] Around 80% of patients with HAS develop biliary strictures.[33,34] Its course is usually asymptomatic and has a delayed onset, with a mean time of 100 d.[34] When HAS presents in an early period, its most frequent location is the anastomotic site and is usually related to technical issues and excessive manipulation of the artery.[33] DUS is crucial in its diagnosis, showing typical focal turbulent flow and a peak systolic velocity >200 cm/s,[35] with an intrahepatic Tardus-Parvus wave.[36]

Treatment is aimed at preventing graft ischemia and biliary complications. Best options include percutaneous transluminal balloon angioplasty or stent placement.[37-42]

A differential diagnosis to HAS is arterial kinking. Kinking usually arises from a technical imperfection because of redundant arterial tissue and is a known factor for the development of HAT.[15] It is generally diagnosed intraoperatively, with DUS findings similar with those of HAS, showing elevated peak systolic velocities.[36] The most accepted prevention strategy employed is the use of a "short artery" to avoid redundancy when the liver is placed in its normal position.[42] When kinking is identified, surgeons usually opt for "hepatic artery antikinking methods," which include the use of surgical celluloid or omentum patches to correct the kinking.[43] Preventing anastomotic kinking is a key factor in the prevention of HAT, which, as previously mentioned, can greatly compromise graft survival and is a leading cause of retransplantation.

To conclude, celiac axis alterations must be taken into consideration. Both atherosclerosis and a median arcuate ligament (MAL) can determine a celiac stenosis, compromising the graft's only arterial supply.[44] MAL compression of the axis usually presents as a narrowing 5 mm distal from the ostium with a typical "fish hook" appearance. Diagnosis is usually preoperative, with Doppler findings including 2-fold peak systolic velocities (>200 cm/s) in the celiac artery compared with the aorta with respiratory variation.[45] Confirmation with dynamic imaging (computed tomography [CT] scans/DA) is recommended. Patients with celiac stenosis caused by MAL are commonly asymptomatic because of the development of collateral vessels through the GDA, which are systematically ligated in LT, leaving the graft with the sole supply from the compromised celiac axis. When detected intraoperatively, MAL section can resolve the stenosis. If, despite the section, DUS continues to show low HAF, an aorto-hepatic conduit to salvage the graft should be used.[46]

As seen, excessive portal flow can be deleterious for the graft and may determine a decreased arterial flow via HABR and other mechanisms. However, there are clinical situations where portal flow is diminished, leading to graft compromise. Some of these inflow alterations include portal steal syndrome (PSS) and large for size syndrome (LFSS).

PSS

Portal hypertension as a consequence of cirrhosis is directly related to increased vascular resistance through the liver. Resistance can be so high that portal flow may become hepatofugal and constitute a "portal steal" in ESLD.[47] In these cases, blood flows from the liver to newly developed portosystemic shunts. The main diversion route is through the esophageal and paraesophageal vein plexus. Alternative routes include the retroperitoneum through splenorenal shunts, and the abdominal wall through a permeabilized umbilical vein and cavernous transformation of the hepatogastric ligament. These collaterals usually disappear some time after OLT because of the sudden decrease in liver vascular resistance, but large (>10 mm) vessels may persist even years after OLT.[48,49] There are certain entities that may increase graft vascular resistance, such as rejection, ischemia-reperfusion damage, outflow obstruction, and infection/cholangitis.[50] In these scenarios, obliterated shunts may be reopened, and portal flow may once again become hepatofugal, stealing flow from the graft, jeopardizing its survival. These changes are illustrated in Figure 3.

PSS and portosystemic shunts have long been identified as potential threats to graft integrity.[47-49,51] In 1992, De Carlis et al[47] presented a series of 70 OLTs in ESLD, 3 of which presenting themselves with PSS. Thirty-four patients with preoperative angiography were divided into 3 groups according to the presence and type of shunt present: group 1, those with no shunts; group 2, those with angiographical shunts that were interrupted at transplantation; and group 3, those with large collaterals that were not interrupted in the procedure (splenorenal, inferior mesenteric, and gastroduodenal coronary vein shunts). Significant differences in AST levels at 2 wk were found between shunt and nonshunt groups, as well as rejection rates between these groups. These findings show that the presence of portosystemic shunts have a great influence on graft ischemia, which becomes much more evident when in concomitance with acute rejection.

Early diagnosis is of the essence. DUS is the main element for detection and follow-up of portal flow and portosystemic shunts. Fujimoto et al[48] analyzed results of 83 pediatric LDLTs. DUS was used to analyze portal flow before, during, and after transplantation. Thirty patients presented reversed portal flow preoperatively. After portal vein reconstruction, 22 patients presented portal flows of <10 mL/min/kg of body weight, 6 of which presenting no intrahepatic flow whatsoever. None of these presented technical alterations (ie, outflow impairment, stretching/kinking of anastomosis or thrombosis). After collateral ligation, an increased hepatopetal portal blood flow was seen in 20 patients, and none presented with PSS. The 28 remaining patients with portal flow of >10 mL/min/kg after portal reconstruction did not undergo simultaneous collateral ligation and did not present PSS but did persist with clinically irrelevant patent shunts up to 2 y after transplantation.

PSS is commonly diagnosed by a sum of serological abnormalities, clinical conditions, and serial DUS. DUS findings

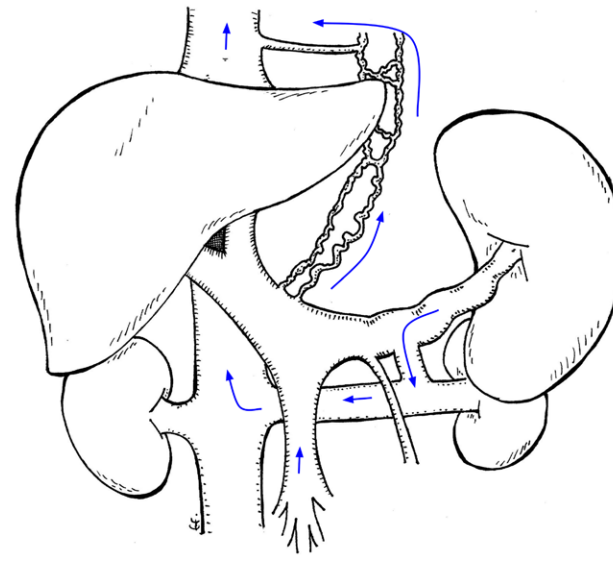

FIGURE 3. Portal steal syndrome. Because of prior high liver resistances due to cirrhosis, alternative venous collaterals may have developed. After transplantation, these enlarged vessels may "steal" the flow away from the liver through various shunts (splenorenal, gastroesophageal, and mesocaval).

may include decreased intrahepatic velocities, bidirectional, or even reversed portal flow.[52-54]

Management and treatment of PSS and shunts still remain a debate. Numerous studies show that surgical shunts (ie, mesocaval shunts) should be closed after LT to avoid flow diversion.[55-58] Several authors advocate for the closure of all shunts (surgical or spontaneous) at the time of surgery, with favorable results.[50] Lee et al[59] presented ligation of the left renal vein as a valid method for intraoperative correction of splenorenal shunts, despite the possible effects on left kidney function and survival. Other authors have validated their findings.[60,61] When these shunts are not corrected before the transplant or intraoperatively, they may be required to be closed afterward. Several studies show the safety, success, and technical feasibility of percutaneous approaches.[62-64]

The main pitfall of portal inflow control by shunt closure lies in ignoring the negative effects of portal hypertension in the newly implanted graft. High portal pressures may derive in graft lesions with ischemia, as previously described in the SFSS/SFFS. The authors believe that the best management of these complications lies in intraoperative "tailoring" of portal flow and pressure, with vascular occlusion tests controlled by DUS to assess whether shunt closure will adequately improve portal flow to the graft or rather damage it by excessive flow.

Although inflow alterations are the most common hemodynamic syndromes, graft survival may also be jeopardized by outflow obstruction. Presentation may be similar to other scenarios, and DUS will be very important in its diagnosis, as seen in the next section.

DO NOT FORGET THE OUTFLOW: VENOUS OUTFLOW IMPAIRMENTS

Acute obstruction to the outflow leads to an elevated intrahepatic sinusoidal pressure and congestion. This translates directly with an elevated RI in portal and arterial flow, potentially compromising graft irrigation and vitality. Signs of hepatic venous outflow obstruction (HVOO) include intractable ascites and right-side pleural effusion, many times in the context of a normal liver function.[65] When associated with hepatocellular impairment, it may mimic acute cellular rejection. Seldomly, HVOO can cause graft failure with the necessity of retransplantation. HVOO is most frequently associated with anastomotic technical issues. Therefore, it is important to correctly detect and diagnose it in the intraoperative period, in which simple measures can be used to correct the alterations. There are some differences regarding the reasons for HVOO in OLT and LDLT/split transplantation that must be addressed to better comprehend its physiopathology.

To limit the deleterious effects of complete caval clamping and removal of the retrohepatic vena cava in the recipient, Calne and Williams[66] in 1968 and Tzakis et al[67] in 1989 developed a novel technique for caval sparring named "piggyback technique" (PB) in which the donor's vena cava was directly sutured to the recipient's hepatic veins. The benefits of this technique include the lack of need of a venovenous shunt and its complications,[68-71] a faster operative time and warm ischemia,[72,73] risk reduction associated with retrocaval dissection, and fewer systemic complications, such as hemodynamic instability, metabolic alterations, and renal failure.

A multicentric retrospective study[74] analyzed vascular complications in different caval-sparring techniques for 1361 patients. HVOO and Budd-Chiari syndrome (BCS) was present in 1.5% (21 patients), making it a rare complication. Eighty percent were diagnosed in the first 24 h. Of these 21 patients, 8 presented with positional BCS, 5 with major graft congestion, 1 with caval thrombosis, and 3 with anastomotic torsion. The complications were resolved using graft rotation or diaphragmatic placement for patients with positional BCS, anastomotic reconstruction with thrombectomy, or even balloon dilation for 2 cases of anastomotic stenosis. Despite its low incidence, HVOO mortality was elevated, with a total of 5 deaths (24%).

In the same year, a Spanish multicentric study[75] specifically analyzed PB technique complications in 1112 patients. A total of 2.5% of the patients had intraoperative complications of PB technique, consisting mainly of graft congestion after revascularization. This was solved in most cases by creation of a "neobed" to adjust size discrepancy between the graft and the hepatic bed. Additional strategies included lateral cavocaval anastomosis or a PG reanastomosis.

In the first week, an additional 1% of the patients suffered a venous complication. Nine patients presented acute BCS in the first 48 h, in which 7 patients had an anastomosis with the recipients' central and left HV. Finally, 3 patients developed chronic BCS in the late postoperative period, which was managed with diuretics. PB-related mortality was low (0.5%). Complications were significantly higher in those patients in which anastomosis was performed with the left and middle HV rather than all 3 veins ($P < 0.05$). This was explained by 3 hypotheses: a larger diameter (>1 cm), a shorter venous segment, and that with a "2 vein patch," the anastomosis is located to the left side of the vena cava, as opposed to the gravitational center of the graft that is located to its right. There are several other series that also report a low but variable incidence of HVOO (0.5%–4.6%)[76-78] in PB-OLT.

A key technical factor associated with HVOO is the use of the recipient "2 vein patch" for the anastomosis instead of using an LHV middle hepatic vein (MHV) + cavoplasty or a LHV + MHV + right hepatic vein orifice. This has been demonstrated by several groups.[74,75,77] A recent study correlates recipient HV patterns with HVOO[79] in PB-OLT. The angle between the recipient main vein trunk and the vena cava and the distance between the confluence of recipient and graft's HV were identified as risk factors for HVOO, with the highest rates of HVOO in patients that had a common LV/MV trunk. A greater angle signified a venous outflow located at the patient's left side, opposed to the graft's center of gravity located in the right side, favoring kinking a turbulent flow.

As can be seen, HVOO in OLT is a rare complication that is most frequently seen in PB reconstruction. Its incidence is directly related to the technical aspects of the anastomosis, specifically to the use of recipient left and MHVs as a "2 vein patch" as opposed to 3 vein patches or the use of cavoplasty. Mortality ranges widely and depends greatly on the moment of diagnosis, which is usually intraoperative at the moment of portal reperfusion. In these cases, the solution is rather simple, being resolved many times with adjustment in graft positioning, fixation of the graft to the anterior wall, creation of a "neobed," or eventually anastomotic reconstruction.

HVOO may also present itself in partial graft transplantation such as LDLT or split grafts for pediatric recipients. These cases share a similar mechanism related to hepatic and venous anatomy. A main concern of right-lobe LDLT

when it is retrieved without the MHV is that it lacks 2 of its usual outflow pathways, the accessory and the MHV.[80] When adequately retrieved, right-lobe grafts have a low incidence of HVOO, as reported by Marcos et al[81] (2/48 patients). Preservation of collaterals and maximization of HV diameters by retrieving veins close to the MHV allow for larger ostiums and better outflow reconstruction. Most centers routinely retrieve the middle hepatic vein and reconstruct segment 5 and 8 veins when backtable flushing is uneven.[82-84] As proposed by Marcos et al,[81] graft survival depends on the intercorrelation of outflow capacity, inflow, and GWRW, in which all 3 factors may determine a "small for flow graft."

There are several strategies to either prevent or treat HVOO. Prevention strategies include modifications in the caval anastomosis, correct positioning, and fixation of the graft to avoid kinking or stenosis. In LDLT, a correct retrieving of the hepatic veins is of the essence.[82,83,85] If HVOO is detected after surgery, there are various therapeutic options. The most common strategy is the use of percutaneous balloon dilation, which has a high success rate, ranging from 70% to 100%.[86-89] Although repeat procedures are often necessary. Stent placement is also a feasible option for both pediatric and adult recipients. Several studies have shown success of stenting in pediatric patients with little to no adverse effects.[86,90] However, several centers have stressed their concerns regarding stent placement in pediatric recipients. Reasons include the possibility of intimal hyperplasia, stent migration, the fixed diameter of a stent that may cause a stenosis when the graft grows in size, and the technical difficulty related to the stent in the case of repeat transplantation.[91,92] Both balloon dilation and stent placement have high success rates in adult recipients as well.

"LARGE FOR SIZE SYNDROME"

LFSS constitutes the other end of size-mismatch complications. It is a rare complication mostly seen in LDLT and pediatric patients. Ever since the first LDLT in 1989,[3] several groups have studied the effects of "small" and "large" LT.[93-96] As previously mentioned, ideal graft size is still a matter of debate, but studies suggest that GWRW should be in the range of 0.8% to 2%.[97]

In pediatric recipients, right- and left-lobe grafts can easily exceed these thresholds, usually in patients <10 kg. A GWRW of >4% has been defined as a "large for size" graft[96,98] and can lead to LFSS. This is a result of graft compression secondary to a tense abdominal wall closure, leading to abdominal compartment syndrome, with inflow and outflow impairment (Figure 4) and low tissue oxygenation,[99] conditioning possible primary graft failure.[100] Reduced left lateral (monosegments) and hyperreduced left lateral (reduced monosegments) are graft reduction techniques that have been developed to prevent LFSS in small infants. Many studies have shown these techniques to be not only safe but also effective, with graft survival rates and overall survival rates similar to OLT and split transplantation.[98,100-105] In 2019, our group published a cohort study of 59 cases of hyperreduced graft transplantation in <10 kg recipients between 1994 and 2018.[106] Our series had overall survival comparable with OLT and split transplantation, with a 1-, 3-, 5-, and 10-y OS of 92%, 83%, 79%, and 74%, respectively. Only 2 patients required repeat transplantation. The study concluded hyper reduced graft transplantation to be a feasible, safe, and effective technique for infants with ESLD in which LT cannot be delayed until adequate size is achieved.

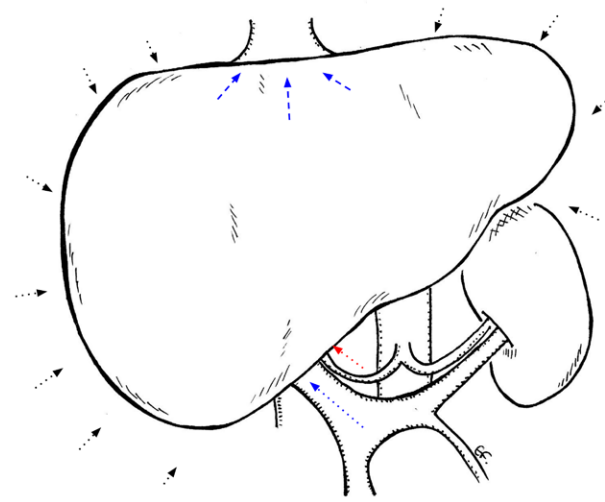

FIGURE 4. "Large for size" syndrome. Extrinsic compression to the liver (black arrows) determines higher pressures, thus diminishing arterial and portal flow toward the liver (blue and red dotted arrows), as well as venous drainage from the hepatic veins toward the inferior vena cava (blue interrupted arrows).

Several indexes and parameters that were validated to determine the cutoff point from which a graft is no longer safe to transplant because of its large size are still a matter of debate. For LDLT, GWRW and SLV are widely used and accepted as tools to estimate donor-to-recipient size mismatch.[107]

LFSS is rarely seen in adult OLT. The main reason is the availability of a wider donor pool, which reduces the chances of size mismatch. Another variable that "prevents" LFSS in adult OLT is the existence of chronic hepatopathy. Patients with ESLD have a complacent abdominal cavity because of ascites and hepatosplenomegaly, thus allowing larger discrepancies between the new graft and the recipient cavity.

Despite these "protective measures" against LFSS in OLT, the use of marginal livers in obese patients with steatosis and OLT in the context of a fulminant hepatitis with no time for abdominal cavity compliance may determine an LFSS in OLT recipients. To avoid size and diameter discrepancies, several formulas have been developed.

Fukazawa et al[93,94] attempted to validate the body surface area index (BSAi) (BSA of the donor/BSA of recipient) for OLT. In 2011, they analyzed results of 1228 OLTs, stratified into 3 groups: BSAi <0.6, BSAi 0.6 to 1.4, and BSAi >1.4. They concluded that BSAi was an accurate correlate to SLV for OLT, in which volumetry is not a possibility, because of the urgency of the matter. They also observed poorer outcomes regarding overall survival and graft survival on either extreme of BSAi (<0.6 and >1.4 BSAi).[93] In 2013,[94] a total of 24 509 patients were included in the analysis, concluding that BSAi >1.24 resulted in decreased graft survival. Other authors have used different indexes, such as a GWRW of >2.5%,[108] a standardized total liver volume recipient total liver volume ratio of >1.25[109] and even a ratio between the graft weight and the anteroposterior diameter of >100.[110] The greatest limitation of these calculations is the margin of error when calculating cirrhotic recipients' weight,[111] which can be influenced by edema, ascites, malnutrition, and sarcopenia. A recent meta-analysis

identified that patients at risk for LFS OLT were female, were small (62.5 kg), and underwent transplantation for fulminant hepatitis or in the context of retransplantation.[112] Small recipients in urgent need for an organ (fulminant hepatitis), a hostile abdomen (retransplantation), and size and morphological mismatch (male donors) were all present in the patients. Mortality of 16% was identified in this group. This study failed to determine superiority of a formula over another.

When unable to further reduce graft size to accommodate it to a small abdominal cavity, abdominal wall closure strategies are used to prevent LFSS. One of the strategies used in low-weight recipients is skin closure or partial skin closure. Sometimes even adequate or even small GWRW requires skin closure because of the discrepancies in graft shape, especially in patients with fulminant hepatic failure, in which cirrhosis and ascites have not yet altered normal anatomy.[100] Primary abdominal wall closure may be considered when portal flow can be measured before and after wall closure to determine any compromise in graft blood flow. Akdur et al[113] reported 18 LFSSs in their study of 58 pediatric patients <10 kg. In their study, 10 patients were diagnosed by DUS after abdominal wall closure. Another valid technique is Bogota bag closure of the abdominal wall. Several other studies advocate for vacuum techniques to assist in abdominal closure and prevent LFSS.[114,115] Closure strategies attempt to lower abdominal pressures after transplant to reduce risk of abdominal compartment syndrome and its effects on graft perfusion.

DIFFERENT ENTITIES OR SIMPLY DIFFERENT FACES OF THE SAME COIN? A CASE THAT LINKS THESE SYNDROMES

A 50-y-old female patient with diagnosis of primary biliary cirrhosis was admitted for OLT. ABO group was 0+ and MELD 34. Height was 149 cm and weight 43 kg. Xiphoid perimeter was 86 cm. The patient had a history of portal vein thrombosis (Figure 5). Donor was a 63-y-old male patient deceased from a hemorrhagic stroke with a height of 153 cm, weight of 60 kg, and xiphoid perimeter of 80 cm. Donor weight to recipient weight ratio was 1.39, with the recipient presenting a wider xiphoid perimeter than the donor; thus, the anthropometrics were deemed adequate. An uneventful OLT was performed with no technical difficulties. Caval anastomosis was fashioned using PB technique, and an end-to-end choledo-choledocal anastomosis was performed, with a total ischemia time of 5:20 h. Intraoperative reperfusion syndrome was managed with epinephrine, calcium, and bicarbonate. Intraoperative DUS showed adequate portal and arterial flows and RI, even after closure.

Immediate DUS showed no alterations. Twelve hours later, the patient developed a tense abdomen and laboratory results with elevated liver enzymes, lactic acid, and hemodynamic repercussions, suggesting graft ischemia. Urgent DUS revealed hepatic vein thrombosis extending onto the PB, as well as diminished arterial flow, with bidirectional portal flow. Suspecting LFSS and concomitant abdominal compartment syndrome, emergency laparotomy was performed. An extremely congestive graft with disseminated venous thrombosis was identified (Figure 6). Anastomotic revision was performed with thrombus cleansing, and cavocaval shunt was performed to divert blood flow from the congestive liver, observing a slight improvement in ultrasound flows. Vacuum abdomen technique was used for wall closure to avoid a new LFSS.

The patient continued in critical condition, with laboratory works showing graft failure and DUS with little to no improvement, and was interpreted as graft failure secondary to extreme LFS. Because of lack of an immediate new donor and "toxic liver syndrome," graft explant was performed with inferior vena cava preservation and a mesocaval shunt for bowel decompression, as shown in Figure 7. The patient remained on the emergency waiting list for an additional 30 h, and because of lack of donors, the first available compatible organ was allocated. The new donor was a 63-y-old woman with a height of 169 cm, weight of 80 kg, and xiphoid perimeter of 95 cm. Initial anthropometrics did not require graft reduction a priori (donor weight to recipient weight 1.86), and the use of a wall-expanding mesh (Gore-Tex) was foreseen. Additionally, because of the long anhepatic period the patient was subject to, the goal was to spare the most amount of functioning liver tissue as possible. However, the wide anteroposterior diameter of the donor liver made the graft fit unevenly into the receptor's abdominal cavity, compromising its inflow, which was observed both macroscopically and by DUS. Because of these circumstances, an in situ right hepatectomy was performed to reduce liver size (Figure 8).

The patient evolved slowly but favorably after repeat transplantation, with clinical improvement and normalization of blood tests and DUS values. The patient developed a transection line fistula and biloma, which was managed with percutaneous drainage. In the following 20 d, the patient

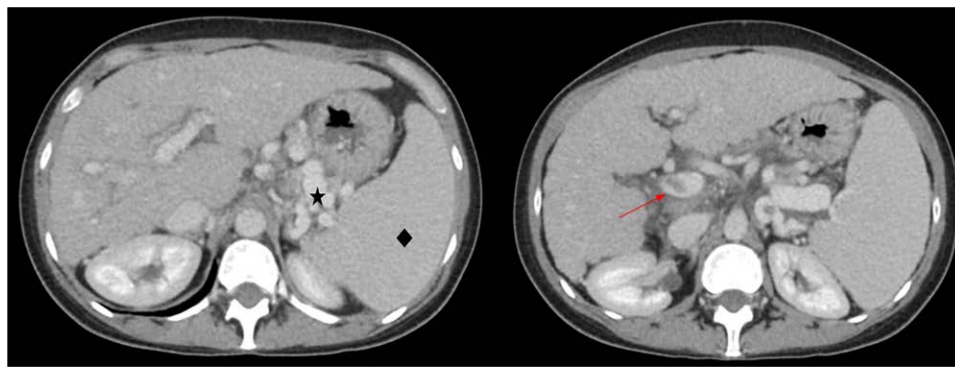

FIGURE 5. Preoperative CT scan. The first image shows splenomegaly marked with a black diamond and prominent collateral esophageal vessels marked with a star. The second image shows portal vein thrombosis marked with a red arrow. CT, computed tomography.

presented with high ascitic output from the surgical drains and a slowly but steadily worsening liver function (TBil, prothrombin, and factor V levels) with consecutively normal DUS. MRI and biopsy were performed without diagnosis. In this context, the patient was diagnosed with SFSS. Although the patient had undergone right hepatectomy, the remaining liver was >0.8 GWRW. Reviewing images before the OLT, we observed that the patient had splenomegaly and a large splenic artery. Suspecting a possible SASS, the patient was submitted to DA with splenic artery embolization (Figure 9). Immediately after embolization, HAF greatly improved, and portal flow diminished. The patient had a favorable outcome, with normalization of hepatic function and decrease of ascites. The patient is alive and well 3 y after transplantation. Bilirubin levels are shown in Figure 10.

CONCLUSIONS AND TAKEAWAY MESSAGES

Although rare in most transplant centers, these syndromes may occasionally occur. Transplant teams must have very clear concepts on how to avoid and treat them.

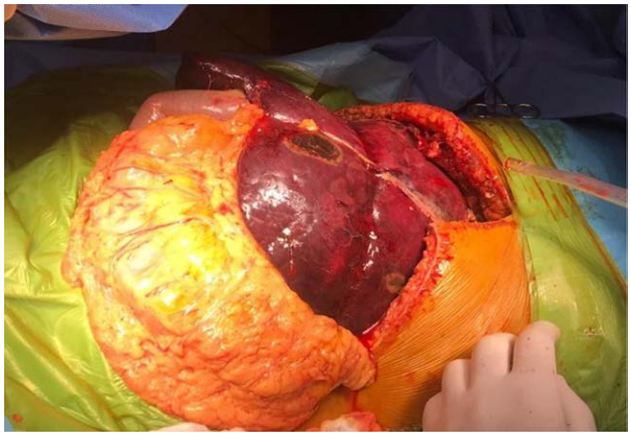

FIGURE 6. Liver graft at surgical revision. An extremely congestive, enlarged liver can be seen, with disseminated venous thrombosis and ischemic areas.

DUS has proved an extremely useful tool in the early diagnosis of these syndromes. Confirmation is usually required in the form of an invasive study such as DA but has the benefit of diagnosing and potentially treating the condition. It is rather uncommon that any of these conditions require surgical intervention if diagnosed early on, and graft failure and mortality are decreasing because of a better understanding of these complications. Pretransplant CT scan has proven useful in LDLT. Its benefits lie in its ability to esteem the remaining liver volume, detect vascular abnormalities, and assess liver parenchyma for unseen fibrosis/cirrhosis or other pathologies. In the setting of a deceased donor, time is of the essence, and availability of equipment and specialized radiologists can often be scarce, especially in developing countries in which deceased donors may be available in low complexity centers with no immediate availability of CT scan. Given these limitations, there are almost no studies that assess deceased donor volumetrics and its correlation with SFS and LFS grafts. However, there are various reports of postmortem liver weight CT measurements, which predict liver weight with <160 gr of error.[116] Quite recently, Robb et al[117] analyzed the impact of pretransplant CT on deceased donor transplantation. They concluded that pretransplant CT aided in decision making in almost a third of the cases studied, surpassing body mass index as a significant decision-making factor. Indeed, a CT scan might greatly aid to avoid size discrepancies and greatly facilitate the transplant process but still must be further developed.

Inflow and outflow alterations derived from prior patient conditions and size abnormalities must be weighed into the diagnosis when assessing graft function impairment, especially when technical issues have been ruled out. As seen previously, many of these syndromes have direct correlation with each other, and must be assessed thoroughly, to avoid shifting hemodynamics from one end to another, and potentially damaging the graft, as happened with the case shown (from LFSS to SFSS).

Understanding the complexity of these scenarios, our group has attempted to develop a simple algorithm to assist decision making when any of these situations arise. This algorithm is shown in Figure 11.

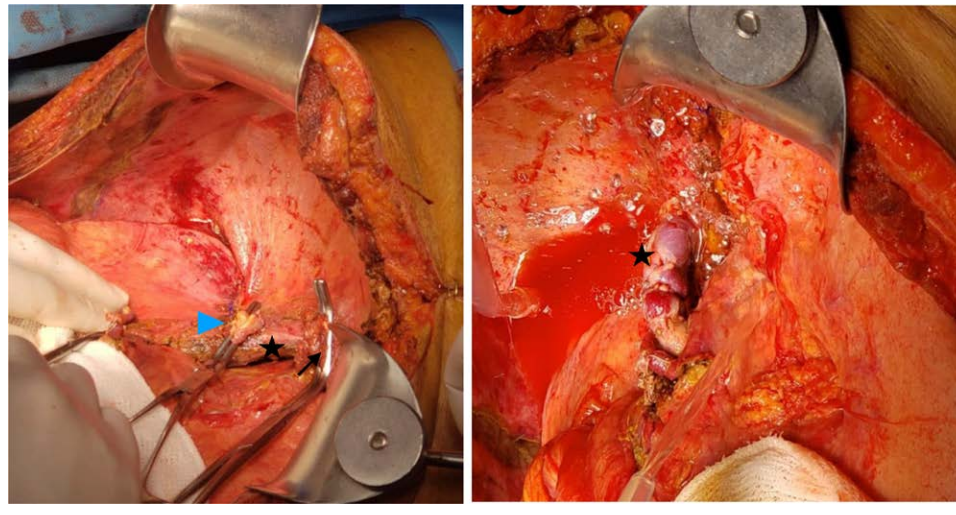

FIGURE 7. Anhepatic phase. In the first image, inferior vena cava (black star), hepatic veins (black arrow), and portal vein (arrowhead) can be seen after graft explant. The second image shows the portocaval shunt (black star) performed to divert the splanchnic flow toward systemic circulation.

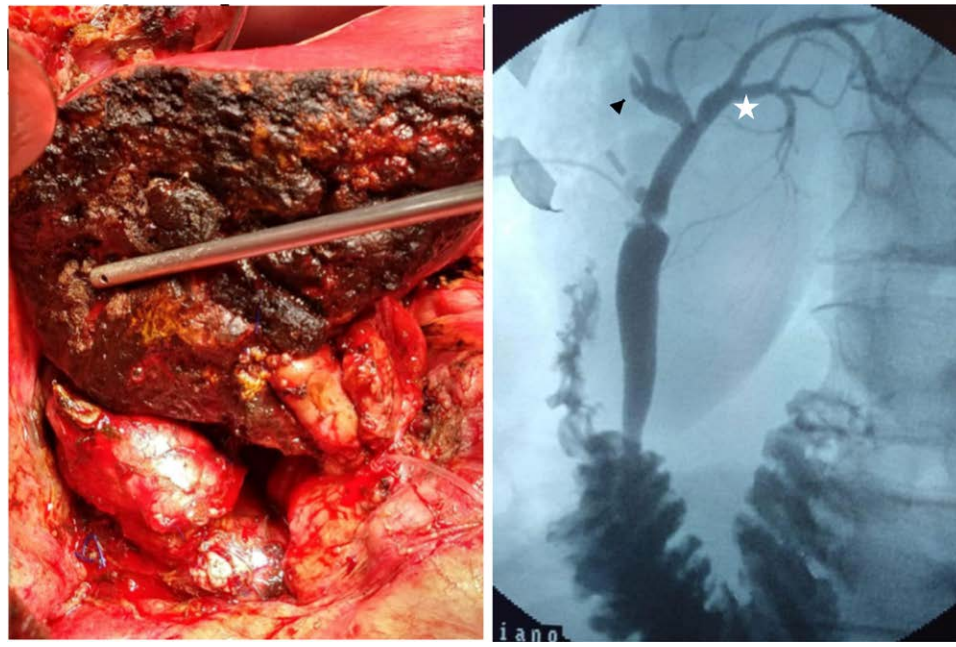

FIGURE 8. In situ hepatectomy. Section border after in situ right hepatectomy. Complete left bile duct system can be seen in the intraoperative cholangiography (white star) and the sectioned right-side ducts (arrowhead).

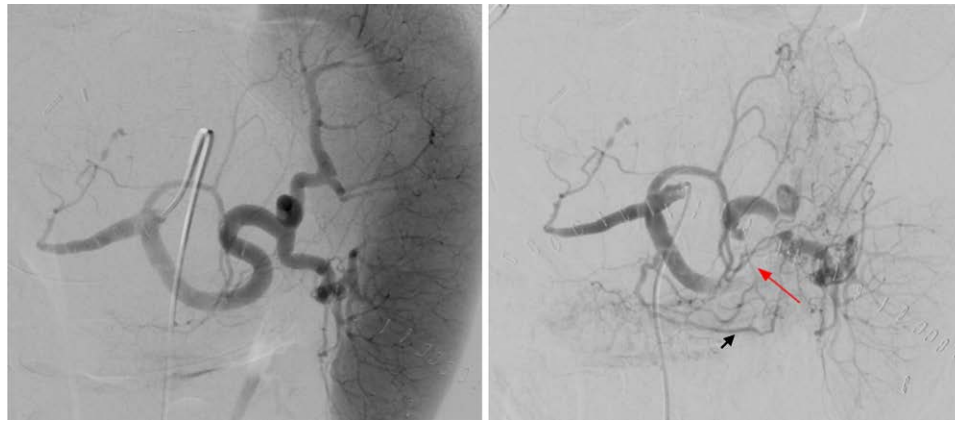

FIGURE 9. Digital angiography of the celiac axis. First image shows sluggish flow in the hepatic and coronary arteries, with a quick splenic filling. The second image was taken after splenic artery embolization with coils. Hepatic artery filling is no longer sluggish, and splenic vessels are no longer prominent (red arrow). Splenic irrigation is provided by collateral vessels such as gastroepiploic vessels (arrowhead).

In conclusion, although rare entities, hemodynamic phenomena can greatly disturb a successful transplant if not diagnosed and treated early on. To do so, DUS and DA are essential tools. However, most importantly, the team must always keep in mind that portal hemodynamics are anything but static and consider the particularities of each recipient in the preoperative and intraoperative moments to detect and avoid possible complications.

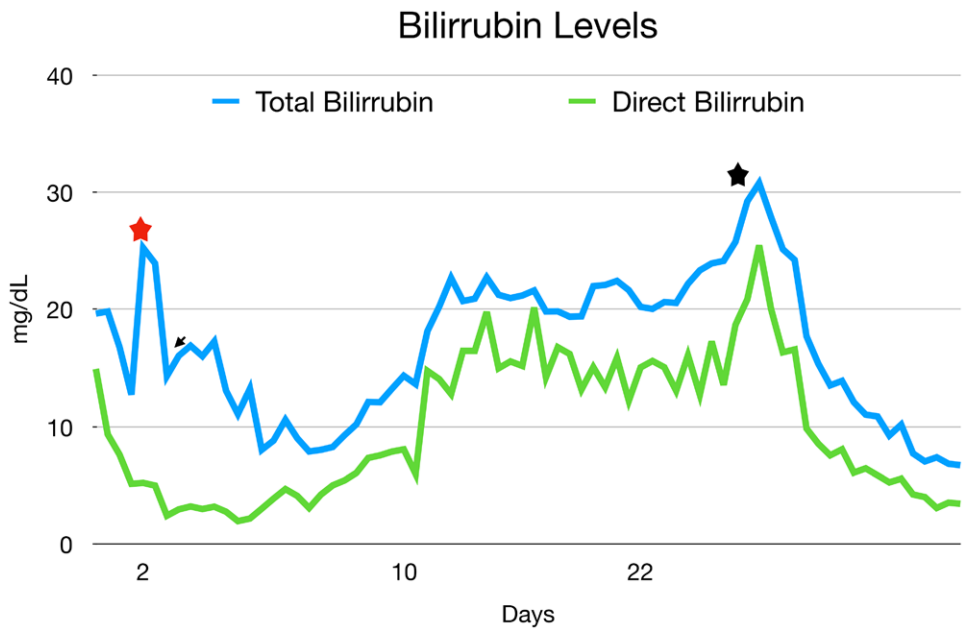

FIGURE 10. Bilirubin levels following transplantation. The red star marks peak levels at graft explant (day +2). The black arrowhead marks the moment of repeat transplantation with improvement in total and direct bilirubin levels. After day 10, a progressive increase in bilirubin levels can be seen up until the moment splenic artery embolization is performed (black star), with immediate decrease afterward.

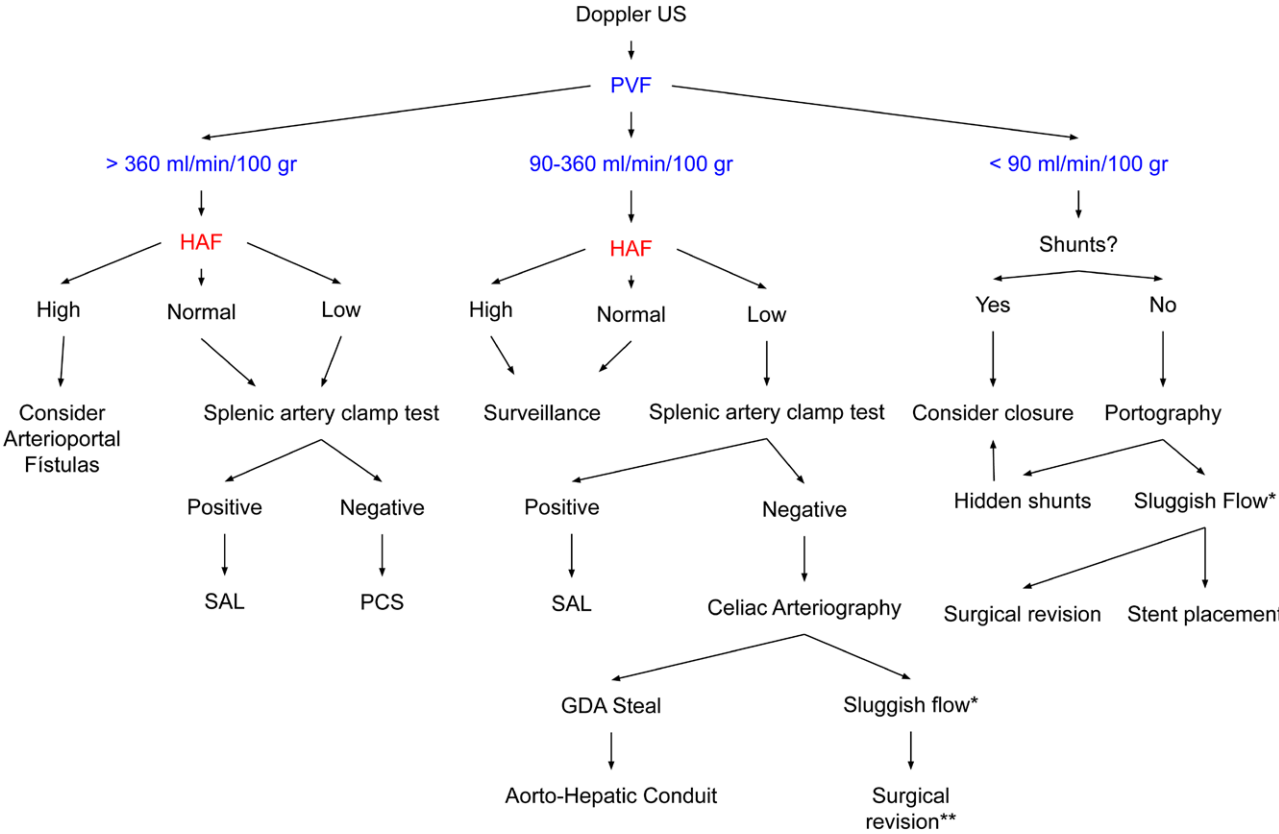

FIGURE 11. Algorithm for intraoperative diagnosis and management of hemodynamic syndromes. Efforts should be made to obtain measurements in optimal systemic conditions and optimal graft outflow. Doppler US should be used intraoperatively as the first diagnostic tool, and satisfactory PVF measurements should be acquired. After PVF measurements, HAF should be obtained. Once PVF and HAF have been measured, the surgeon can use objective values to evaluate the existence of small-for-size syndrome, splenic artery steal syndrome, and portal steal syndrome and perform additional tests (splenic clamp test and angiography/portography) to determine whether inflow modulation procedures (SAL, PCS, aorto-hepatic conduits) or surgical anastomotic revision are necessary. *Sluggish flow: most likely due to anastomotic stenosis, consider either surgical revision or stent placement. **Surgical revision: when considering anastomotic revision of hepatic artery reconstruction, assessment of celiac trunk anatomy and the possibility of an arcuate ligament syndrome is in order. GDA, gastroduodenal artery; HAF, hepatic artery flow; PCS, portocaval shunt; PVF, portal vein flow; SAL, splenic artery ligation; US, ultrasound.

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