Nonocclusive Hepatic Artery Hypoperfusion Syndrome (Splenic Steal Syndrome) in Liver Transplant Recipients

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Abstract

Keywords

- transplant
- liver
- vascular complications
- splenic steal
- hypoperfusion
- buffer response

There are numerous causes of reduced arterial inline flow to the liver transplant despite a patent hepatic artery. These include causes of increased peripheral resistance in the hepatic arterial bed, siphoning of the hepatic arterial flow by a dominant splenic artery (splenic steal syndrome), functional reduction of hepatic arterial flow in response to hyperdynamic portal inline flow, and small hepatic graft relative to normal portal inline flow (relative increase of portal flow). These causes are incompletely understood, and perhaps the most controversial of all is the splenic steal syndrome, which is possibly an underrecognized cause of graft ischemia in the United States. Splenic steal syndrome presents nonspecifically as graft dysfunction; if overlooked, it may lead to graft failure. Its incidence is reported to be 0.6 to 10.1% in liver transplant recipients, with some institutions performing prophylactic and/or posttransplant treatment procedures in up to a quarter of their transplant recipients. This wide disparity in the incidence of the diagnosis is probably because there are no objective diagnostic imaging criteria. This article presents a review of the literature that addresses the differential diagnostic considerations of hepatic artery hypoperfusion (splenic steal syndrome included) in the absence of an anatomical defect (hepatic artery stenosis, thrombosis, and/or kinks).

Objectives: On completion of this article, the reader will be able to describe the nonocclusive hepatic artery hypoperfusion syndrome, and understand the diagnosis and treatment options for patients presenting with the syndrome.

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Splenic steal syndrome is a controversial diagnosis of hepatic artery hypoperfusion and may represent an under-

recognized cause of graft ischemia in the United States.^{1–3} Splenic steal syndrome presents nonspecifically as graft dysfunction and, if overlooked, may lead to graft failure.^{4,5} Its incidence in the literature ranges widely from 0.6 to 10.1% in liver transplant recipients^{2,4,6–13} with some institutions performing prophylactic and/or posttransplant treatment procedures in up to 22 to 26% of their transplant recipients^{12,13} (► **Table 1**). This wide disparity in the incidence of the diagnosis is probably because there are no objective diagnostic imaging criteria.³ It has been proposed that the condition is probably underrecognized in certain institutions (particularly in the United States and Japan), and it is probably overdiagnosed in others (particularly in Germany and Austria).³ The subject is being discussed in this issue of *Seminars of Interventional Radiology* because it has a cause-and-effect relationship

Table 1 Incidence of Splenic Steal Syndrome Posttransplantation in Studies Evaluating More Than 50 Transplant Recipients

Study and Year	No. of Splenic Steal Syndrome Cases	Incidence (%)
Rasmussen et al ²	N = 1/70	1.4
Geissler et al ⁶	N = 1/165	0.6
Uflacker et al ⁷	N = 11/350	3.2
Nüssler et al ⁴	N = 44/1171	3.8
Sevmis et al ⁸	N = 10/118	8.4
Kirbas et al ⁹	N = 10/198	5.1
Granov et al ¹⁰	N = 3/54	5.6
Mogl et al ^{11*}	N = 28/553	5.1
Grieser et al ^{12*}	N = 12/119	10.1
Total 1997–2010		
(N > 50 transplants/study)	N = 120/2798	4.3

*These studies have 22 to 26% of transplant recipients undergoing prophylactic procedures and/or treatment for splenic steal syndrome. The incidences in the table are the posttransplant incidences in recipients who have *not* undergone prophylactic procedures.

on the portal hemodynamics of liver transplant recipients. This is a review of the entire literature that addresses the differential diagnosis of hepatic artery hypoperfusion in the absence of an anatomical defect (hepatic artery stenosis, thrombosis, and/or kinks).

Clinical Presentation

Hepatic artery hypoperfusion, including splenic steal syndrome and gastroduodenal steal syndrome, in the absence of occlusive disease of the hepatic artery usually presents in the early posttransplant period with >80% diagnosed within 60 days of liver transplantation.^{3,4,6,8,11,14,15} Hepatic artery hypoperfusion presents with nonspecific graft dysfunction as evidenced by abnormal liver function tests.^{1–4,6–13,16–20} In the later posttransplant period (after 2 months from the liver transplant), patients may present with ascites (again, a nonspecific finding) as an overt form of hepatic dysfunction.³

Subjective Imaging Diagnosis

Angiography is the gold standard diagnostic modality in the evaluation of splenic steal syndrome.^{1,2,4,6–12} The diagnosis is defined by sluggish flow in the hepatic artery (subjective slow flow in the hepatic artery relative to the flow in the splenic artery) in the absence of significant (>50% arterial diameter reduction) arterial anatomical defects such as hepatic arterial stenosis, thrombosis, and/or kinks^{1,2,4,6–12} (► **Fig. 1A**). However, arterial flow in the hepatic artery is usually less impressive than in the splenic artery because the spleen is normally more vascular than the liver from an arterial/arteriography standpoint. This is because ~20 to 25% of the blood flow to the liver is arterial, and 100% of the blood flow to the spleen is arterial. Furthermore, liver transplant recipients are commonly cirrhotic patients with years of underlying portal hypertension, and, as a result, these patients have a high likelihood of having splenomegaly at baseline.

The most objective angiographic definition was provided by Uflacker and coworkers who only diagnosed splenic steal syndrome when there was visualization of the hepatic artery during the portal-venous phase of the angiogram.⁷ In other words, the contrast has gone through the splenic vasculature into the intrahepatic portal vein, “catching up” with the contrast that is still seen in the hepatic artery. This finding represents very slow hepatic arterial flow. This definition, however, may set the threshold too high, to the extent that the sensitivity of this angiographic finding is so low that operators would be missing a potentially significant number of splenic steal cases. By this definition, however, there may be such a severe form of splenic steal syndrome to the extent that there is a state of increased/hyperdynamic portal flow. Other authors diagnosing splenic steal syndrome have not used this threshold as a definition of splenic steal syndrome.^{1,2,4,6,8–12}

Other investigators have attempted to evaluate the incidence of splenomegaly, and they have reported that a splenic volume >830 cm³ has a 75% accuracy in diagnosing splenic steal syndrome.^{9,12} Nominal splenic artery diameters >4 mm and/or a splenic artery to hepatic artery diameter ratio >1.5 are also considered significant indicators of splenic steal syndrome.^{9,12} However, as mentioned earlier, most transplant patients have had chronic cirrhosis prior to their transplantation, and therefore splenomegaly and splenomegaly-related findings are not uncommon.³ These, in my opinion, are probably secondary/supporting findings and do not make the diagnosis.

Doppler ultrasound findings are not specific and are not commonly or consistently described in reported splenic steal syndrome cases.^{5,11,14,21} In fact, of 12 studies reporting 113 arterial steal cases in liver transplant recipients, only 34 (30%) of cases had descriptions of Doppler findings.^{3–9,11,13,14,22} The most commonly described Doppler finding is an abnormally high arterial resistive index (RI: >0.80).^{5,7,11,13,14,21,22} Actual arterial velocities are rarely reported.^{3,22,23}

Figure 1 Middle-age woman status post-liver transplantation 3 months previously, presenting with abnormal liver function tests (dysfunctional graft). Liver biopsy was suggestive of ischemia. (A) Selective image from a celiac angiogram of the liver transplant recipient demonstrating delayed arterial flow (open arrow) in the hepatic artery with no peripheral filling of the hepatic arterial distribution at this stage of the angiogram. In contrast, the splenic artery is filling peripherally into the spleen (Sp). (B) Selective image from a celiac angiogram of the same liver transplant recipient after balloon-test occlusion (asterisk) of the proximal splenic artery. This is the same catheter positioning, injection rate and volume, and frame within the angiogram as (A). The distal splenic artery (open arrows) is filling distally via reconstitution via the left gastroepiploic (LGA-PA) and left gastric arteries (LGAs) and continues to supply the spleen (Sp). In contrast with (A), obtained before the temporary splenic artery occlusion, the peripheral hepatic arterial branches are now filling in normal fashion.

Commonly, when Doppler parameters are mentioned, the arterial RI of the hepatic artery is 1.00 due to reversed end diastolic volume.^{13,21,22} However, transiently high RI in the early postoperative period, when most splenic steal cases are diagnosed, is not uncommon, although its pathogenesis is not understood. One explanation for the transiently high arterial RIs in the early postoperative period is increased resistance in the peripheral hepatic arterial vasculature.^{11,21,24}

In cases of splenic steal syndrome, portal vein velocities are sporadically reported. When reported, portal vein velocities have been valued at >50 cm/second and may be up to 150 cm/second.^{5,13,14} However, portal vein velocities have also been reported as low as 13 to 37 cm/second in the setting of splenic steal syndrome.^{12,23,25} An interesting and invari-

able Doppler ultrasound phenomenon, when mentioned, is the response of the Doppler parameters to splenic embolization. In this setting, the hepatic arterial RI decreases from that seen pre-embolization, as well as an increase in the hepatic arterial velocities and a decrease in the portal venous velocities.^{3,5,13,14}

Pathogenesis and Hemodynamics of Hepatic Artery Hypoperfusion in Liver Transplant Recipients

One cannot attempt to discuss the diagnosis splenic steal syndrome without discussing the hemodynamics of the splanchnic vasculature and the various hypotheses behind the cause of splenic steal syndrome.³ Unfortunately, splanchnic hemodynamics are not fully understood, adding to the difficulty in making this diagnosis and provoking a controversy regarding the exact cause of the hepatic artery hypoperfusion in splenic steal syndrome. Initially when it was first described (1990), splenic steal syndrome was considered a true arterial flow problem with flow preferentially going to the splenic artery and being diverted away from the hepatic artery (a true steal phenomenon), hence the name of the syndrome.^{16–18,26} However, in 2008 it was proposed that the hepatic arterial hypoperfusion is in response to portal venous hyperperfusion, mediated by the hepatic arterial buffer response (HABR).^{13,27} Quintini and coworkers made a very important observation, which is that these patients have high portal venous flow represented by a increased portal vein velocity (the presumption is that portal vein diameter remains the same).¹³ These investigators were rather dogmatic about the sole causal relationship between portal hyperperfusion and hepatic artery hypoperfusion leading to splenic steal syndrome.¹³ However, the hypothesis of portal hyperperfusion as the sole etiology of “splenic steal syndrome” was challenged by Saad.³ From a scientific standpoint, making a finding that correlates well with a disease process does not confirm causality, especially in such a complex and poorly understood hemodynamic setting.³ The finding of high portal venous velocity/flow could be a secondary effect or contributing factor and not necessarily the cause. I do agree with Quintini and coworkers, however, that adequate response by occluding the splenic artery does not necessarily support the steal theory over the portal hyperperfusion theory because embolizing the splenic artery does also reduce the portal venous flow.^{3,11,13}

In the previous report, I explained there are two subsets of this broader hypoperfusion “steal” phenomenon that were overlooked and do not fit with the theory suggested by Quintini and coworkers.^{3,13} (►Figs. 2 and 3). First is the gastroduodenal artery (GDA) steal cases. GDA steal is a recognized steal phenomenon similar to splenic steal, which represents up to 18% of steal cases in liver transplant recipients.^{4,8,11,16,19} I doubt that embolizing the GDA has a significant effect on the portal venous flow.³ This challenges the explanation that splenic steal syndrome is just a portal venous hyperperfusion phenomenon.³ Moreover, Nishida and coworkers described a GDA steal case with pre- and

Figure 2 Schematic of the splanchnic circulation demonstrating the full circle between the celiac axis (CAx), the spleen, the splenic vein, the portal vein, and the liver. To complete the circle is the mediation of the portal venous inflow with the hepatic arterial inflow of the liver by the hepatic artery buffer response (HABR).

post-GDA ligation in which they described arterial Doppler ultrasound measurements (unfortunately, the portal vein velocity was not disclosed).²² From pre- to post-GDA ligation, the peak hepatic arterial systolic velocity increased from 13 to 79 cm/second, the end diastolic velocity increased from zero to 23 cm/second, the mean arterial velocity increased from 4 to 49 cm/second, and the arterial RI decreased from 1.0 to 0.71.²² These findings mirror those of studies describing

Doppler findings post-splenic artery embolization for splenic steal syndrome.^{5,13,14}

The second aspect that does not fit the Quintini hyperperfusion theory is that arterial steal phenomena (GDA and/or splenic) do not occur in liver transplant recipients with direct aorto-hepatic conduits.^{1,11} In fact, aorto-hepatic conduits are a treatment of posttransplant arterial steal.^{6,7,17,21} If the arterial hypoperfusion is in response to the portal hyperperfusion, then the arterial flow in the conduit should also respond in like fashion (unless one speculates that a conduit makes the hepatic artery less denervated and/or makes the hepatic artery “extra-splanchnic”).³ Conversely, the HABR is accepted to be an intrinsic intrahepatic mediator due to intrahepatic adenosine washout by the hyperdynamic portal circulation.^{28–32} Adenosine is a known vasodilator that naturally accumulates in the liver and helps regulate hepatic arterial flow. When portal flow increases, the influx of portal venous flow washes the adenosine out of the hepatic vascular bed ridding the hepatic arterial flow of this vasodilator, thereby causing a relative hepatic artery vasoconstriction.^{28–32}

Saad added that Quintini and coworkers are not necessarily wrong (they may very well be partly correct), but merely that they have no proof of causality and that their important finding does not fit all “arterial steal scenarios.” There may be several causes/types of scenarios with multiple predisposing factors and/or precipitators.³ Unfortunately, there are so many variables affecting the splanchnic circulation that it is impossible at this time to make any assertion as to the cause (s) of splenic steal syndrome/subjective hepatic artery hypoperfusion.³

The complexity of the governing hemodynamics of the splanchnic circulation can be simplified if explained in a fluid-mechanics manner. —Figs. 2 and 3 help demonstrate the numerous variables that can affect hepatic arterial inflow. Simply, it is a partly reciprocal relationship between two arterial components in balance, and two venous components that are relative to one another. The arterial and venous components are intertwined/mediated by the partly reciprocal HABR. Unfortunately, the details of what is known and what is hypothesized about the HABR is beyond the scope of this article.^{28–32} The partly reciprocal relationship between the hepatic arterial inflow and the hepatic portal venous inflow is simplified in —Fig. 4.^{28–32}

From the arterial standpoint, there is a hypothetical balance between the resistance of the splenic arterial bed and the resistance of the hepatic arterial bed. A low resistant splenic bed relative to a higher resistant hepatic bed would favor flow toward the spleen (splenic steal). This has been partly proposed by prior authors, where they proposed that causes of increased hepatic arterial resistance may aggravate the splenic steal condition.^{1,2,4,7,8,21,22} These posttransplant conditions include preservation injury, stiff graft (poorly compliant graft), postoperative edema, large subcapsular hematoma, graft rejection, and abdominal compartment syndrome.^{1,2,4,7,8,21,22} Rarely have prior authors included significant hepatic arterial stenosis (>50% diameter reduction) as a superadded condition causing increased hepatic

Figure 3 Schematic of the splanchnic circulation showing the hemodynamic balances between the splenic peripheral vascular resistance (splenic vasc. bed) and the hepatic peripheral vascular resistance (hepatic vasc. bed) on the arterial side of the splanchnic circulation. On the portovenous side of the splanchnic circulation, there is a balance between the graft size (and graft quality) and the portal venous flow itself. Mediation between the arterial (top half of schematic) and the venous (bottom half of schematic) circulation is by the hepatic arterial buffer response (HABR), which is a partly reciprocal relationship between the hepatic arterial and hepatic portovenous inflow (Fig. 4).

Figure 4 Simplified schematic of the hepatic arterial buffer response (HABR), which is a partly reciprocal relationship between the hepatic arterial and hepatic portovenous inflow. This relationship is incompletely understood in both native livers and after liver transplantation. The HABR is speculated to be altered in the relatively denervated transplant hepatic graft, but in what way and to what extent, if any, is unknown. At baseline (normal, top row), ~30% of the hepatic arterial inline flow shunts to the portal circulation. The middle rows shows the largely agreed-upon arterial and portal responses. The response specific to nonocclusive hepatic artery hypoperfusion syndrome or splenic steal syndrome is an increase in inline portal venous flow (hyperdynamic portal circulation), causing a hepatic arterial response with a concomitant reduction of inline arterial flow and a reduction of the hepatic artery-to-portal shunting (more than the normally expected 30% of hepatic arterial flow). The HABR may be mediated by adenosine washout. Adenosine, a known vasodilator, accumulates in the liver. When inline portal venous flow increases, it washes out the adenosine and its vasodilator effect, leading to vasoconstriction, an increase in peripheral arterial resistance, and diminished hepatic arterial flow.

arterial resistance and thus accentuating splenic steal syndrome.⁴ As do most authors, I consider splenic steal syndrome and hemodynamically significant hepatic arterial stenosis as mutually exclusive.^{1,21,22} In other words, one cannot diagnose splenic steal syndrome in the presence of a significant hepatic arterial stenosis, kink, or hepatic artery thrombosis.^{1,3,21,22}

From the portal venous standpoint, there is a hypothetical balance of relativity between the portal venous inflow to the hepatic graft and the size of the graft itself.³ The problem can be an undersized graft (and/or poor graft quality), portal venous hyperperfusion, or both.³ The relative portal venous hyperperfusion, irrespective of the cause, would induce a reduction in the hepatic arterial inflow mediated by the HABR

(washout of adenosine and its vasodilator effect). Again, this has been partly proposed by prior authors.^{11,13,33–36} Mogl and coworkers proposed that a known problem in split grafts that are undersized (small-for-size-syndrome), where there is relative portal venous hyperperfusion, may induce arterial hypoperfusion mediated by the HABR.¹¹ Mogl and coworkers speculated that this phenomenon may exist in whole grafts where the portal inflow is relatively higher than the whole graft size.¹¹

As can be concluded from the previous discussion and delineated by the illustration in **Fig. 3**, all these variables are relative to one another, and the clinical descriptions and associations in the literature are fragmented.³ No one study has delineated causality.³ However, this problem, with all its

relative variables, was discussed globally by Saad regarding arterial complications after liver transplantation.³ He speculated that splenic steal syndrome may have several causes and, if certain causes predominate, there may be several types.³ Furthermore, in his speculation, there may be predisposing factors such as splenomegaly relative to hepatic graft size with a dual hypoperfusion effect (due to the splenomegaly) on the hepatic artery. This work led to a true arterial steal and HABR-mediated response precipitated or accentuated by increased peripheral arterial resistance and/or GDA steal.³ Moreover, there may be degrees of severity of this or these hypoperfusion phenomena.³ Saad did agree, however, with Quintini and coworkers that “splenic artery steal syndrome” may be a misleading name because we are not sure whether it is actually a steal (siphon) phenomenon in all cases or in none at all.³ Saad proposed a new name based on what is known: posttransplant “non-occlusive hepatic artery hypoperfusion syndrome” (NOHAH syndrome), allowing all the potential and relative hypothetical causes to be included without a commitment to a particular concomitant finding, cause, predisposing/precipitating factors, and/or type.³

Splenic artery occlusion by embolization, banding, or surgical ligation has been proven to resolve the hypoperfusion syndrome.^{4,7,11,19,21} However, this is not proof that the hypoperfusion syndrome (NOHAH syndrome) is a true steal phenomenon.³ As can be seen in ►Figs. 2 and 3, the governing relationships create a full circle linked by the HABR. Breaking this circle (by occluding the splenic artery) reverses the hepatic artery hypoperfusion.³

Management

Management of nonocclusive hepatic artery hypoperfusion syndrome is to attempt to identify the problem(s) and treat them.³ Careful evaluation of the celiac angiogram to look for underlying anatomical defects (hepatic artery stenosis, thrombosis, and/or kinks) is the first step.^{1,3} Tomogram rotational angiography may aid in identifying anatomical defects, particularly in tortuous projection-overlapping arterial anatomy.³ Allograft rejection should be diagnosed by biopsy, immune-suppressant serum values, clinical suspicion, and should be managed medically.³ Large subcapsular hematomas should be evacuated or percutaneously decompressed. Abdominal compartment syndrome should be relieved by celiotomy. Unfortunately, other causes of increased hepatic arterial peripheral resistance are not easy to diagnose and manage.^{1,3} In cases of gastroduodenal (GDA) steal syndrome, a lateral aortogram evaluating for a significant superior mesenteric artery (SMA) stenosis is required, with resolution of the SMA stenosis with angioplasty/stent placement or arcuate ligament release with or without GDA embolization.¹

If there is preferential flow to the splenic artery, and if surgically feasible, an aortohepatic conduit can be created, taking the spleen out of the equation by ridding the common origin of the hepatic and splenic arteries.^{6,7,17,21} A more common alternative, if there is preferential flow to the splenic artery, are methods to restrict flow to the spleen^{1,2,4,6–13} (►Fig. 1). These include splenic artery banding, ligation,

hourglass stents, and embolization.^{1,2,4,6–13} Splenic artery ligation or banding are usually resorted to as prophylactic measures, although they can be utilized if the diagnosis is made posttransplant.^{12,13} Splenic artery embolization is the predominant method for reducing the preferential flow to the splenic artery and thus redirecting flow to the posttransplant hepatic artery as well as decreasing the hyperdynamic portal flow.^{1,2,4,6–13,16–20} The technical results are excellent (►Fig. 1), and the risk of splenic infarction is minimized by embolizing the splenic artery proximally (►Fig. 1B).^{1,2,4,6–13,16–20} There are currently no clearly described objective definitions to define clinical success.^{1,2,4,6–13,16–20}

If hepatic artery hypoperfusion is suspected along with hypersplenism (large spleen in the presence of pancytopenia, especially thrombocytopenia), graduated particulate distal splenic artery embolization can be performed to reduce the size of the spleen; this would also help with the NOHAH syndrome.³ This particulate embolization would utilize standard microcatheter techniques and embolic materials that may include polyvinyl alcohol, Gelfoam (Upjohn, Inc., Kalamazoo, MI), or other commercially available bland particulate embolic products. Usually two to three sessions are required to help sufficiently reduce the splenic volume with a decreased risk of splenic infarction and abscess formation. Approximately 30 to 40% of the spleen is devascularized each session.^{1,3} However, this technique may take time (months) to reach the desired effect from a splenic steal standpoint. It is effective, however, for hypersplenism (which is a matter of less urgency).³ Details of partial spleen embolization and outcomes are discussed elsewhere in this issue.

Conclusion

Hepatic artery hypoperfusion in the absence of significant hepatic artery stenosis, thrombosis, or kinks may have multiple well-described causes that are interrelated and may coexist. The complexity of splanchnic hemodynamics, the numerous variables associated with liver transplantation, and the lack of objective diagnostic imaging criteria make it difficult to identify the cause(s) and further fuel the controversy regarding the diagnosis of splenic steal syndrome. Saad has proposed that the global hepatic artery hypoperfusion be renamed NOHAH and not after a particular cause or partial cause that is defined in splenic steal syndrome.³ Regardless of the causes, splenic embolization breaks the splenic-to-portal circulation and has been shown to improve hepatic arterial flow.

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