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SPLENIC ARTERY EMBOLIZATION IN THE MANAGEMENT OF COMPLICATIONS AFTER ORTHOTOPIC LIVER TRANSPLANTATION

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ABSTRACT

Aim: The aim of this study is to present the role of splenic artery embolization (SAE) in the management of selected complications following orthotopic liver transplantation (OLTx), with particular emphasis on its clinical application.

Methods: A narrative literature review was conducted, including studies on post-OLTx complications and the use of interventional radiology techniques. Publications addressing the mechanisms underlying these complications and their management using SAE were analyzed.

Results: SAE is a minimally invasive endovascular procedure involving occlusion of blood flow within the splenic artery using embolic materials. It is applied in the treatment of selected complications after OLTx, including splenic artery steal syndrome, refractory ascites, and hypersplenism. By reducing blood flow within the splenic vascular bed, SAE improves arterial graft perfusion, decreases portal inflow, and modulates hemodynamic disturbances. Available data indicate high efficacy of this method in selected clinical settings.

Conclusion: SAE is an effective and minimally invasive method for the management of selected complications following OLTx. Its use in clinical practice is expected to increase with the growing role of minimally invasive techniques in post-transplant care.

KEYWORDS

Liver Transplantation, Splenic Artery Embolization, Splenic Artery Steal Syndrome, Interventional Radiology, Splanchnic Circulation

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1. Introduction

Orthotopic liver transplantation (OLTx) is currently the standard treatment for end-stage liver disease, as well as for selected oncological and metabolic disorders. In patients with advanced liver conditions - such as cirrhosis, certain hepatic malignancies, or congenital and acquired anatomical abnormalities and metabolic disorders - OLTx often represents the only curative therapeutic option (De Carlis et al., 1993).

Despite significant advances in surgical techniques, immunosuppressive strategies, and perioperative care, post-transplant complications remain a major clinical challenge, affecting both early and long-term outcomes. Of particular importance are hemodynamic disturbances within the splanchnic and portal circulation, which may lead to impaired graft perfusion and the development of serious complications.

In recent years, minimally invasive endovascular techniques have gained increasing importance in the management of post-transplant complications. Splenic artery embolization (SAE) has emerged as one of the key interventional approaches, enabling targeted modulation of portal-splanchnic hemodynamics and improving arterial graft perfusion.

The aim of this study is to present the physiology of splanchnic circulation in the context of post-OLTx changes, to discuss the hemodynamic mechanisms underlying post-transplant complications, and to highlight the role of SAE as an effective and safe therapeutic modality in their management.

2. Physiology of Splanchnic Circulation and Hemodynamic Changes After Orthotopic Liver Transplantation

The hepatic portal system constitutes the main component of the splanchnic circulation, with its primary function being the drainage of venous blood from the abdominal organs to the liver via the portal vein. It is formed by the confluence of the splenic vein and the superior mesenteric vein and represents the central vessel of the portal system, transporting nutrient-rich blood from the spleen, stomach, intestines, and pancreas directly to the liver (Harkins & Ahmad, 2023). The second source of hepatic blood supply is the proper hepatic artery, which arises from the common hepatic artery, a branch of the celiac trunk. It delivers well-oxygenated arterial

blood to the liver, which mixes with portal venous blood within the hepatic sinusoids. Under physiological conditions, the proportions of blood flow through these two inflows reflect the unique hepatic hemodynamics: approximately 75% of total hepatic blood flow is derived from the portal vein, while about 25% is supplied by the hepatic artery, highlighting the dual blood supply of the liver (Harkins & Ahmad, 2023).

Following orthotopic liver transplantation, significant alterations in circulatory physiology occur. A sudden reduction in vascular resistance within the graft leads to dynamic changes in blood flow within the portal-splanchnic system (Feng et al., 2014). Simultaneously, long-standing adaptive mechanisms developed in the course of chronic liver disease and portal hypertension - such as extensive collateral circulation and altered relationships between hepatic arterial and portal venous flow - are reversed. This abrupt shift may result in transient or persistent hemodynamic imbalance within the graft. The relationship between portal vein flow and hepatic artery flow is referred to as the *hepatic artery buffer response (HABR)*. This mechanism is based on the interplay between portal blood flow and adenosine concentration within the hepatic arterial resistance vessels. With relatively constant adenosine production, its local concentration depends on the rate of washout, which is proportional to portal flow. A decrease in portal flow reduces adenosine clearance from the perivascular space, leading to increased local concentration and consequent vasodilation of the hepatic artery. Conversely, increased portal flow enhances adenosine washout, resulting in hepatic arterial vasoconstriction (Lautt, 1985). The hepatic arterial system, characterized by high pressure and significant vascular resistance, is subject to physiological autoregulation mediated by the *HABR*. This mechanism ensures relative stability of total hepatic blood flow and adequate oxygen delivery - decreased portal flow is compensated by increased arterial inflow, whereas increased portal flow leads to a proportional reduction in hepatic arterial perfusion (Jakab et al., 1995).

In the post-transplant setting, however, the *HABR* may exert detrimental effects on the graft. Presser et al. (2015) demonstrated that significant portal hyperperfusion following OLTx, associated with increased adenosine washout, may result in marked hepatic arterial vasoconstriction and subsequent arterial hypoperfusion. Moreover, independently of excessive hepatic arterial vasoconstriction due to overactivation of the *HABR*, increased portal flow may directly damage the graft through elevated portal pressure acting on hepatic sinusoids (Payen et al., 1990). Dysfunction of the *HABR* may lead not only to ischemic injury of the graft but also to the development of complications such as splenic artery steal syndrome, refractory ascites, and hypersplenism. Consequently, impairment of this mechanism represents a key factor in post-transplant hemodynamic disturbances, significantly influencing both early and late clinical outcomes after OLTx.

3. Splenic Artery Embolization: Anatomical Considerations and Technical Aspects

Under typical anatomical conditions, the celiac trunk arises from the abdominal aorta and divides into three main branches: the left gastric artery, the splenic artery, and the common hepatic artery (Juszczak et al., 2021). The splenic artery is the largest branch of the celiac trunk and typically divides into two main branches - superior and inferior - which subsequently undergo further subdivision within the splenic parenchyma. Its primary function is to supply arterial blood to the spleen; however, it also gives rise to numerous branches supplying other abdominal organs, including the pancreas and the stomach. Knowledge of these branches is of significant clinical importance in the planning and performance of splenic artery embolization (Lee et al., 2017). The second major branch of the celiac trunk is the common hepatic artery, which typically divides into the gastroduodenal artery and the proper hepatic artery (Hiatt et al., 1994). From a clinical perspective, anatomical variations of the celiac trunk are also important, including the hepatosplenic trunk and other branching patterns that may influence vascular anatomy during surgical and endovascular procedures (Pandey et al., 2004).

Splenic artery embolization (SAE) is an endovascular procedure involving the occlusion of blood flow within the splenic artery in order to modify portal-splanchnic hemodynamics. The procedure is performed via catheterization of the celiac trunk followed by selective catheterization of the splenic artery, where embolic materials such as coils or vascular plugs are deployed. Blood flow is assessed using digital subtraction angiography (DSA) before and after embolization. The mechanism of action of SAE involves two principal components. First, reduction of blood flow within the splenic vascular bed results in a relative redistribution of flow within the celiac axis toward the hepatic artery, thereby increasing arterial inflow to the graft. Second, decreased inflow into the portal system leads to reduced portal venous flow, which - through modulation of

HABR - results in increased adenosine concentration and secondary vasodilation of the hepatic artery. Consequently, this leads to enhanced arterial perfusion and improved graft blood supply.

Two main SAE techniques are distinguished: proximal and distal embolization. Proximal embolization, performed in the proximal segment of the splenic artery, preserves splenic perfusion via collateral circulation and is technically less demanding. In contrast, distal embolization involves more peripheral branches, closer to the splenic hilum. However, it has been demonstrated that the location of embolization (proximal vs. distal) does not significantly affect long-term outcomes (Fleckenstein et al., 2022). SAE is characterized by high efficacy in rapidly reversing graft hypoperfusion and stabilizing its hemodynamics. Reduction of flow within the splenic artery leads to improved hepatic arterial flow, which is critical for proper graft function following liver transplantation (Usai et al., 2022).

4. Role of Splenic Artery Embolization in the Management of Selected Complications After Orthotopic Liver Transplantation

4.1. Splenic Artery Steal Syndrome

Arterial steal syndromes are defined as a reduction in perfusion of one arterial branch due to redistribution of blood flow toward another branch arising from the same vascular trunk. Splenic artery steal syndrome (SASS), first described in 1991 by Manner et al., occurs in approximately 6–7% of orthotopic liver transplant recipients (Manner et al., 1991; Quintini et al., 2008; Vogl et al., 2001). In SASS, blood flow is redistributed from the hepatic artery to the splenic artery, resulting in inadequate perfusion of the transplanted liver. Graft hypoperfusion may lead to early graft dysfunction and, in severe cases, graft loss (Fleckenstein et al., 2022). The clinical presentation includes progressive elevation of aminotransferase levels and abnormalities in cholestatic parameters, while reduced hepatic arterial flow may contribute to early graft dysfunction (De Carlis et al., 1993). Doppler ultrasound typically demonstrates an increased resistive index (RI) in the hepatic artery and reduced diastolic flow. In cases of more severe ischemia, biliary complications may develop, including biliary strictures and ischemic cholangiopathy (Usai et al., 2022).

The etiology of SASS is multifactorial and includes disturbances in *HABR*, pre-existing splenomegaly with increased splenic flow, low vascular resistance within the splenic arterial bed, as well as factors related to the transplantation procedure and graft preservation. SASS is most often a diagnosis of exclusion and should be considered only after ruling out other causes of graft dysfunction, such as acute cellular rejection, infection, or drug toxicity (Pinto et al., 2014). An important factor influencing the risk of SASS is the proper reconstruction of hepatic arterial inflow. Vascular complications following transplantation - including stenosis, thrombosis, and aneurysms - may increase the risk of SASS. These abnormalities may occur in the recipient artery, at the site of vascular anastomosis, or within the graft artery, and may involve both extrahepatic and intrahepatic vessels (Settmacher et al., 2000). SASS may develop at any time after transplantation; however, it is most commonly diagnosed within the first two months postoperatively. Early-onset cases are more frequently observed in patients with pre-existing hyperdynamic circulation and splenomegaly, which are associated with reduced resistance in the splenic vascular bed and increased splenic arterial flow even prior to transplantation. In contrast, late-onset SASS may result from secondary increases in splenic flow, for example in the setting of graft rejection or viral hepatitis (De Carlis et al., 1993; Uflacker et al., 2002).

Numerous studies have demonstrated the high efficacy of splenic artery embolization (SAE) in the treatment of SASS, with a low associated complication rate (Kim et al., 2011). In a systematic review including 219 patients with SASS after OLTx, SAE was performed in 94.7% of cases, resulting in improvement of Doppler hemodynamic parameters and liver function tests in 96.3% of patients, confirming its high effectiveness in correcting graft hypoperfusion (Li et al., 2017). The therapeutic effect of SAE in SASS is based on reduction of blood flow within the splenic vascular bed, which decreases flow redistribution away from the hepatic artery and increases effective arterial inflow to the graft. Additionally, reduced inflow to the portal system decreases portal hyperperfusion and, through modulation of *HABR*, promotes hepatic arterial vasodilation. As a result, graft perfusion improves, thereby reducing the risk of ischemic complications, including biliary injury, ischemic cholangiopathy, and early graft dysfunction.

It has been suggested that SASS may be more common than previously recognized, and that early diagnosis and prompt treatment with SAE are crucial for preventing graft failure. Identification of high-risk patients may further contribute to reducing the incidence of this complication (Jiang et al., 2022).

4.2 Refractory Ascites

In most recipients, ascites resolves spontaneously within approximately six months after OLTx, and management is typically limited to diuretic therapy and periodic paracentesis. Refractory ascites - defined as the persistence or rapid recurrence of fluid in the peritoneal cavity despite optimal medical therapy, and the need for frequent paracenteses due to treatment inefficacy or intolerance - represents a rare but clinically significant complication of OLTx, associated with increased morbidity and mortality (Cirera et al., 2000). The pathogenesis of refractory ascites after OLTx is multifactorial and includes persistent hemodynamic disturbances, such as increased portal flow and elevated portal pressure, leading to enhanced fluid transudation into the peritoneal cavity. Activation of neurohormonal systems, including the renin–angiotensin–aldosterone system, also plays a significant role by promoting sodium and water retention.

Therapeutic management should be directed toward identifying and treating the underlying cause. In cases of unclear etiology, transjugular intrahepatic portosystemic shunt (TIPS) placement and splenic artery embolization (SAE) may be considered as strategies to modulate portal hemodynamics. SAE is currently regarded as a promising therapeutic option in the treatment of post-OLTx ascites. The best clinical response is observed in patients with significant splenomegaly, particularly when the spleen-to-graft volume ratio exceeds 0.5. Reduction of flow within the splenic circulation leads to decreased portal pressure and alleviation of portal hypertension, resulting in reduced portal venous flow, decreased need for diuretic therapy, weight loss, and gradual resolution of ascites (Cirera et al., 2000; Luca et al., 2006). In recent years, SAE has increasingly been considered as an alternative to TIPS. Available data suggest that this method may be effective both in the treatment and prevention of postoperative ascites, with hemodynamic improvement often leading to complete resolution of symptoms. Due to its high efficacy and favorable safety profile, SAE represents a valuable therapeutic option, particularly in cases requiring rapid reduction of portal inflow (Quintini et al., 2011; Chang et al., 2007).

4.3 Hypersplenism

Hypersplenism and splenomegaly may persist or recur after orthotopic liver transplantation despite elimination of the primary cause of cirrhosis and portal hypertension. Persistent disturbances in portal hemodynamics and alterations in splenic function may contribute to the development of hypersplenism, manifested by thrombocytopenia, leukopenia, and anemia.

In a subset of patients after OLTx, splenomegaly and altered portal circulation persist, which may result from incomplete resolution of portal hypertension, persistence of portosystemic collaterals, and chronically increased portal flow. Consequently, enhanced sequestration of blood cellular components within the spleen occurs, leading to persistent cytopenias despite adequate graft function (Chezmar et al., 1992).

Therapeutic management is usually based on pharmacological treatment; however, in cases of severe, persistent thrombocytopenia of unclear etiology, splenectomy may be considered (Altaca et al., 1997). This procedure, however, is associated with a significant risk of complications, including intraoperative bleeding and increased susceptibility to infections.

An alternative to surgical treatment is splenic artery embolization (SAE), which is a less invasive procedure typically performed under local anesthesia and associated with a lower complication rate. Preservation of the spleen allows maintenance of its immunological function, while the risk of severe infections, including sepsis, remains relatively low. The clinical efficacy of SAE is reflected in a significant increase in platelet and leukocyte counts, with a low mortality rate estimated at below 6% (Rysmakhanov et al., 2015). Available data indicate that SAE is a safe and effective method for the treatment of hypersplenism both in the peri-procedural period and during long-term follow-up after OLTx (Obri et al., 2023).

5. Conclusions

Splenic artery embolization is an effective and safe method for the treatment of selected hemodynamic complications following OLTx. Early identification of hemodynamic disturbances and appropriate patient selection for interventional treatment are crucial for improving post-transplant outcomes (Wehrle et al., 2025). Further studies are necessary to clearly define optimal indications, timing of intervention, and long-term outcomes of splenic artery embolization in this patient population.

Conflicts of Interest: No conflicts of interest to declare.

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