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Review Article

# Non-variceal Extrahepatic Portosystemic Shunts: A Review of Pathogenesis, Diagnosis, and Treatment

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## Abstract

Extrahepatic portosystemic shunts (EPS) are abnormal connections between the portal and systemic circulations. Acquired EPS occur most commonly in adults and are usually associated with portal hypertension due to cirrhosis. Acquired EPS cases can be further subdivided into two types: variceal (pre-existing) EPS and non-variceal EPS (NVEPS). Variceal EPS arise from originally small vessels with pre-existing dual portal and systemic drainage. Due to elevated portal pressure, these vessels dilate and undergo a reversal of flow, sending blood back to the systemic circulation. A much less common and, therefore, underappreciated subset of acquired EPS is NVEPS, which consists of aberrant connections that did not previously exist between the portal vein and large systemic vessels, usually in the presence of portal hypertension. Neoangiogenesis results in the development of abnormal anastomoses between the portal vein and other large veins, resulting in splenorenal, gastrorenal, portocaval, and mesocaval shunts. While not uncommon, they are frequently overlooked in the diagnosis and treatment of portal hypertension and can pose significant diagnostic and therapeutic challenges. Because the treatment of variceal EPS and NVEPS can differ markedly, it is important to correctly diagnose NVEPS and institute appropriate management. The aim of this article was to review acquired EPS, with particular attention to NVEPS, updating the pathogenesis, diagnosis, and treatment.

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## Introduction

Extrahepatic portosystemic shunts (EPS) are defined as communications between the portal and systemic venous circulations.[1] EPS cases are frequently categorized as congenital or acquired, depending on the timing of appearance and features at diagnosis. Congenital EPS cases are spontaneous (not formed due to hepatocellular disease), rare, and often present in childhood with various hepatic complications in the absence of portal hypertension. These shunts are quite unique, a product of embryologic vascular abnormality, and therefore beyond the scope of this review. Acquired EPS occur most commonly in adults and are usually associated with portal hypertension due to cirrhosis. Acquired EPS cases can be further subdivided into two types: variceal (pre-existing) extrahepatic portosystemic shunts and non-variceal extrahepatic portosystemic shunts (NVEPS). Variceal EPS are common and include esophageal/gastric varices, hemorrhoids, and portal cutaneous shunts, caused by dilation of originally small pre-existing vessels with dual portal and systemic drainage. Due to portal hypertension, there is also a reversal of the direction of flow from the portal to the systemic circulation. Symptoms of hepatic decompensation are frequently present, especially gastrointestinal (GI) bleeding. Because they are common and well-understood, variceal EPS will not be covered in this review. On the other hand, NVEPS are an interesting subset of acquired EPS, consisting of aberrant anastomoses not previously present, but which develop due to neoangiogenesis between the portal vein and other large veins, creating splenorenal, gastrorenal, portocaval, and mesocaval shunts. These shunts are not uncommon, but they are not frequently considered in the diagnosis and treatment of portal hypertension, and they can pose significant diagnostic and therapeutic challenges. While these diversionary pathways were once considered to be beneficial compensatory mechanisms to decompress diseased or damaged portal systems and provide alternate pathways of blood flow to return to the heart, it is now well established that NVEPS are often inefficient in reducing portal pressure, and also deprive the liver of adequate blood flow, which contributes to the deterioration of liver function and can worsen outcomes.[1] The presence of NVEPS is associated with higher mortality and complications, including the development of portosystemic shunt syndrome, characterized by a progressive deterioration of hepatic function, hepatic encephalopathy (HE), GI bleeding, ascites, and sometimes portal vein thrombosis (PVT).

Because the recommended treatment for cases of variceal EPS and NVEPS often differs substantially, it is important to distinguish these entities so that each is diagnosed correctly and appropriate management can be employed. The aim of this review was to update information on acquired EPS, with particular attention to the pathogenesis, diagnosis, and treatment of NVEPS.

**Keywords:** Extrahepatic portosystemic shunts; Acquired extrahepatic shunts; Variceal extrahepatic portosystemic shunts; Non-variceal extrahepatic portosystemic shunts; Cirrhosis; Portal hypertension.

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**Table 1. Prevalence and types of NVEPS as identified by Doppler ultrasound, CT, and MRI**

| Imaging modality | Total sample size | Prevalence of NVEPS (%) | Type of NVEPS (in order of prevalence) | Reference |
|---|---|---|---|---|
| Doppler US | 109 | 38% | Splenorenal; Paraumbilical; Gastric | Von Herbay *et al*.[2] |
| Doppler US | 326 | 39.9% | Splenorenal; Left gastric vein; Paraumbilical | Zardi *et al*.[3] |
| Doppler US | 86 | 42% | Paraumbilical; Left gastric vein; Splenorenal; Short gastric vein | Berzigotti *et al*.[5] |
| Doppler US | 982 | 34% | Paraumbilical; Splenorenal; Mesenteric | Lipinski *et al*.[4] |
| CT (1,630); MRI (99) | 1,729 | 60% | Splenorenal; Paraumbilical; Gastrorenal; Mesocaval; IMV cava | Simon-Talero *et al*.[6] |
| CT/MRI | 326 | 80.7% | N/A | Rodriguez *et al*.[7] |
| CT | 741 | N/A | Splenorenal | Saks *et al*.[8] |
| CT | 451 | N/A | Splenorenal; Gastrorenal; Paraumbilical; Mesocaval | Achiwa *et al*.[9] |

US, ultrasound; CT, computed tomography; MRI, magnetic resonance imaging; N/A, not applicable; NVEPS, non-variceal extrahepatic portosystemic shunts.

## Epidemiology

Many studies have identified several types of NVEPS, with splenorenal, paraumbilical, gastrorenal, and mesocaval shunts reported to be the most prevalent (see Table 1).[2–9] Doppler ultrasound, used to identify NVEPS, has estimated the global incidence to range from 34–42% of individuals with liver cirrhosis.[2–5,10] Studies using contrast-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) have reported a general prevalence estimate between 60–80% in cirrhotic patients.[6,7,11] A recent study, conducted by the Baveno VI Cooperation Group, evaluated 1,729 cirrhotic patients, of whom 60% had NVEPS.[6] Rodriguez *et al*. provided data collected from 2013–2017 from a cohort of 326 cirrhotic patients who were candidates for liver transplant, in which a high rate of NVEPS was found—nearly 80%.[7] Spontaneous mesocaval and portocaval shunts are quite rare, with the former noted only in a few case reports and the latter representing as few as 4% of all shunts found on imaging of cirrhotic individuals.[6]

## Classification

NVEPS can be classified as large or small according to their maximum diameter, with a cut-off of >8mm or <8mm, respectively, determined from the smallest symptomatic shunt embolized.[1] They can also be classified as left- and right-sided shunts, relative to the splenoportomesenteric confluence.[1] Further, and more recently, portosystemic shunts are classified according to their drainage into the superior or inferior vena cava, based on the outflow pathway of collateral vessels.[12] NVEPS generally drain into the inferior vena cava, a distinguishing feature when compared to variceal EPS, such as esophageal, paraesophageal, and gastric varices, which drain into the superior vena cava.[12] A schematic diagram of the most prevalent NVEPS discussed in this review is displayed in Figure 1. Philips *et al*. published maximum-intensity-projection contrast-enhanced CT images of most of these shunts (Fig. 2) in their detailed review of the historical, anatomical, and hemodynamic aspects of portosystemic collaterals in cirrhosis.

## Pathogenesis

The pathogenesis of NVEPS has traditionally been considered a mechanical consequence of increased portal pressure, resulting in the passive opening of pre-existing embryonic channels connecting the portal and systemic venous systems. Recent studies, however, have established the role of active angiogenesis in the development of these aberrant vessels.[13] The process of neoangiogenesis has been studied in murine models.[13,14] Fernandez *et al*. demonstrated that the formation of collateral vasculature was mediated by vascular endothelial growth factor in cirrhotic mice.[15] Overexpression of vascular endothelial growth factor, stimulated by factors such as hypoxia, oxidative stress, inflammation, and shear stress, leads to nitric oxide production by endothelial nitric oxide synthase, increasing vascular permeability and promoting angiogenesis.[15] It has also been shown that platelet-derived growth factor and placental growth factor play a role in stabilizing the vascular architecture and enhancing growth by stimulating endothelial and smooth muscle cell proliferation.[15]

NVEPS initially reduce portal pressure, offering protective effects. However, over time, NVEPS have detrimental effects on liver function. The formation of collateral vessels ultimately contributes to a decrease in hepatocyte perfusion, tissue hypoxia, and the promotion of further neoangiogenesis in the splanchnic circulation. This leads to a positive feedback loop with the progressive amplification of the mechanisms promoting a hyperdynamic splanchnic circulation state.[13,14] Kumamoto *et al*. proposed the term "portosystemic shunt syndrome," referring to significant liver deterioration over five years (with worsening Child-Pugh scores) in patients with cirrhosis and portal hypertension who have splenorenal shunts, compared with patients without these shunts.[16] Saad *et al*. elaborated on this concept and described a complete syndrome, highlighting three stages of clinical manifestations and imaging findings: 1) early stage, with few episodes of HE and relatively preserved liver function; 2) late stage, with more frequent HE and deteriorating liver function, atrophy, disappearance of portal branches, possible PVT, and sluggish hepatopetal flow on imaging; and 3) terminal stage, with disabling HE, advanced liver failure, PVT, liver atrophy, and hepatofugal flow.[17]

Several studies have examined the relationship between portal hypertension complications and NVEPS, with variable conclusions. Earlier studies suggested a protective effect of large NVEPS on the development of esophageal varices and ascites, especially in patients with HE. However, more recent studies have suggested that large NVEPS and HE are predictive of clinically significant portal hypertension and the

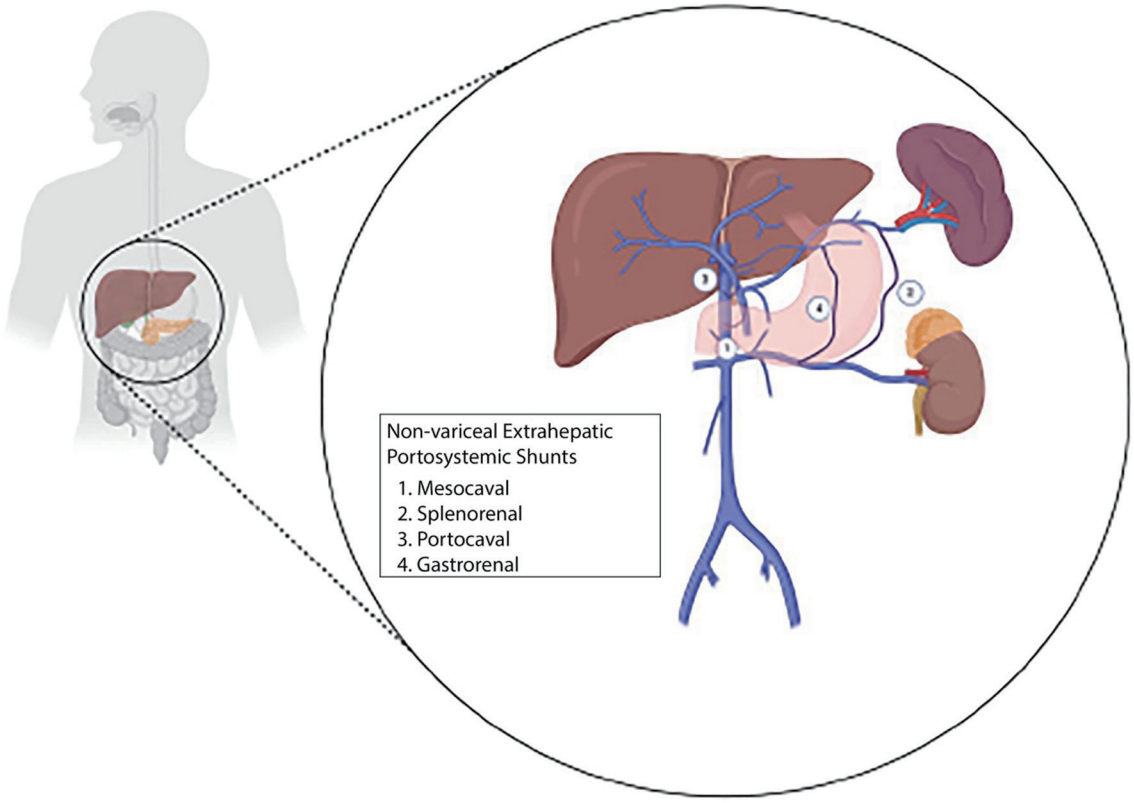


**Fig. 1. Schematic diagram of prevalent NVEPS.** NVEPS, non-variceal extrahepatic portosystemic shunts.

development of ascites and varices. These contradictory findings can be explained by the dynamic nature of liver disease, highlighting the complex progression of cirrhosis and the compensatory mechanisms at play. Most of these studies analyzed only a snapshot in time, and earlier studies, especially, were retrospective and had small sample sizes.

## Diagnosis

The diagnosis of NVEPS is made using noninvasive imaging modalities such as Doppler ultrasound and CT or MRI.[1] Ultrasonography is the most convenient method for evaluating NVEPS, as it is inexpensive and allows for the detection of most shunts. In addition, ultrasonography enables the evaluation of liver morphology, spleen size, and changes in portal vein caliber and flow in patients with cirrhosis.[18] Limitations depend on operator experience, as well as patient factors such as obesity or intestinal gas.[18] Small NVEPS and those deeply localized may not be identifiable by ultrasonography.

CT and MRI are more sensitive and specific for the detection and assessment of NVEPS.[18] Both imaging techniques can evaluate the entire abdomen, providing a comprehensive picture of the splenoportal system, and are less susceptible to variations in body volume or the presence of gas in the digestive tract compared to ultrasonography.[18] CT imaging has notable advantages over MRI due to its availability, rapid acquisition, high spatial resolution, and capability for multiplanar reconstruction.[18] MRI is equally accurate in assessing the anatomic course of collaterals, especially when using thin-section acquisitions and gadolinium-based contrast. Limitations of MRI include availability, cost, and longer acquisition times.[18]

## Presentation

The presentation of NVEPS is variable and generally depends on location relative to the splenoportomesenteric confluence. Splenorenal shunts, consisting of communicating venous pathways between the splenic vein and left renal vein, do not involve the splanchnic circulation and, therefore, do not contribute to the formation of varices or the risk of spontaneous bleeding. However, HE and PVT can be presenting conditions.[18,19] Gastrorenal shunts are often found in patients with cardiofundic gastric varices and carry a significant risk for variceal bleeding as their presentation.[18,19] The less commonly encountered NVEPS, such as mesocaval, portocaval, and mesorenal shunts, most commonly present with HE.

## Complications

### *HE*

The relationship between NVEPS and HE is well-known. Lam *et al*. examined the effect of large NVEPS on the incidence of variceal hemorrhage and HE. Twenty cases of chronic liver disease with large NVEPS were compared with a group of patients with liver disease and Cruveilhier-Baumgarten murmurs, a venous hum produced by recanalization of umbilical and paraumbilical veins in patients with cirrhosis and portal hypertension, and with a control group having liver disease but without large shunts. It was demonstrated that HE occurred more frequently in those with large shunts and Cruveilhier-Baumgarten murmurs.[20] Riggio *et al*. described a case-control study of NVEPS in 28 patients with cirrhosis, 14 of whom had recurrent (two or more HE episodes within six months) or persistent (changes in behavior that are always

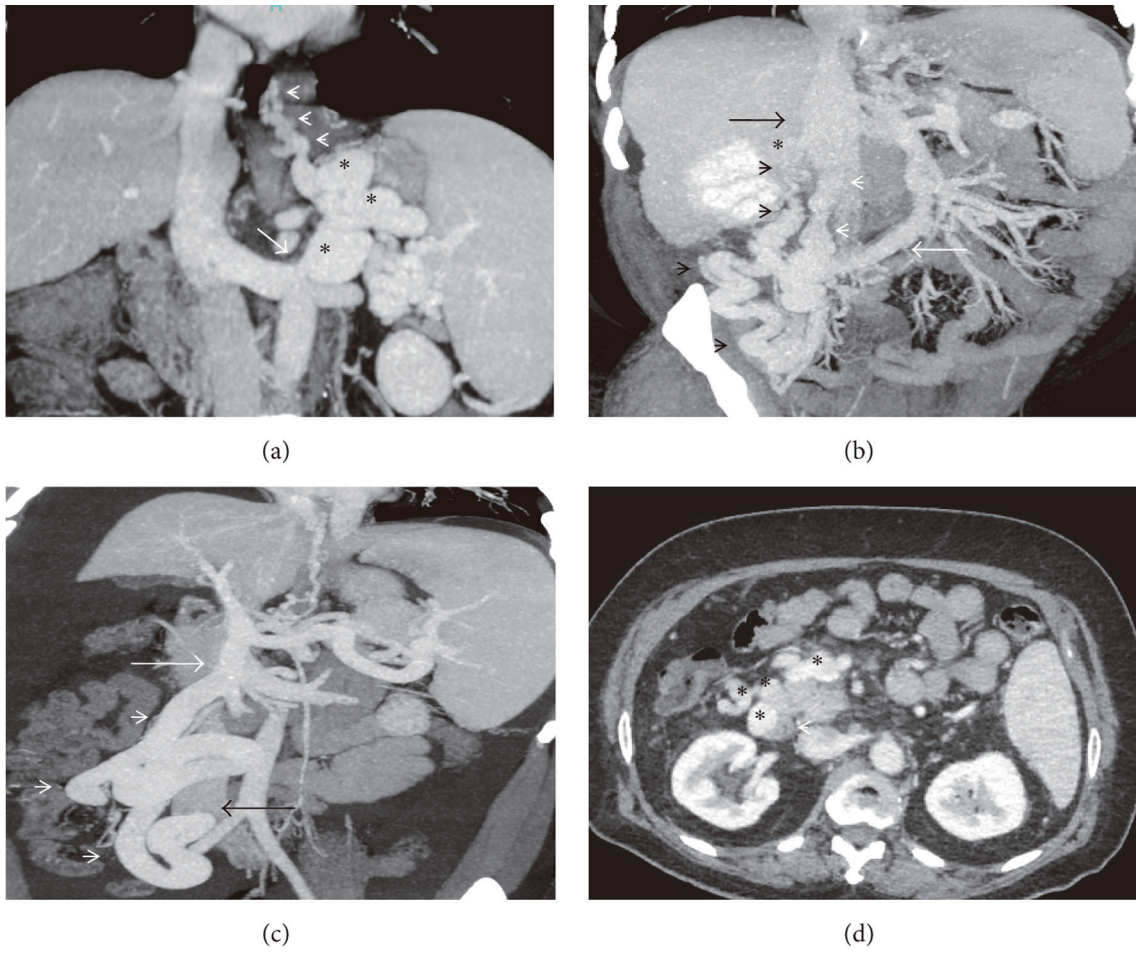


**Fig. 2. Maximum-intensity-projection, contrast-enhanced CT imaging of various NVEPS.** (a) Coronal-oblique image showing a tortuous gastro-lieno-renal shunt (asterisks) draining multiple gastroesophageal collaterals (arrowheads) into the superior aspect of the left renal vein (arrow); (b) Coronal image demonstrating a mesorenal and a mesocaval shunt in the same patient (black and white arrowheads, respectively). Asterisk: right renal vein; black arrow: IVC; white arrow: superior mesenteric vein (double portosystemic shunts); (c) Coronal-oblique image demonstrating a dilated and tortuous mesocaval shunt (arrowheads) communicating between the superior mesenteric vein (white arrow) and the inferior vena cava (black arrow); (d) Axial image showing multiple duodenal and paraduodenal collaterals (asterisks). Arrowhead denotes the duodenum. [Philips CA, et al. A Comprehensive Review of Portosystemic Collaterals in Cirrhosis: Historical Aspects, Anatomy, and Classifications. Int J Hepatol. 2016;2016:6170243. doi: 10.1155/2016/6170243. Creative Commons license to copy. No alterations were made to original photos]. CT, computed tomography; NVEPS, non-variceal extrahepatic portosystemic shunts.

present) HE, and 14 of whom did not have any past or present signs of HE, matched for age and degree of liver failure. The authors demonstrated that those with large shunts often developed chronic HE and were also associated with bouts of overt HE (recurrent HE). Large shunts were identified in 10 patients with HE and in only two patients without HE (71% vs. 14%; $p < 0.002$).[21] Simón-Talero *et al*. studied patients with large versus small NVEPS and found that episodic HE occurred more frequently in patients with large NVEPS (48% vs. 34%, $p < 0.001$) and that they had a higher prevalence of persistent and chronic HE (52% vs. 44%, $p < 0.007$).[6] Additionally, when stratifying patients based on the model for end-stage liver disease (MELD) score, the presence and size of NVEPS were found to be significantly associated with HE, regardless of liver function, suggesting that the presence of NVEPS is an independent risk factor for the development of HE. More recently, Praktiknjo *et al.* revealed that patients with large NVEPS had a higher risk of developing HE and notably higher ammonia levels.[22] Moreover, the presence of hepatofugal blood flow in NVEPS has also been identified as an important contributing factor for the development of HE.[23] In fact, it is likely that the extent of shunting, in combination with the degree of normal liver function, together dictates whether HE occurs. Splenorenal shunts and mesorenal shunts have been found to be most frequently associated with HE.

### *Esophago-gastric varices*

The relationship between NVEPS and esophago-gastric varices, and subsequently the risk of bleeding, seems at odds in the literature. Riggio *et al*. reported that patients with chronic HE and large NVEPS had fewer esophageal varices and less portal hypertensive gastropathy than those without NVEPS, supporting a decompressive effect on gastrointestinal bleeding.[21] Qi *et al*. found that cirrhotic patients with NVEPS had a lower prevalence of acute upper GI bleeding than those without NVEPS, but the difference was not statistically significant.[24] However, there have been reports that the presence of NVEPS was not associated with a lower risk of bleeding. Berzigotti *et al*. found that NVEPS did not prevent enlargement of varices and may have promoted the formation and

progression of varices.[5] Additionally, a retrospective study performed by Aseni *et al*. demonstrated that there was no correlation between the risk of esophageal hemorrhage and the type of NVEPS.[25] Therefore, despite the presence of large NVEPS, which should theoretically decompress the vascular system and prevent the formation of varices, the risk of bleeding remains. Some reports have made a correlation between specific types of NVEPS and bleeding risk. For varices, the location of drainage has been associated with the type of varix. Gastro-esophageal/esophageal varices are commonly supplied by the coronary/left gastric vein and are linked to gastrocaval shunts, whereas gastric varices alone are frequently supplied by the short or posterior gastric veins and are closely linked to gastrorenal shunts.[26]

### *Ascites*

The association between NVEPS and ascites remains unclear. Renzulli *et al*. described a retrospective cohort study of 184 advanced chronic liver disease patients studied for the incidence of hepatic decompensation, PVT, hepatocellular carcinoma, transjugular intrahepatic portosystemic shunt (TIPS) placement, and liver transplantation. It was found that the presence of ascites was an independent predictor of decompensation-free survival and the most common decompensation event in cirrhotic patients with NVEPS.[27] In contrast, Saks *et al*. retrospectively analyzed 741 patients with decompensated cirrhosis undergoing liver transplantation evaluation over a 15-year period to evaluate the influence of NVEPS, specifically splenorenal shunts, as an independent prognostic marker for mortality and liver transplantation outcomes. It was discovered that patients with splenorenal shunts were significantly less likely to have ascites (43% vs. 59%).[8] In particular, the presence of large NVEPS tended to correlate with a decrease in the rate of ascites.[21,23] Nevertheless, with contrasting evidence and scarce data elsewhere in the literature, the relationship between NVEPS and ascites remains uncertain.

### *PVT*

The presence of PVT may be either a cause or an effect of NVEPS, and therefore, the association between NVEPS and PVT is also unclear. Cirrhosis as a disease entity poses both pro- and anti-thrombotic risks simultaneously, with PVT being a well-known complication, especially in individuals with advanced disease. The development of PVT is multifactorial, involving portal flow stasis due to deranged liver architecture, complex acquired hypercoagulable disorders (factor deficiencies, platelet hyperactivity), and exogenous factors leading to endothelial dysfunction (endoscopic therapy of bleeding esophageal varices, abdominal surgery, inflammation, trauma).[28] Therefore, in the absence of recanalization, the presence of PVT leads to the development of splenorenal and paraumbilical shunts specifically to maintain portal venous flow. Nardelli *et al*. described a retrospective study of 222 cirrhotic patients, using CT imaging to evaluate the presence, types, and sizes of NVEPS.[29] Both multivariable logistic analysis and competitive risk analysis were employed to identify the clinical characteristics associated with the presence and size of NVEPS and complications/mortality, respectively.[29] It was shown that PVT was independently and significantly associated with the presence of NVEPS (any size) and was statistically associated specifically with NVEPS larger than 1 cm.[29]

Conversely, the presence of NVEPS may precipitate PVT due to a "steal" syndrome, causing a reduction in portal flow and venous stasis through collateral vasculature. Maruyama *et al*. studied 150 patients with cirrhosis but without PVT at baseline to assess the predictive factors and long-term outcomes of *de novo* development of PVT.[30] The presence of collateral vessels with a flow volume of more than 400 mL/m and a flow velocity of more than 10 cm/s proved to be a significant risk factor for the occurrence of PVT.[30]

## Management

While the complications of NVEPS remain controversial in the literature, early identification of NVEPS and prompt intervention have been shown to lead to fewer complications of portal hypertension overall and improved survival. In fact, Yi *et al*. studied the impact of one of the most common shunts, splenorenal shunts, on the long-term survival of liver cirrhosis.[31] They enrolled 122 cirrhotic patients over a five-year period, 37 of whom (~30%) were found to have splenorenal shunts on CT or MRI. Retrospective measurements were made of the maximum diameters of such shunts, the liver-to-abdominal area ratio, Child-Pugh and MELD scores to determine the effect on liver function and ultimate prognosis in liver cirrhosis.[31] Patients with splenorenal shunts, with an average median diameter of 13.5 mm, had significantly lower diameters of the right portal vein (9 mm vs. 11.2 mm, $p = 0.001$) and main portal vein (15.3 mm vs. 16.8 mm, $p = 0.017$) than those without these shunts.[31] This leads to the "steal" phenomenon, whereby portal venous blood bypasses the liver, impairs hepatic nutrition, and ultimately leads to worsening liver function and progression of cirrhosis.[31] Patients with splenorenal shunts also had significantly lower liver-to-abdominal area ratio scores (25.39 vs. 31.58, $p < 0.001$) and higher Child-Pugh scores (7 vs. 6, $p = 0.046$) and MELD scores (12.17 vs. 9.79, $p < 0.006$) than those without such shunts, indicative of a more rapid progression of disease.[31] Lastly, patients with splenorenal shunts had a significantly lower cumulative survival rate than those without such shunts ($p = 0.014$). Cox regression analysis also showed that the presence of splenorenal shunts was a risk factor for death in cirrhotic patients (hazard ratio = 4.161, 95% confidence interval = 1.215–14.255, $p = 0.023$) due to the factors mentioned, including lower portal vein diameter, smaller liver volume, and worse liver function.[31] For these reasons, careful and timely patient selection is paramount to prevent further progression of cirrhosis while also mitigating the significant risks associated with the treatment of NVEPS.

Interventional radiologic embolization is a useful technique for managing the portal hypertension complications of NVEPS. The main indications for intervention include bleeding from gastric varices sustained by a gastrorenal shunt and recurrent HE due to the presence of NVEPS.[32]

Embolization of large shunts has been shown to be both effective and safe in the treatment of recurrent or refractory HE. Balloon-occluded retrograde transvenous obliteration is an effective therapy to treat gastric varices and refractory HE associated with NVEPS by placing a balloon catheter retrograde into a gastrorenal shunt and injecting sclerosant into the varices to promote thrombus formation while the blood flow is occluded.[32] Newer techniques, such as plug-assisted retrograde transvenous obliteration and coil-assisted retrograde transvenous obliteration, use both a plug and coil, respectively, to achieve the same outcome as balloon-occluded retrograde transvenous obliteration with reduced procedure time and fewer complications, especially eliminating the risk of sclerosing agent migration into the portal vein.[33,34] Several studies, as delineated in Table 2,[32,35–40] reported resolution of HE in approximately 60% of patients within three months post-embolization and 49–55% within one to two years, respectively. Late recurrences of HE, attributed to the

**Table 2. Shunt embolization and development of recurrent hepatic encephalopathy**

| Type of shunt | Technique | Embolization success rate (%) | HE status | Reference |
|---|---|---|---|---|
| Splenorenal; Paraumbilical; Mesocaval; Mesorenal | CARTO; PARTO | 100% | Short term: 100 days 59.4%; Long term: two years 48.6% | Laleman *et al*.[35] |
| Splenorenal; Others | CARTO; PARTO | 100% | Short term: 1–4 mo. 100%; Long term: 6–12 mo. 92% | Lynn *et al*.[36] |
| Splenorenal; Paraumbilical | CARTO; PARTO | 100% | Recurrence of HE for two years: 39.9% | An *et al*.[37] |
| Splenorenal; Gastrorenal; Mesocaval; Portocaval | BRTO; CARTO | 92.9% | HE disappearance in one-two weeks: 93% | Naeshiro *et al*.[38] |
| Splenorenal | BRTO | 100% | HE improvement: 100% | Inoue *et al*.[39] |
| Splenorenal; Mesocaval; Other | CARTO; PARTO; SSO | 95.2% | HE improvement:; Short-term: 71%; Long-term: 23% | Philips *et al*.[32] |
| Splenorenal; Mesocaval; Paraumbilical; Others | BRTO; CARTO; PARTO; CAATO | 100% | Recurrence of HE at 9 mo:; 4.5% - early embolization group, 28.6% - late embolization group | Philips *et al*.[40] |

CARTO, coil-assisted retrograde transvenous obliteration; BRTO, balloon-occluded retrograde transvenous obliteration; PARTO, plug-assisted retrograde transvenous obliteration; SSO, selective shunt occlusion; CAATO, coil-assisted antegrade transvenous obliteration.

development of new collaterals or recanalization of occluded shunts, occurred in a small proportion of patients, especially those with high MELD scores at baseline and thus at greater risk.[40] In addition, studies have shown that blood flow within the portal vein increased significantly within the first three months post-embolization, which led to notable improvement in liver function, as reflected by increased serum albumin in individuals classified as Child-Pugh A and B.[41]

Procedural complications of shunt embolization included new onset or worsening ascites and acute esophageal variceal bleeding.[42] Patients with recurrent or refractory ascites or large gastroesophageal varices were not ideal candidates for shunt occlusion. Risk stratification based on MELD score (11–15), Child-Pugh score (<11), and liver elastography (up to 21.6 kPa) reportedly predicted favorable outcomes, and such screening measures were recommended prior to shunt embolization.[15,43]

While bleeding from gastric varices occurred less frequently than from esophageal varices (10%–30%, respectively), there was a higher severity of bleeding and often greater difficulty in achieving hemostasis. In addition, there was an increased risk for complications such as early rebleeding and recurrent bleeding.[44] This is largely because gastric varices are associated with large gastrorenal shunts in up to 85% of cases.[44] Gastric varices act like low-pressure, high-volume channels and can bleed at lower pressures than esophageal varices (15–20 mm Hg vs. 21–23 mm Hg, respectively) and at portosystemic gradients <12 mm Hg in a small subset.[45,46] Thus, the management of gastric variceal hemorrhage requires a different therapeutic approach, where interventional radiology embolization remains a cornerstone. However, the risk of rebleeding is substantial, and therefore, optimal treatment remains inconclusive.

## NVEPS and TIPS

An additional consideration in the management of NVEPS is the influence of NVEPS on TIPS. TIPS is most efficacious in decompressing the portal system and decreasing the risk of bleeding in patients with isolated esophageal varices or type I gastro-esophageal varices.[47] Splenoportography during TIPS often reveals large NVEPS. Logically, such NVEPS should decrease or collapse after TIPS placement due to the normalization of portal pressure and subsequent decrease in blood flow to these collateral vessels. However, it has been shown that even after TIPS placement, approximately one-third of NVEPS remain unchanged and can potentially compete with TIPS for portal flow.[48] These shunts, especially when associated with varices, can lead to an increased incidence of rebleeding. Furthermore, the placement of TIPS in such patients has been shown to increase the risk of HE because TIPS causes additional portosystemic shunting and decreases the already compromised hepatic perfusion. It is unclear whether a coexistent NVEPS has an impact on post-TIPS outcomes, and more importantly, whether they need to be embolized. Occlusion of NVEPS during TIPS might decrease the incidence of HE and improve hepatic synthetic function but may also theoretically lead to exacerbation of portal hypertension and rebleeding risk. He *et al*. performed a retrospective study that showed pre-existing large nonvariceal NVEPS were associated with a higher risk of overt HE, which was decreased by prophylactic embolization of NVEPS during TIPS placement.[49] It was noted that embolization had no clear influence on clinical relapse, shunt dysfunction, or mortality after TIPS. Further, Leng *et al*. investigated the combination of TIPS and shunt embolization in variceal bleeding and had similar results.[50] Lv *et al*. performed a randomized controlled trial to investigate whether prophylactic embolization of large NVEPS at the time of TIPS could reduce the incidence of overt HE after TIPS in patients with cirrhosis and variceal bleeding.[51] Over a three-year period, 56 patients were assigned to receive TIPS alone (TIPS) or TIPS plus simultaneous NVEPS embolization (TIPS + E). The primary endpoint for analysis was met in 15 patients (51.7%) in the TIPS group and six patients (22.2%) in the TIPS + E group ($p$ = 0.045).[51] During a median follow-up of two years, the incidence of overt HE was significantly lower in the TIPS + E group compared with the TIPS group (21.2% vs. 48.3%; HR, 0.38; 95% CI, 0.15–0.97; $p$ = 0.043).[51] Of note, the two-year incidence of secondary endpoints, including recurrent bleeding (15.4% vs. 25.1%; $p$ = 0.522), shunt dysfunction (12.3% vs. 18.6%, $p$ = 0.593), death (15.0% vs. 6.9%, $p$ = 0.352), and other adverse events, was not significantly different between the two groups.[51] Therefore, it remains convincing that concurrent NVEPS embolization at the time of TIPS creation prevents post-TIPS overt HE.

However, effects on other complications such as the risk of rebleeding and shunt dysfunction remain unclear, requiring further investigation. Based on current evidence, it is recommended to evaluate the effects of TIPS with close monitoring of diameter and portal pressure in case adjustment or revision is required. If significant bleeding recurs, embolization of the NVEPS would be the next consideration, taking into account that complete occlusion may restore high portal pressure and increase the risk of variceal bleeding. For NVEPS not associated with varices, a decision can be made based on shunt size and post-TIPS splenoportogram. If any NVEPS show contrast opacification on splenoportogram, they should ideally be embolized to decrease the risk of post-TIPS HE, liver failure, and early TIPS dysfunction.

## NVEPS and liver transplantation

Much like the relationship between NVEPS and TIPS creation, the influence of NVEPS on liver transplantation should be critically evaluated to ensure successful transplant outcomes. Some previous studies reported that the persistence of large NVEPS after transplantation can cause primary nonfunction/dysfunction of the graft, a higher risk of PVT, and development of HE due to a "portal steal" phenomenon driven by the diminished blood flow to the graft.[52–58] Gómez-Gavara *et al*. performed a study of patients with splenorenal shunts >1 cm, with approximately half having the shunt ligated during surgery and the remaining having the shunt left in place.[57] Ligation of NVEPS during transplantation was found to be associated with less postoperative morbidity, HE, and PVT, as well as better patient and graft long-term survival during a 25-month follow-up.[57] Recently, Allard *et al*. observed that pre-transplant PVT and the size of splenorenal shunts were predictors of portal complications (e.g., portal stenosis, thrombosis, or hepatofugal flow) in recipients of living-donor liver transplantation.[58] In particular, they reported that PVT and increasing splenorenal shunt diameter led to an increased risk of portal complications (diameter <8 mm conferred 8.3% risk, diameter 8–15 mm, 16.7%, and diameters >15 mm, 38.5%).[58]

In contrast, Saks *et al*. performed a retrospective analysis of patient outcomes undergoing liver transplantation with intact splenorenal shunts and found that the presence of NVEPS was not associated with post-transplant mortality or graft failure compared with cirrhotic patients without shunts. Despite no ligation, almost half of the evaluated shunts spontaneously decreased in size after liver transplantation.[8] Rodríguez *et al*. performed a study in which the majority of NVEPS were not ligated and found that NVEPS did not influence graft survival or patient morbidity, regardless of the size of collaterals or type of graft used.[7] In summary, the management of NVEPS after liver transplantation remains unclear. Current recommendations suggest a conservative approach to NVEPS during liver transplantation, with possible consideration for intraoperative intervention for large NVEPS associated with a higher risk of portal complications (e.g., pre-liver transplant PVT).

## Conclusions

Acquired SEPS are associated with portal hypertension, cirrhosis, and complications of decompensation. In particular, the effects of NVEPS on portal pressure are variable, but there is evidence of mitigation of portal hypertension and consequent gastro-esophageal varices. The risk of PVT may be increased due to decreased portal flow. HE may be a difficult management problem. The finding of end-stage cirrhosis with poorly responsive HE in the absence of esophageal or gastric varices, umbilical or hemorrhoidal venous dilatation, may be clues to the presence of NVEPS. The portal steal phenomenon, which leads to poor liver nutritional metabolism and toxic clearance, deterioration of liver function, and small liver volume, ultimately leads to higher mortality rates than those without NVEPS. Percutaneous endovascular therapy and TIPS have been widely investigated and implemented to mitigate complications and prevent further progression of cirrhosis. Accurate diagnosis, management, surveillance strategy, and timely intervention of NVEPS are paramount for potential improvement in survival outcomes. However, due to the complex and dynamic nature of liver function, the influence of both variceal SEPS and NVEPS, and other individual patient characteristics, management decisions need to be made on a case-by-case basis.

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## Conflict of interest

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## Author contributions

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