Journal of Clinical and Translational Hepatology **2026**
DOI: 10.14218/JCTH.2026.00334

Review Article

Advances in Precise Assessment of Portal Pressure in Porto-sinusoidal Vascular Disease

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Received: April 27, 2026 | **Revised:** May 15, 2026 | **Accepted:** May 26, 2026 | **Published online:** June 26, 2026

Abstract

Porto-sinusoidal vascular disease (PSVD) is a non-cirrhotic vascular liver disorder characterized by portal and sinusoidal microvascular lesions and is frequently complicated by portal hypertension. Accurate assessment of portal pressure is essential for diagnosis, risk stratification, therapeutic decision-making, and prognostic evaluation in PSVD. However, unlike cirrhosis, portal hypertension in PSVD is predominantly presinusoidal, making hepatic venous pressure gradient measurement prone to underestimating true portal pressure. Recent advances have promoted a transition from conventional invasive assessment toward a multimodal and precision-oriented strategy integrating non-invasive and minimally invasive techniques. Ultrasound elastography, computed tomography, and magnetic resonance imaging—particularly radiomics-based approaches—provide valuable tools for differentiating PSVD from cirrhosis and estimating the severity of portal hypertension. Endoscopic ultrasound-guided portal pressure gradient measurement has emerged as a promising minimally invasive technique for direct hemodynamic assessment and prognostic stratification. In addition, laboratory biomarkers, digital modeling, and artificial intelligence-assisted analysis may further improve individualized risk prediction and dynamic monitoring. This review summarizes current advances in portal pressure assessment in PSVD, critically discusses the strengths and limitations of existing approaches, and highlights future directions toward non-invasive, digital, and precision-guided management.

Citation of this article: Zhu K, Fu Q, Liu M. Advances in Precise Assessment of Portal Pressure in Porto-sinusoidal Vascular Disease. J Clin Transl Hepatol 2026. doi: 10.14218/JCTH.2026.00334.

Keywords: Porto-sinusoidal vascular disease; Portal hypertension; Hepatic venous pressure gradient; Endoscopic ultrasound; Elastography; Radiomics; Artificial intelligence; Portal pressure assessment.

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Introduction

Porto-sinusoidal vascular disease (PSVD) is a clinicopathological entity characterized by microvascular lesions involving the portal vein and hepatic sinusoids. Pathologically, it is defined by characteristic vascular lesions in the absence of established cirrhosis, although it may coexist with other chronic liver diseases and is often associated with immune disorders, hematologic diseases, and prothrombotic states. Clinically, patients may or may not present with portal hypertension.[1,2] Portal hypertension is a major consequence of liver disease progression. Although cirrhosis is the most common cause, non-cirrhotic causes should not be overlooked. Traditionally, "portal hypertension without cirrhosis or portal vein thrombosis" was termed idiopathic non-cirrhotic portal hypertension (INCPH). However, INCPH lacks specific diagnostic markers, relies largely on exclusion, and has been described by multiple overlapping terms—such as portal sclerosis, non-cirrhotic portal fibrosis, incomplete septal cirrhosis, and nodular regenerative hyperplasia—creating confusion in clinical practice and research.[3] To address these limitations, the Vascular Liver Disease Interest Group proposed the more precise term PSVD in 2019. Compared with INCPH, PSVD does not require portal hypertension for diagnosis, thereby including early or asymptomatic stages; it allows coexistence with other liver diseases, better reflecting clinical reality; and it highlights the key anatomical sites of injury—the portal vein and hepatic sinusoids—reflecting a more accurate understanding of the underlying pathophysiology.[1] Unlike cirrhosis, portal hypertension in PSVD is predominantly presinusoidal and is characterized by heterogeneous microvascular remodeling, making conventional hepatic venous pressure gradient (HVPG) assessment less reliable in some patients. Precise assessment of portal pressure is therefore not only essential for diagnosis and risk stratification in PSVD, but also increasingly important for therapeutic decision-making, monitoring treatment response, and individualized management. In this review, we summarize recent advances in invasive and non-invasive portal pressure assessment in PSVD, with particular emphasis on emerging imaging technologies, endoscopic ultrasound (EUS)-guided pressure measurement, and digital and artificial intelligence (AI)-assisted assessment strategies.

Previous reviews on PSVD have primarily focused on its pathological features, clinical manifestations, and general

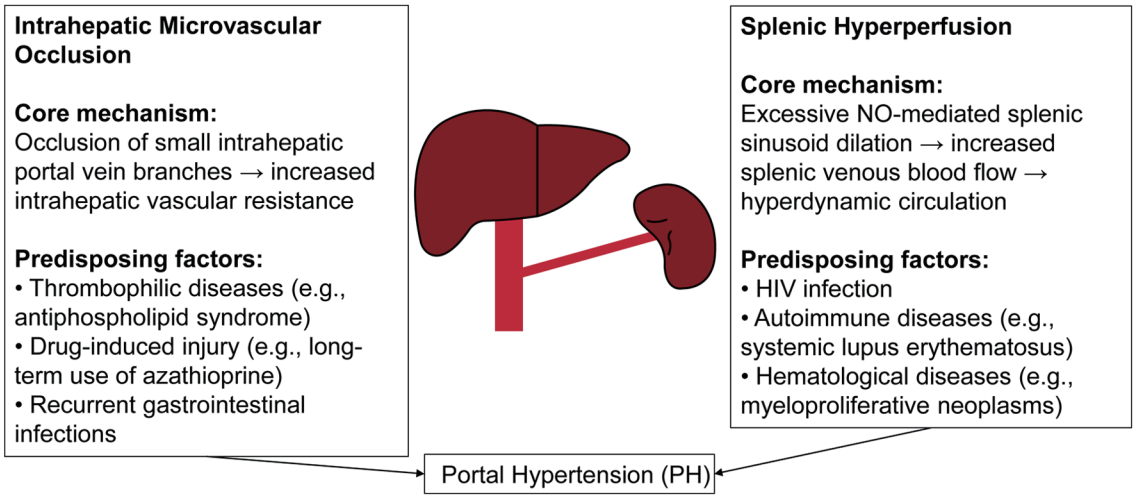

Fig. 1. Schematic diagram of abnormal portal venous system hemodynamics in PSVD patients. PSVD, Porto-sinusoidal vascular disease.

management strategies. In contrast, the present review specifically focuses on precise assessment of portal pressure in PSVD, emphasizing the unique hemodynamic characteristics of presinusoidal portal hypertension and the limitations of conventional HVPG measurement in this setting. Furthermore, this review comprehensively summarizes recent advances in direct and non-invasive portal pressure assessment, including EUS-guided portal pressure measurement, elastography, advanced imaging techniques, radiomics, and emerging AI-assisted assessment strategies. Importantly, we also provide a critical appraisal of the strengths and limitations of current approaches and discuss future directions toward precision-guided management in PSVD.

Epidemiological and pathophysiological characteristics

PSVD shows marked geographical and demographic heterogeneity. Its prevalence is higher in developing countries, accounting for 15%–34% of portal hypertension cases,[4,5] compared with only 3%–6% in Western countries,[6] and has generally declined over time (e.g., in Japan, from 31% in 1975 to 11 cases per year).[7] Demographic patterns also vary. PSVD is more common in males in India and Western countries but predominates in females in Japan.[6,8,9] The age of onset is earlier in India (25–35 years), intermediate in the West (around 40 years), and later in Japan (43–56 years).[8–10] It can also occur in children.[11]

The pathophysiology of PSVD remains incompletely understood but thought to center on abnormal portal hemodynamics, involving hyperdynamic circulation and increased intrahepatic vascular resistance.[12] Two hemodynamic subtypes have been proposed: one is characterized by increased splenic venous flow and moderately elevated portal pressure, possibly related to excessive nitric oxide production and splenic sinusoidal dilation; the other is characterized by increased vascular resistance due to occlusion of small intrahepatic portal vein branches, corresponding to obliterative portal venopathy.[5] These mechanisms support the "double-hit" hypothesis, in which splenic hyperperfusion and intrahepatic microvascular occlusion jointly increase portal pressure (Fig. 1). Moreover, 43%–58% of patients have associated precipitating factors, including immune disorders, hematologic diseases, thrombophilia, HIV infection, recurrent gastrointestinal infections, congenital or familial defects (e.g., telomerase complex, KCNN3, or DGUOK mutations; Adams–Oliver syndrome; Turner syndrome; cystic fibrosis), and drug exposure.[1,5,9,10,13] These factors may promote disease by damaging the portal venous endothelial microenvironment, making comorbidity screening essential in clinical practice.

Advances in portal vein pressure assessment techniques

The diagnosis of PSVD requires adequate liver histopathology (ideally a core biopsy length ≥20 mm) to confirm the absence of established cirrhosis and the presence of characteristic portal/sinusoidal vascular lesions, regardless of clinical manifestations of portal hypertension. Current criteria require either at least one specific clinical or pathological sign, or both a non-specific clinical sign and a non-specific pathological sign (Table 1).[14] Importantly, the term "incomplete septal fibrosis/cirrhosis" in older literature refers to incomplete fibrous septa and should not be considered established cirrhosis.

Regardless of etiology, portal hypertension remains the central clinical issue in PSVD and is closely associated with complications such as variceal bleeding, ascites, and portal vein thrombosis.[15] Because PSVD often presents with preserved liver function and atypical hemodynamic features, accurate portal pressure assessment is essential for differential diagnosis, risk stratification, therapeutic decision-making, and dynamic monitoring of disease progression and treatment response.

Invasive methods for assessing portal vein pressure in patients with PSVD

Hepatic vein angiography

In PSVD, portal hypertension is mainly presinusoidal; thus, wedged hepatic venous pressure (WHVP) may remain normal, causing HVPG to underestimate true portal pressure. Hepatic venography shows intrahepatic venovenous communications in over 50% of patients, further impairing WHVP measurement and resulting in normal or only mildly elevated HVPG values (often <10 mmHg) (Fig. 2).[16–18]

Table 1. Clinical and pathological signs for the diagnosis of porto-sinusoidal vascular disease

Clinical signs	Pathological signs
Specific	
Varices	Obliterative portal venopathy
Portal hypertensive bleeding	Nodular regenerative hyperplasia
Portosystemic collaterals	Incomplete septal fibrosis/cirrhosis
Non-specific	
Ascites	Portal tract abnormalities (arterial hyperplasia and dilation, periportal vascular channels, and aberrant vasculature formation)
Thrombocytopenia	Architectural disarray (abnormal distribution of portal tracts and central veins)
Splenomegaly	Non-zonal sinusoidal dilation
	Mild perisinusoidal fibrosis

These findings support the presinusoidal hemodynamic mechanism of PSVD.

Measurement and clinical value of HVPG

HVPG is the gold standard for assessing portal hypertension in cirrhosis. It is measured indirectly using a balloon catheter placed in the hepatic vein to obtain free hepatic venous pressure (FHVP) and WHVP; HVPG is calculated as WHVP minus FHVP (Fig. 2).[19] This reflects the pressure gradient between the hepatic sinusoid and systemic circulation. However, HVPG has limited value in PSVD because portal hypertension is mainly presinusoidal and often accompanied by intrahepatic venovenous shunts, leading to systematic underestimation of true portal pressure. In a study of 53 PSVD patients, the mean HVPG was only 8.9 mmHg, and many patients with ascites or varices had HVPG <10 mmHg. HVPG also correlated poorly with platelet count and failed to predict major outcomes such as ascites, varices, or death.[20]

Thus, HVPG may underestimate disease severity in PSVD and has limited prognostic value, making routine transjugular HVPG measurement debatable.

Direct portal pressure measurement under EUS guidance

EUS-guided direct portal pressure gradient (PPG) measurement is a novel technique for assessing presinusoidal portal hypertension, including PSVD.[21,22] Using a pressure sensor system and linear-array EUS guidance, the portal vein and hepatic vein (or inferior vena cava) are punctured, and PPG is calculated as the difference between portal and systemic venous pressures.[19] Unlike HVPG, EUS-PPG directly measures

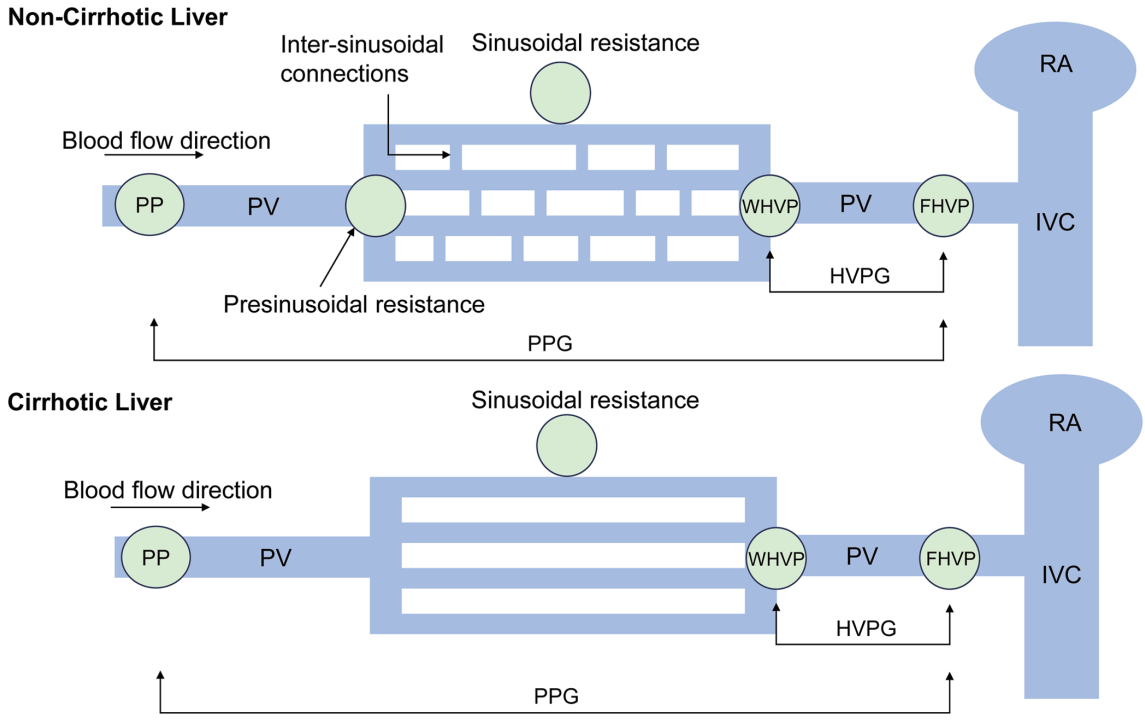

Fig. 2. Direct and indirect measurement of portal vein pressure. PP, portal pressure; WHVP, wedged hepatic venous pressure; FHVP, free hepatic venous pressure; HVPG, hepatic venous pressure gradient; PPG, portal pressure gradient; IVC, inferior vena cava; RA, right atrium; PV, portal vein.

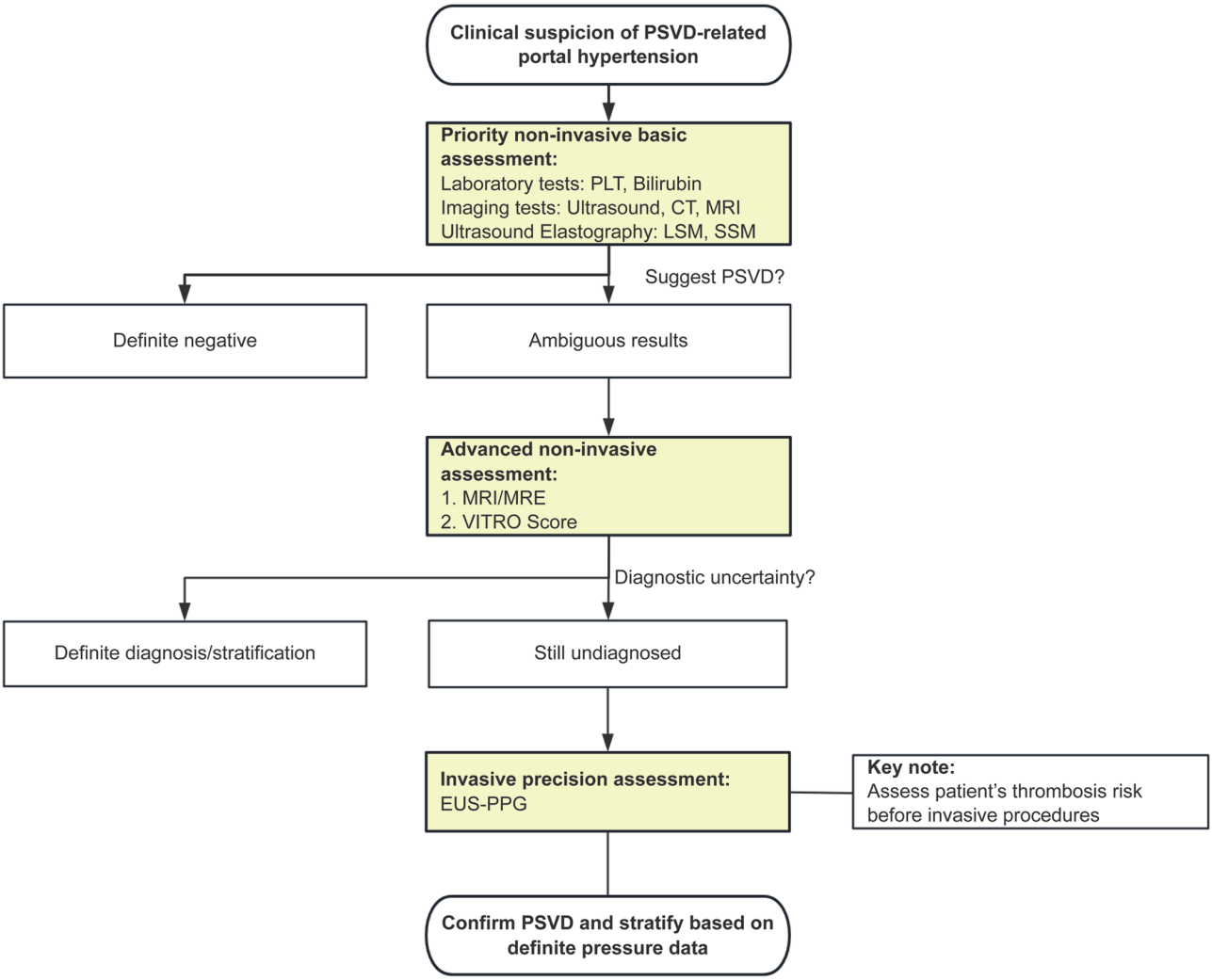

Fig. 3. Stepwise selection flowchart for accurate assessment of portal vein pressure in PSVD patients. PSVD, porto-sinusoidal vascular disease; PLT, platelet count; CT, computed tomography; MRI, magnetic resonance imaging; LSM, liver stiffness measurement; SSM, spleen stiffness measurement; MRE, magnetic resonance elastography; VITRO Score, von Willebrand factor antigen-to-platelet ratio; EUS-PPG, endoscopic ultrasound-guided portal pressure gradient.

portal pressure and more accurately reflects true pressure in presinusoidal portal hypertension.[19]

Preliminary studies support the diagnostic and prognostic value of EUS-PPG in PSVD. Case reports and prospective studies have shown that EUS-PPG is significantly higher than HVPG, confirming that HVPG markedly underestimates portal pressure in PSVD.[23,24] In a prospective cohort of 26 patients, EUS-PPG was the only independent predictor of liver decompensation, with greater prognostic value than HVPG.[23] EUS-PPG may also be useful for treatment monitoring. In nine patients receiving carvedilol, mean EUS-PPG decreased from 17.1 to 15.1 mmHg, although only 44.4% met traditional HVPG-based response criteria, suggesting that PSVD-specific hemodynamic targets may be needed.[23]

Despite its promise, EUS-PPG faces challenges in safety, necessity, and standardization. Its invasive nature may increase the risk of portal vein thrombosis, particularly in PSVD patients with a hypercoagulable state.[25–27] Its clinical necessity is also debated, as it may add limited value in patients with an established diagnosis and clear portal hypertension, while being more useful in diagnostic gray-zone cases.[28] In addition, although EUS-PPG has a high technical success rate, deep sedation may affect portal hemodynamics and measurement accuracy.[29] At present, its use is limited to specialized centers, and wider adoption will require further validation of long-term safety, standardized protocols, and multicenter studies.

Although EUS-PPG provides direct assessment of portal pressure and may better reflect presinusoidal hemodynamics in PSVD, current evidence is still limited by small sample sizes, lack of standardized protocols, and the absence of validated disease-specific cutoff values.

Non-invasive assessment for portal vein pressure in patients with PSVD

Portal pressure assessment in PSVD should follow a stepwise strategy, starting with basic non-invasive methods, progressing to advanced assessment, and using minimally invasive techniques when necessary. This approach aims to accurately quantify portal pressure while minimizing invasive risks, based on clinical signs, underlying disease features, and the applicability of non-invasive tools (Fig. 3).

Ultrasonic elastography

Ultrasonic elastography is a key non-invasive tool for assessing portal hypertension. Its main parameters, liver stiffness measurement (LSM) and spleen stiffness measurement (SSM), are valuable for diagnosing and differentiating PSVD. In PSVD, presinusoidal resistance increases while the liver parenchyma is relatively preserved, so LSM is usually normal or mildly elevated, unlike the markedly increased LSM seen in cirrhosis. In contrast, SSM is often significantly elevated due to splenic congestion and fibrosis. Thus, the characteristic pattern of low/normal LSM with high SSM is an important feature distinguishing PSVD from cirrhosis.[30]

Ultrasound elastography has evolved from single-parameter assessment to multiparametric decision making.[31] The Baveno VII consensus suggests that LSM ≤15 kPa with a platelet count ≥150 × 10^9/L can rule out clinically significant portal hypertension, while SSM provides additional stratification.[14] However, these cirrhosis-derived thresholds should be used cautiously in PSVD. A characteristic non-invasive feature of PSVD is the dissociation between normal or mildly elevated LSM and disproportionately increased SSM.

In PSVD patients without prior variceal bleeding, SSM ≤40 kPa combined with bilirubin <1 mg/dL can safely exclude high-risk varices and reduce unnecessary endoscopy.[32] However, elastography may be limited by obesity and acute inflammatory activity.[33] Overall, combined LSM and SSM assessment is an important non-invasive tool for PSVD diagnosis and risk stratification.

SSM has demonstrated promising diagnostic performance for clinically significant portal hypertension; however, substantial overlap with cirrhosis and variability across platforms may limit reproducibility and generalizability.

Computed tomography (CT)

Dynamic contrast-enhanced CT is useful for diagnosing PSVD. Compared with cirrhosis, PSVD more often shows heterogeneous liver enhancement and characteristic morphological changes, including enlargement of segments IV and I with peripheral atrophy, suggesting abnormal portal flow.[34–37]

CT radiomics enables non-invasive assessment of portal pressure and risk stratification in PSVD. Quantitative imaging features combined with machine learning can reflect portal pressure status. Key models include liver surface nodularity, which correlates with portal hypertension (AUC = 0.88),[38] and CT-based virtual portal pressure gradient (vPPG), which strongly correlates with invasive PPG (r = 0.92).[39] Splenic CT radiomics can also predict high-risk varices and reduce unnecessary endoscopy.[40,41]

Magnetic resonance imaging (MRI)

MRI is important for evaluating and differentiating PSVD. On hepatobiliary agent-enhanced MRI, PSVD typically shows homogeneous high signal intensity in the hepatobiliary phase due to preserved hepatocellular function, unlike the heterogeneous low signal seen in cirrhosis, making it a useful distinguishing feature.

Portal vein hyperintensity on hepatobiliary-phase MRI may indicate more severe portal hypertension,[37,42] although validation against direct pressure measurements is needed. MRI also helps differentiate focal lesions: FNH-like nodules, common in PSVD, often show arterial enhancement without washout and high hepatobiliary-phase signal, mimicking but distinguishable from HCC.[37]

Magnetic resonance elastography quantitatively assesses liver and spleen stiffness. In PSVD, markedly increased spleen stiffness with normal or mildly elevated liver stiffness reflects presinusoidal portal hypertension and helps differentiate PSVD from cirrhosis.

MRI-based radiomics enables non-invasive modeling and risk stratification of portal hypertension in PSVD. Quantitative MRI features and machine learning can detect microvascular lesions and correlate with portal pressure, while splenic extracellular volume fraction, 4D-flow MRI, and virtual HVPG models show promise for non-invasive pressure assessment and variceal risk prediction.[39,43–46] These tools may improve diagnosis, differential diagnosis, and risk stratification in PSVD.

Radiomics-based models have achieved high predictive accuracy in retrospective studies; however, most analyses were conducted in small, single-center cohorts without external validation, raising concerns regarding overfitting and clinical applicability.

Digital and intelligent assessment strategies

Recent advances in radiomics, AI, and computational modeling are promoting the transition of portal pressure assessment in PSVD from conventional morphological evaluation toward quantitative and intelligent analysis. CT- and MRI-based radiomics models can extract quantitative imaging features from the liver, spleen, and portal venous system to predict portal hypertension severity, high-risk varices, and clinical outcomes.[38–41,43–46] Among these approaches, CT angiography-derived vPPG shows a strong correlation with invasive portal pressure measurements (r = 0.92),[39] suggesting that digital hemodynamic modeling may become a promising non-invasive tool for portal pressure estimation.

AI-assisted analysis may further improve the differentiation of PSVD from cirrhosis by integrating imaging, elastography, and clinical parameters into multiparametric predictive models. In addition, emerging techniques such as 4D-flow MRI and computational fluid dynamics may enable individualized assessment of portal venous hemodynamics and dynamic monitoring of disease progression.[43–46] Multi-omics integration combined with AI algorithms may also facilitate PSVD-specific phenotyping and individualized risk stratification.[1,5,47,48]

However, current evidence remains limited by small sample sizes, retrospective study designs, and lack of standardized imaging protocols or external validation cohorts. Furthermore, the interpretability and generalizability of AI-based models remain important challenges. Future multicenter prospective studies are needed to validate digital and intelligent assessment tools before their widespread clinical application in PSVD.

Laboratory and molecular biomarkers

Laboratory and molecular biomarkers are important non-invasive tools for assessing portal hypertension in PSVD, aiding risk stratification and prognosis evaluation. Research has shifted from single markers to multiparametric models; however, most evidence is derived from cirrhosis and should be applied cautiously in PSVD.

Platelet count is a basic serum marker that reflects hypersplenism and inversely correlates with portal pressure. However, its diagnostic accuracy alone is limited, making it more useful as part of composite models such as the Baveno VII criteria and other algorithms.[14]

Among molecular markers, von Willebrand factor antigen is promising because it reflects sinusoidal endothelial injury and correlates strongly with HVPG, with high accuracy for diagnosing CSPH.[49,50] Combined with platelet count as the VITRO score, it further improves risk stratification and re-

duces the diagnostic gray zone of the Baveno VII criteria.[51,52]

Other biomarkers, including the ELF panel, coagulation factors, and soluble CD163, are also associated with portal hypertension and may improve assessment when combined.[53–55] However, current results should be interpreted cautiously due to study population limitations.

Because PSVD is characterized by sinusoidal endothelial injury and portal microvascular occlusion rather than cirrhotic fibrosis, current cirrhosis-derived biomarkers are insufficient for its early diagnosis and prognostic stratification. In addition, biomarker standardization remains challenging due to inter-platform variability.[56,57] Future research should focus on markers of endothelial injury, hypercoagulability, and PSVD-specific genomic/proteomic signatures, supported by large multicenter validation studies.[47,48]

Impact of therapeutic interventions on portal pressure in PSVD

Non-selective β-blockers remain the first-line therapy for the prevention of variceal bleeding in PSVD. However, because PSVD is predominantly characterized by presinusoidal portal hypertension, the applicability of conventional cirrhosis-derived hemodynamic response criteria remains uncertain. Preliminary data showed that carvedilol reduced the mean EUS-PPG from 17.1 mmHg to 15.1 mmHg, whereas only 44.4% of patients achieved the traditional HVPG response threshold.[23] These findings suggest that HVPG may underestimate the true portal decompressive effect in PSVD and that disease-specific hemodynamic targets may be required. The unique presinusoidal hemodynamic pattern of PSVD may partly explain why conventional cirrhosis-derived hemodynamic targets are not fully applicable in this population.

Current management strategies adapted from cirrhosis, including non-selective β-blockers combined with endoscopic variceal ligation, appear effective in controlling portal hypertension-related bleeding in PSVD.[58] For patients with refractory portal hypertension complications, transjugular intrahepatic portosystemic shunt serves as an important portal decompression strategy and may improve outcomes in selected patients, including those with portal vein thrombosis.[59] However, transjugular intrahepatic portosystemic shunt placement in PSVD is often technically challenging because of portal venous abnormalities and coexisting prothrombotic conditions, requiring careful patient selection and performance in experienced centers. Although multicenter cohort studies suggest that cirrhosis-derived management strategies remain effective in PSVD, most available data are retrospective and observational, and prospective hemodynamic response studies are lacking.

Portal vein thrombosis is closely associated with abnormal portal hemodynamics in PSVD and may further aggravate portal hypertension by increasing portal venous resistance. Anticoagulation therapy may improve portal vein recanalization and help preserve portal venous flow, although the optimal treatment strategy remains unclear. Future studies should establish PSVD-specific hemodynamic response criteria and integrate emerging tools such as EUS-PPG and AI-assisted assessment to enable more individualized management.

Disease grading and prognostic assessment

Large cohort studies show that portal pressure is a key prognostic driver in PSVD. Compared with decompensated cirrhosis, PSVD generally has a better prognosis, with 1-, 5-, and 10-year transplant-free survival rates of 97%, 83%, and 72%, respectively.[58] This favorable outcome is likely related to relatively moderate portal hypertension in most patients, highlighting the importance of pressure control.[23]

Portal vein thrombosis is a major complication of PSVD, with a 5-year incidence of about 16%.[58] Portal vein thrombosis and portal hypertension form a vicious cycle, worsening clinical outcomes and increasing mortality.[60,61] Continuous portal pressure monitoring may help identify patients at risk of portal vein thrombosis early.[61]

Prognostic assessment in PSVD is now based on a multidimensional framework centered on portal pressure, together with portal vein thrombosis, biochemical markers, and comorbidities. Advanced age, severe comorbidities, ascites, hyperbilirubinemia, renal dysfunction, and hypoalbuminemia are major predictors of poor outcome.[23] Although HVPG correlates with prognosis, it adds limited value to clinical–biochemical models. Incorporating accurate pressure metrics such as EUS-PPG and vPPG may further improve individualized risk stratification and prediction of complications.[23]

Limitations

Despite recent advances in portal pressure assessment, several important challenges remain in PSVD. Conventional HVPG measurement may underestimate the true severity of portal hypertension, limiting its value for risk stratification and therapeutic monitoring in some patients. In addition, the lack of PSVD-specific hemodynamic thresholds further complicates the interpretation of treatment response and disease progression.

Emerging techniques, including EUS-PPG measurement, elastography, radiomics, and AI-assisted imaging analysis, have shown promising potential for improving portal pressure assessment. However, important limitations remain. EUS-PPG is still invasive and lacks standardized cutoff values, while elastography may be influenced by fibrosis and splenic congestion. Similarly, radiomics and AI-based models are currently limited by retrospective study designs, small sample sizes, and insufficient external validation. The interpretability and reproducibility of AI algorithms also remain important concerns before widespread clinical application.

Future directions

Future research should focus on establishing standardized and disease-specific assessment strategies for PSVD. Integration of multimodal approaches, including direct portal pressure measurement, advanced imaging, elastography, molecular biomarkers, and AI-assisted analysis, may enable more accurate hemodynamic evaluation, dynamic treatment monitoring, and individualized management. Importantly, future studies should also determine whether dynamic changes in portal pressure are superior to static baseline measurements for predicting clinical outcomes in PSVD. Large prospective multicenter studies are needed to validate these emerging technologies and clarify their role in routine clinical practice.

Conclusions

Precise portal pressure assessment is increasingly driving precision management in PSVD. The diagnostic paradigm has shifted from exclusion-based criteria to the PSVD concept centered on specific vascular lesions, while assessment has evolved from reliance on invasive HVPG to a stratified strategy led by multimodal non-invasive tools and supported by minimally invasive methods. Ultrasound elastography identifies the characteristic liver–spleen stiffness dissociation

pattern, and CT/MRI radiomics further enable non-invasive quantitative pressure modeling.

Future research will move toward non-invasiveness, digitalization, and intelligent systems. Standardization of EUS-PPG and emerging tools such as sublingual vein imaging may improve direct or surrogate pressure assessment. Meanwhile, AI integrating clinical, imaging, and multi-omics data may enable precise subtyping and dynamic prognostic prediction of complications.

Ultimately, combining optimized pressure assessment with data-driven precision stratification will enable more individualized interventions and improve survival in PSVD patients.

Collectively, these advances may facilitate the transition from conventional portal hypertension assessment toward precision-guided management in PSVD.

Funding

This work was supported by the Shanghai Medical Young Talent Development Program [Project Number: SHWSRS (2023)], the Youth Program of the Health Commission of Changning District, Shanghai [Project Number: 2023QN27], the "Three-Aviation" Military Medicine Talent Program of the Naval Medical University, the Innovative Cultivation Program for New Medical Technologies of the Naval Medical University [Project Number: XJS2024C22], the Shanghai Rising Star of Medical Talents Youth Development Program [Project Number: SHWSRS (2023)], and the "Deep Blue Ningju" Talent Development Program of the 905th Hospital of the Navy [Project Number: RC 2024(001)].

Conflict of interest

The authors have no conflict of interests related to this publication.

Author contributions

Literature search, data collection, drafting of the initial manuscript (KZ), critical revision of the manuscript for important intellectual content (QF, ML), conception and design, discussion and revision, read and approved the final version for submission (KZ, QF, ML). All authors have approved the final version and publication of the manuscript.

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