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Porto-sinusoidal vascular disease: redefining indications for liver transplantation

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Abstract

Introduction. *Portal hypertension (PH), traditionally regarded as a consequence of liver cirrhosis (LC), in recent decades become the focus of new conceptual frameworks describing its development in non-cirrhotic liver diseases. These observations have led to the identification of a new nosological entity termed a porto-sinusoidal vascular disease of the liver (PSVD), and first proposed by the VALDIG expert group in 2019.*

Case presentation. *This report describes a clinical case of a 34-year-old female patient with PSVD complicated by severe PH, who underwent living donor liver transplantation (LDLT). The presented case highlights the diagnostic challenges and clinical heterogeneity of PSVD, emphasizing the importance of timely recognition of this condition and the need for an individualized approach to determining indications for liver transplantation.*

Discussion. *Despite the emergence of the new PSVD concept, many key aspects of management remain controversial. In particular, there is ongoing debate regarding the selection criteria for liver transplant candidates and the effectiveness of existing therapeutic algorithms. The applicability of standard scoring systems such as Child–Turcotte–Pugh and MELD 3.0 in this pathology also remains uncertain.*

Conclusion. *The diagnosis of PSVD presents significant challenges, largely due to limited awareness among clinicians, as well as the lack of standardized diagnostic criteria and clinical guidelines. Furthermore, indications for liver transplantation in PSVD have not yet been fully standardized. This report aims to discuss these issues and raise awareness about PSVD, which we believe may contribute to improving the quality and effectiveness of patient care.*

Keywords: liver transplantation, porto-sinusoidal vascular disease, portal hypertension, liver cirrhosis, nodular regenerative hyperplasia

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CTP, Child– Turcotte –Pugh score
ESV, esophageal varices
JCI, Joint Commission International
LC, liver cirrhosis
LT, liver transplantation
NRH, nodular regenerative hyperplasia
PH, portal hypertension
PSVD, portosinusoidal vascular disease of the liver
PVT, portal vein thrombosis
SSRS, spontaneous splenorenal shunt
VODL, veno-occlusive disease of the liver

Introduction

Portal hypertension (PH) is a clinical and pathophysiological syndrome, the history of studying this phenomenon goes back more than a hundred years. [1]. During this time, the key mechanisms of its occurrence, as well as the pathophysiological principles underlying the clinical course of the disease, have been clearly defined.

Traditionally, parenchymal PH is considered as a consequence of liver cirrhosis (LC), which remains the most common cause of hepatic portal blood flow block [2]. However, in recent decades, there have been increasing reports of patients in whom signs of PH developed in the absence of classical morphological criteria of LC, but structural changes in the liver parenchyma were noted in the form of obliterative venopathy, nodular regenerative hyperplasia (NRH) and incomplete septal cirrhosis [2]. Although clinical diagnoses in such cases often sounded like "cryptogenic liver cirrhosis", it was clear that the classic morphological signs of cirrhosis were not observed.

In a number of patients, morphological signs similar to those described in 1920 in South Africa for the veno-occlusive disease (VODL) of the liver, which was first observed after poisoning with ragwort and which was called “Senecio cirrhosis” (*Senecio jacobaea* or Jacob's ragwort) [3]. In turn, in VODL, the clinical phenotype, as well as morphological changes in the liver, were more often consistent with acute injury [4]. Thus, VODL and sinusoidal obstruction syndrome, defined by the Seattle and Baltimore criteria, differed from other liver diseases that occur with chronic damage to the sinusoids [5]. Moreover, in contrast to pre- and posthepatic portal block, which diagnosis does not cause great difficulties, with respect to parenchymal vascular diseases, the key pathophysiological question remains open: *do vascular anomalies cause primary damage to the parenchyma, or*

do vascular disorders lead to damage to the parenchyma and impaired liver function? [6].

The accumulated clinical and morphological observations, as well as the pronounced terminological pluralism that existed in the literature, necessitated the unification of the classification and nosological criteria for non-cirrhotic and “fibrotic” forms of PH.

Thus, in different years, such terms as non-cirrhotic portal fibrosis, idiopathic non-cirrhotic portal fibrosis, idiopathic portal fibrosis, Banti disease, etc. were used to designate them [6, 7].

The development of modern non-invasive instrumental technologies has revolutionized hepatology [8], significantly expanding the possibilities of visualizing intrahepatic vascular architecture, which has made it possible to clarify the pattern of parenchymal changes in various vascular parenchymal diseases of the liver and to come closer to understanding a single pathogenetic continuum.

As a result, in order to systematize the obtained data and develop unified diagnostic and nosological criteria, in 2019, an expert group – *Vascular Liver Disease Interest Group* (VALDIG) proposed a new, more unified term “porto-sinusoidal vascular disease of the liver” (PSVD), which changed the existing paradigm of understanding the so-called non-cirrhotic PH [2, 9].

This article presents a clinical case of a patient with PSVD complicated by severe PH, who underwent related liver transplantation (LT).

Clinical Case Report

Patient N., born in 1991 (age 34), was referred to the transplant center by a regional hepatologist due to severe splenomegaly (15.8x7.8x18.2 cm) and esophageal varices (EVs). Based on the examination results and the

inability to determine a suitable diagnosis in the local hepatology outpatient setting, the patient was referred to the transplant center.

At the time of presentation, the patient had no specific complaints. Examination revealed a venous varix at a risk of bleeding. Liver ultrasound and fibroelastometry (FibroScan® device (Echosens, France)) revealed minimal signs of fibrosis—parenchymal elasticity of 7.3 kPa, corresponding to stage F2 on the METAVIR scale. The platelet count was 185×10^9/L. Viral, toxic, and metabolic causes of fibrosis were negated (Table 1).

Table. Laboratory characteristics of the patient

Section / Parameter	Result	Reference values
HEMATOLOGICAL PARAMETERS		
Hemoglobin, g/dL	8.0	11.7–15.5
Hematocrit, %	28.4	35–45
Erythrocytes, $\times10^9$ /L	3.74	3.5–5.2
Leukocytes, $\times10^9$ /L	2.04	4.0–10.0
Platelets, $\times10^9$ /L	185	180–320
BIOCHEMICAL PARAMETERS		
Total bilirubin, μmol/L	15.8	0–19
Direct bilirubin, μmol/L	5.4	0–5
Alanine aminotransferase, U/L	22	0–33
Aspartate aminotransferase, U/L	22	0–33
Gamma-glutamyl transpeptidase, U/L	24	6–42
Albumin, g/L	32	35–52
Total protein, g/L	68	64–82
Cholesterol, mmol/L	3.0	0–5.2
Triglycerides, mmol/L	0.28	0–2.25
Sodium, mmol/L	140	136–145
Urea, mmol/L	5.1	2.5–6.4
Creatinine, μmol/L	54.7	44–80
COAGULOGRAM		
International Normalized Ratio (INR)	1.55	0.8–1.2
INFECTIOUS AND IMMUNOLOGICAL MARKERS		
HBsAg	Not detected	-
Anti-HCV	Not detected	-
Antibodies to *Treponema pallidum*	Not detected	-
ANA (antinuclear antibodies), AU/mL	39.7	< 40 – negative
AMA-M2 (antimitochondrial antibodies), kU/L	<0.15	< 0.15 – negative
OTHER AUTOIMMUNE MARKERS including SP 100; GP210; LC1; LKM; ANCA;	Not detected	—

PCR TESTS (BLOOD PLASMA)		
Human herpes virus, type 6	Not detected	—
Cytomegalovirus	Not detected	—
Epstein-Barr virus	Not detected	—
HORMONAL PARAMETERS		
Thyroid-stimulating hormone, mIU/lL	2.2	0.4–4.0
Free T3, mmol/L	13	10–23
Free T4, mmol/L	4.3	3.1–6.8
METABOLIC AND ADDITIONAL STUDIES		
Ceruloplasmin, mg/dL	22	20–60
Blood iron, µmol/L	4.9	6.6–26
Serum copper, mcg/ dL	88	80–155
Ferritin, ng/mL	178	15–150 (female)
IgG4, mg/dL	5	4–86

Further examination allowed us to establish the following diagnosis: "Clinically manifested chronic liver disease due to PSVD. Clinically significant stage III PH with risk of bleeding, esophageal varices (ESV) (GOV II as defined by the Sarin Classification), splenomegaly. Condition after laparoscopic cholecystectomy. Child-Turcotte-Pugh (CTP) class B (scored 7), MELD 3.0 corresponding to 12 points. Large spontaneous splenorenal shunt (SSRS).

Instrumental examinations. Abdominal ultrasound: Ultrasound examination of the liver showed the organ contours being clear and smooth; the echostructure was heterogeneous due to diffuse changes corresponding to fibrosis signs. Severe portal hypertension signs were noted: dilation of the portal (15 mm) and splenic veins (12 mm), pronounced splenomegaly 190 × 90 cm, the presence of multiple large venous collaterals in the retroperitoneal space. A small amount of free fluid was detected in the abdominal cavity.

Fibroelastometry (FibroScan): Parenchyma stiffness 7.3 kPa, steatosis index was 150 CAP, (F2; S0) according to the METAVIR scale.

Fibrogastroduodenoscopy: Five varices of 0.3–0.2–0.2–0.1–0.1 cm in diameter were visualized in the middle and lower third of the

esophagus. The veins were tense, with a “cherry pit” symptom; they continue into the regions of stomach cardia and fundus (Fig. 1).

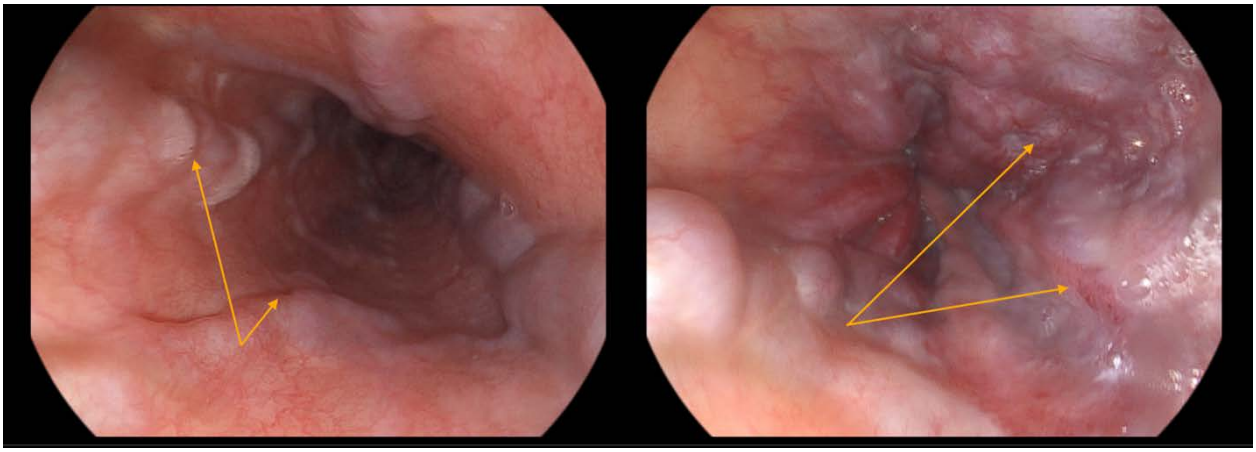


Fig. 1. Esophagogastroduodenoscopy. The presence of esophageal and gastric varices at a risk of bleeding is visualized areas t risk of rupture are marked with arrows

Multislice spiral computed tomography (MSCT): Ectasia of the portal and splenic veins, as well as the veins of the lesser curvature of the stomach and the lower third of the esophagus, were noted. Marked venous ectasia was observed in the area of the splenic hilum with the formation of a large SSRS into the left renal vein. Portal vein mural thrombosis (PVT), type 1, according to Yerdel classification (2000). Splenomegaly, small ascites (Fig. 2.).

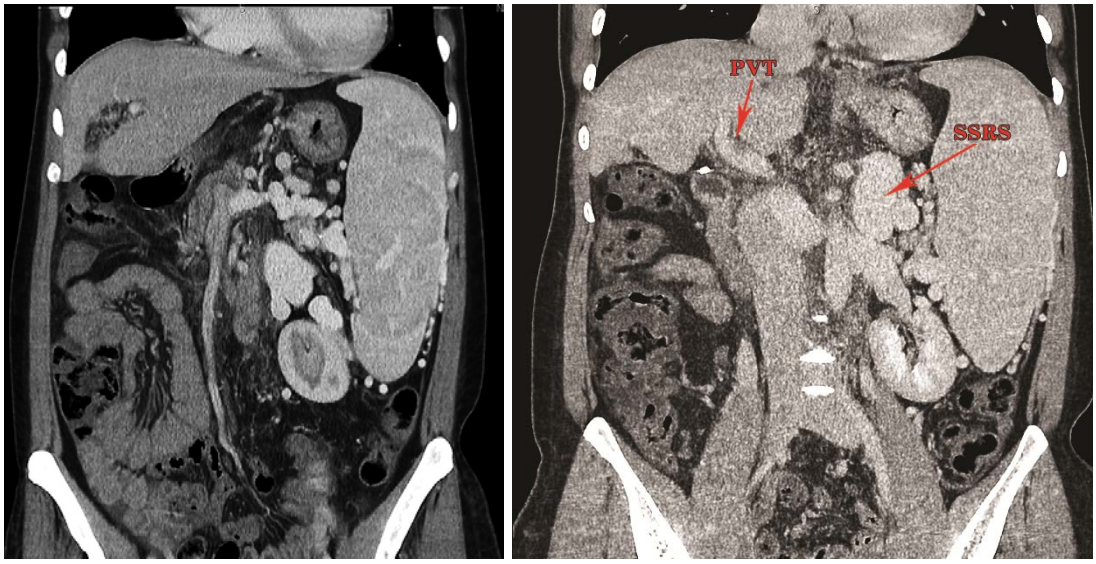


Fig. 2. Three-phase multislice spiral computed tomography scan. The presence of portal vein mural thrombosis and a giant spontaneous splenorenal shunt can be visualized

It is interesting that according to **echocardiography data,** conducted at different times by three independent cardiologists in various clinics having Joint Commission International (JCI) international accreditation, the patient was diagnosed with stage I pulmonary hypertension with an increase in mean pulmonary artery pressure (mPAP) to 38 mm Hg. Although isolated similar cases have been described in the literature [10], we should note that an increase in pulmonary artery pressure in PSVD may be one of the manifestations of severe PH and is regarded as portopulmonary hypertension, which is often an absolute contraindication to LT [11]. This finding served as the basis for right heart catheterization, which results did not confirm the presence of portopulmonary hypertension.

Given the patient's subcompensation – functional class B (CTP) – and the presence of mural PVT, which poses a high risk of rapid decompensation, a related LT was proposed. Intraoperatively, no changes in the organ's macrostructure characteristic of cirrhosis were noted. On palpation, the parenchyma was moderately dense, with isolated foci of a denser, nodular consistency (Fig. 3).

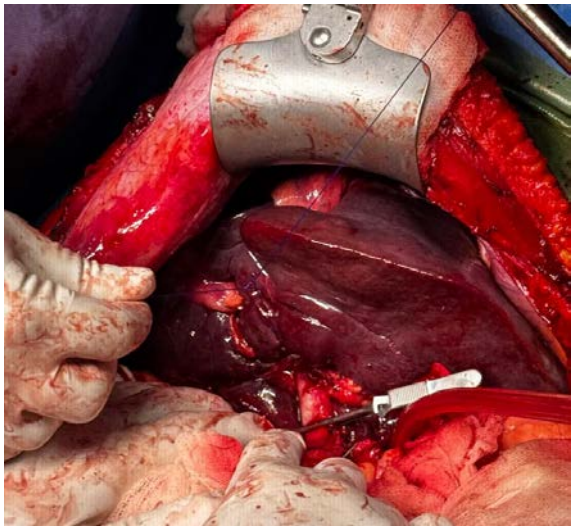


Fig. 3. Intraoperative photo. The liver surface is smooth, the capsule is shiny, with some signs of fibrosis and areas of hardening on palpation

Surgical intervention: LT was performed without intra- and postoperative complications, but with significant technical difficulties associated with severe PH and massive venous collaterals and the need to ligate the left renal vein in order to restore pressure in the portal vein by the exclusion of the SSRS.

Immunosuppressive therapy was administered according to a standard three-component protocol. The patient was transferred from the Intensive Care Unit on the third day and discharged on the 13th postoperative day in satisfactory condition.

Morphological and pathohistochemical examination of the explanted liver revealed the following changes: the parenchymal architecture was preserved, and no signs of bridging fibrosis of hepatocytes were detected. A decrease in the lumen and obliteration of individual portal venules were noted. Focal zones of NRH were observed in some areas. The sinusoids were dilated, predominantly in the centrilobular regions of the lobules. (Fig. 4, 5). Additionally, Perls staining revealed minimal

hemosiderin deposits in the centrilobular hepatocytes. No α_1-antitrypsin-containing deposits were detected; neither morphological signs of granulomatous inflammation, tumor growth, nor other specific changes were detected.

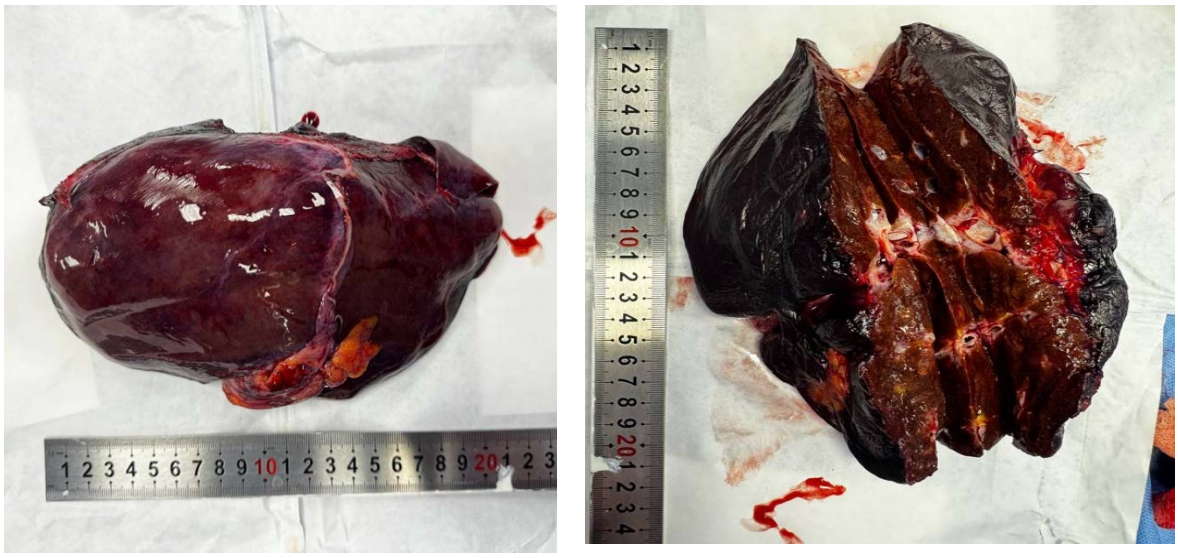
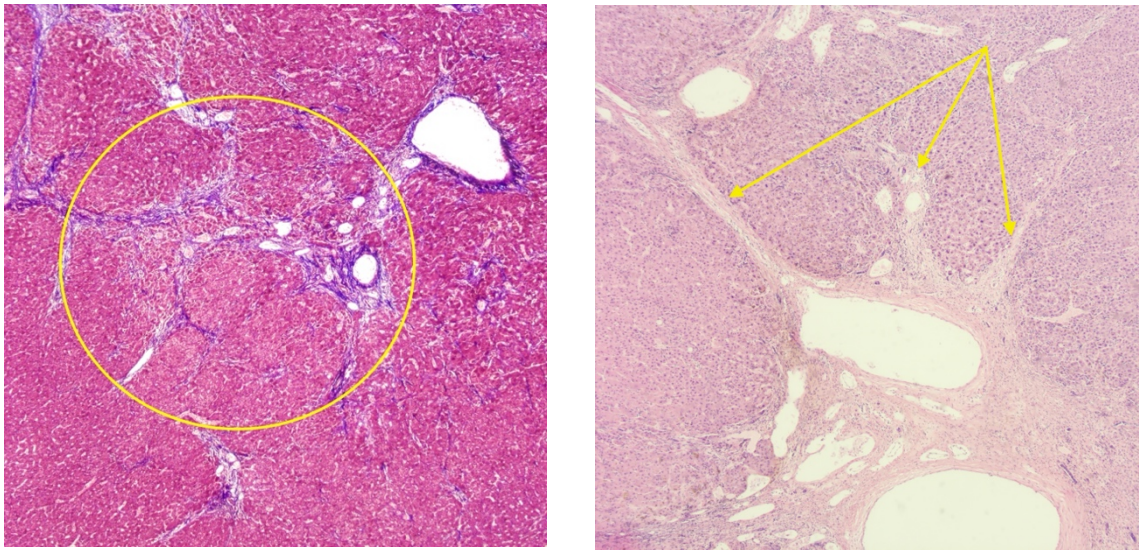


Fig. 4. Macropreparation. Explanted liver



A B

Fig. 5. Microphotography. Histological examination. A, areas of nodular regenerative hyperplasia (NRH) with areas of sinusoidal expansion (NRH foci are marked with a yellow circle); B, incomplete fibrosis (marked with arrows)

Discussion

The term PSVD of the liver proposed in 2019 to designate a group of liver diseases that can lead to the development of PH in the absence of LC, made it possible to unite and systematize our knowledge about diseases previously designated by different terms, while at the same time bringing significant clarity to the understanding of pathological processes associated with sinusoidal obstruction syndrome and other intrahepatic vasculopathies [9].

However, while nomenclature systematization has eliminated terminological pluralism, some uncertainty remains regarding its radical treatment. Thus, despite the fact that, in general, the indications for LT in PSSBP remain the same as for other cirrhotics, data on the results of LT in PSVD of the liver remain very limited [2, 12, 13].

Moreover, progression of liver disease requiring assessment of indications for LT may be associated with significant difficulties when using objective scales such as MELD 3.0 or CTP, based on parameters of liver synthetic function [9].

Thus, in our opinion, the following questions remain open: for whom and when is LT needed? Are existing treatment algorithms for patients with LC applicable to liver PSVD? Can we use the classic CTP and MELD 3.0 scales to determine indications for LT in cases of liver PSVD?

Who needs LT and when? Despite the fact that the indications for LT are generally no different from those used in cirrhosis, the majority of patients with PSVD maintain satisfactory function for a long time, and only in some of them the disease progresses, leading to the need for LT [13, 14]. Thus, the latest large retrospective observational study, which included 587 patients with PSVD, followed in 27 EU centers, only 50 patients (8.5%) underwent LT [14]. Moreover, some studies indicate a

positive role of anticoagulants in the treatment of hypercoagulable status, which often accompanies PSVD [15]. However, no direct randomized trials have been conducted. Furthermore, according to the clinical guidelines “Baveno 2022”, the discussion of indications for LT in patients with PSVD should be carried out individually in each case and only in specialized centers, which once again indicates the lack of standardization regarding indications for transplantation in patients with PSVD of the liver [16].

Are the treatment recommendations for portosinusoidal vascular disease of the liver the same as for liver cirrhosis?

It is known that LT is the only definitive treatment for LC patients, regardless of the presence or absence of PH [17–19]. However, if PSVD is a separate morphological type of cirrhosis, simply defined with a modified term, or a new nosological entity? Currently, opposite points of view remain regarding this issue. Thus, the reason for the discussion was one of the most large-scale publications covering the natural course of liver PSVD, and published in 2025 year in the *Journal of Hepatology*. Its results sparked a vigorous scientific debate among leading experts in the field of hepatology, including S.K. Sarin (former APASL President) and Pierre-Emmanuel Rautou (Hôpital Beaujon, Paris). During the discussion S.K. Sarin publicly noted significant methodological shortcomings of this study related to terminology errors that ultimately led to methodological errors in the study, calling into question the validity of the conclusions obtained from the most extensive and significant population study to date [6].

However, most studies today are still inclined to believe that liver PSVD is not just a terminological change, but the formation of a new nosological paradigm [2, 20, 21].

If this is indeed the case, then it requires additional studies validating the application of existing treatment standards and protocols. This is confirmed by the above and the "Baveno 2022" recommendation: "...with regard to PSVD, there is insufficient data on which method should be preferred for the prevention and control of PH in PSVD..." [16]. Thus, the tactics of the management and prevention of complications developing in PSVD are aimed, first of all, at combating the complications of PH, and most recommendations indicate the need to apply existing treatment algorithms used for any other form of PH [2, 6, 16]. However, it should be taken into account that overall transplant-free survival is higher in the group of patients with PSVD when compared with survival of those with LC, reaching, according to some data, 72% and 82%, respectively over 10 years of follow-up [14, 22]. This fact creates theoretical grounds for a wider use of TIPS, taking into account the lower risks of decompensation, including in the form of encephalopathy [6]. This approach was already considered in a study conducted by Bissonnette, as a result of which the authors concluded that the placement of TIPS in this category of patients leads to excellent results [23]. Evaluation of the role of TIPS would be an extremely important step in the creation of more unified treatment algorithms for patients with PSVD. However, unfortunately, conducting direct randomized trials in this area is difficult due to the rarity of the disease and the heterogeneity of the populations [6].

Using standard rating scales

Today, MELD 3.0 and CHTP scales are the rating scales generally accepted for determining indications for LT and approved for use in most countries of the world; however, it is known that PSVD is often

accompanied by PVT, which can be considered as a manifestation of hypercoagulable status and an element of the PSSBP phenotype [2, 5, 13].

It is known that PVT can significantly worsen the course of the disease and the overall prognosis of patients with LC, significantly affecting the indices of the synthetic function of the liver [24, 25]. Thus, PVT can alternate the true values of compromised liver functioning, overestimating the assessments by RT and MELD 3.0. In this regard, the question arises: can currently accepted scales be used in determining the indications for LT in patients with PSVD, and what is their impact in cases with related and cadaveric donation programs, taking into account the lower degree of alteration of the liver parenchyma?

Conclusion

In our opinion, portosinusoidal vascular liver disease should be considered an independent nosological entity, encompassing a group of previously disparate and terminologically heterogeneous conditions leading to the development of portal hypertension and progressive liver damage. The presented clinical case demonstrates significant differences in both the clinical course and morphological features of portosinusoidal vascular liver disease. Compared to classic liver cirrhosis, these differences, persistent diagnostic difficulties, and the lack of evidence supporting the effectiveness of standardized approaches to managing this patient population necessitate randomized controlled trials to evaluate the feasibility of standard diagnostic and treatment algorithms for portosinusoidal vascular liver disease.

References

1. Nazyrov FG, Devyatov AV, Babadzhanov AKh, Salimov UR. Evolution of azigoportal dissociation technologies in prevention of

bleedings of portal genesis. *Annaly khirurgicheskoy gepatologii = Annals of HPB Surgery*. 2018;23(1):65–73. (In Russ.). https://doi.org/10.16931/1995-5464.2018-1-65-73

2. De Gottardi A, Rautou PE, Schouten J, Rubbia-Brandt L, Leebeek F, Trebicka J, et al. Porto-sinusoidal vascular disease: proposal and description of a novel entity. *Lancet Gastroenterol Hepatol.* 2019;4(5):399–411. PMID: 30957754 https://doi.org/10.1016/S2468-1253(19)30047-0

3. Pai RK, Van Besien K, Hart J, Artz AS, O'Donnell PH. Clinicopathologic features of late onset veno-occlusive disease/sinusoidal obstructive syndrome after high dose busulfan and hematopoietic cell transplantation. *Leuk Lymphoma*. 2012;53(8):1552–1557. PMID: 22280517 https://doi.org/10.3109/10428194.2012.661052

4. Kumar S, DeLeve LD, Kamath PS, Tefferi A. Hepatic veno-occlusive disease (sinusoidal obstruction syndrome) after hematopoietic stem cell transplantation. *Mayo Clin Proc.* 2003;78(5):589–598. PMID: 12744547 https://doi.org/10.4065/78.5.589

5. DeLeve LD, Valla DC, Garcia-Tsao G. Vascular disorders of the liver. *Hepatology*. 2009;49(5):1729–1764. PMID: 19399912 https://doi.org/10.1002/HEP.22772

6. Sarin SK, Lohse AW, Kamath PS. Poor long-term outcome in patients with porto-sinusoidal vascular disease (PSVD): fact or disease misclassification? *J Hepatol*. 2025;82(1):4–6. PMID: 39306284 https://doi.org/10.1016/j.jhep.2024.09.015

7. Mironova M, Gopalakrishna H, Viana Rodriguez GM, Abdul Majeed N, Hitawala AA, Fuss IJ, et al. Prospective evaluation of patients with non-cirrhotic portal hypertension: a single centre study. *Aliment Pharmacol Ther*. 2024;59(12):1527–1538. PMID: 38629442 https://doi.org/10.1111/APT.17987

8. Castera L, Rinella ME, Tsochatzis EA. Noninvasive assessment of liver fibrosis. *N Engl J Med.* 2025;393(17):1715–1729. PMID: 41160822 https://doi.org/10.1056/NEJMRA2403308

9. Mironova M, Gopalakrishna H, Koh C, Kleiner DE, Heller T. Portal sinusoidal vascular diseases: assessment and therapy. *Hepatology.* 2025;10.1097/HEP.0000000000001344 PMID: 40194304 https://doi.org/10.1097/HEP.0000000000001344

10. Pestana I, Noronha Ferreira C, Luís R, Sousa R, Pereira EA, Banhudo A. Porto-sinusoidal vascular disease and downhill varices: separate clinical entities? *GE Port J Gastroenterol.* 2024;32(1):54–60. PMID: 39906509 https://doi.org/10.1159/000539092

11. Xu H, Cheng B, Wang R, Ding M, Gao Y. Portopulmonary hypertension: current developments and future perspectives. *Liver Res.* 2022;6(1):10–20. PMID: 39959808 https://doi.org/10.1016/J.LIVRES.2022.02.002

12. Krasinskas AM, Eghtesad B, Kamath PS, Demetris AJ, Abraham SC. Liver transplantation for severe intrahepatic noncirrhotic portal hypertension. *Liver Transpl.* 2005;11(6):627–634. PMID: 15915493 https://doi.org/10.1002/LT.20431

13. Magaz M, Giudicelli-Lett H, Nicoară-Farcău O, Rajoriya N, Goel A, Raymenants K, et al. Liver transplantation for porto-sinusoidal vascular liver disorder: long-term outcome. *Transplantation.* 2023;107(6):1330–1340. PMID: 36479977 https://doi.org/10.1097/TP.0000000000004444

14. Magaz M, Giudicelli-Lett H, Abraldes JG, Nicoară-Farcău O, Turon F, Rajoriya N, et al. Porto-sinusoidal vascular liver disorder with portal hypertension: natural history and long-term outcome. *J Hepatol.* 2025;82(1):72–83. PMID: 39181213 https://doi.org/10.1016/J.JHEP.2024.07.035

15. Hernández-Gea V, Baiges A, Turon F, Garcia-Pagán JC. Idiopathic portal hypertension. *Hepatology.* 2018;68(6):2413–2423. PMID: 30066417 https://doi.org/10.1002/HEP.30132

16. de Franchis R, Bosch J, Garcia-Tsao G, Reiberger T, Ripoll C. Baveno VII – Renewing consensus in portal hypertension. *J Hepatol.* 2022;76(4):959–974. PMID: 35120736 https://doi.org/10.1016/j.jhep.2021.12.022

17. Cervantes-Alvarez E, Vilatoba M, Limon-De la Rosa N, Mendez-Guerrero O, Kershenobich D, Torre A, et al. Liver transplantation is beneficial regardless of cirrhosis stage or acute-on-chronic liver failure grade: a single-center experience. *World J Gastroenterol.* 2022;28(40):5881–5892. PMID: 36353203 https://doi.org/10.3748/WJG.V28.I40.5881

18. Salimov UR, Stoma IO, Kovalev AA, Hubanova TN, Fedoruk AM, Shcherba AE, et al. Gut microbiota might influence the risk of rejection after liver transplantation. *Journal of Liver Transplantation.* 2023;9:100140. https://doi.org/10.1016/j.liver.2023.100140

19. Salimov UR, Balachandran P, Tulyaganov DB, Ruzibakieva MR, Khadjibaev FA, Anvarov KE. Rapid suppression of HBV replication before living donor liver transplantation in a patient with HDV superinfection. Clinical case report. *Russian Journal of Transplantology and Artificial Organs.* 2024;26(3):66–71. https://doi.org/10.15825/10.15825/1995-1191-2024-3-66-71

20. Rautou PE, Giudicelli-Lett H, Magaz M, García-Pagán JC. Comment on “Poor long-term outcome in patients with porto-sinusoidal vascular disease (PSVD): Fact or disease misclassification?” *J Hepatol.* 2025;82(6):e334–335. PMID: 39608460 https://doi.org/10.1016/j.jhep.2024.11.031

21. Wöran K, Semmler G, Jachs M, Simbrunner B, Bauer DJM, Binter T, et al. Clinical course of porto-sinusoidal vascular disease is distinct from idiopathic noncirrhotic portal hypertension. *Clin Gastroenterol Hepatol.* 2022;20(2):e251–626. PMID: 33279774 https://doi.org/10.1016/J.CGH.2020.11.039

22. Siramolpiwat S, Seijo S, Miquel R, Berzigotti A, Garcia-Criado A, Darnell A, et al. Idiopathic portal hypertension: natural history and long-term outcome. *Hepatology.* 2014;59(6):2276–2285. PMID: 24155091 https://doi.org/10.1002/HEP.26904

23. Bissonnette J, Garcia-Pagán JC, Albillos A, Turon F, Ferreira C, Tellez L, et al. Role of the transjugular intrahepatic portosystemic shunt in the management of severe complications of portal hypertension in idiopathic noncirrhotic portal hypertension. *Hepato-logy.* 2016;64(1):224–231. PMID: 26990687 https://doi.org/10.1002/HEP.28547

24. Turon F, Driever EG, Baiges A, Cerda E, García-Criado Á, Gilabert R, et al. Predicting portal thrombosis in cirrhosis: a prospective study of clinical, ultrasonographic and hemostatic factors. *J Hepatol.* 2021;75(6):1367–1376. PMID: 34333101 https://doi.org/10.1016/J.JHEP.2021.07.020

25. Liu Z, Yang X, Jiang H, Xie R, Wang H. Advancements of direct oral anticoagulants in cirrhotic individuals with portal vein thrombosis. *Clin Res Hepatol Gastroenterol.* 2025;49(4):102553. PMID: 39983831 https://doi.org/10.1016/J.CLINRE.2025.102553

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