UDC 616.14-007.64:616.329]-005.1-084:[616.149:616.12-008.331.1

S.M. Kozlov[1], N.V. Kosei[2], N.O. Leshchynska[3], N.O. Yakovenko[3], Ya.S. Nazarov[4], O.S. Kozlov[3]

Comparative analysis of secondary prophylaxis strategies for esophageal variceal bleeding in clinically manifest portal hypertension: a 12-year single-center study

[1]Bohomolets National Medical University, Kyiv, Ukraine
[2]Center of Innovative Medical Technologies of the NAS of Ukraine, Kyiv
[3]Shupyk National University of Healthcare of Ukraine, Kyiv
[4]Kyiv Municipal Clinical Hospital No. 12, Ukraine

Paediatric Surgery (Ukraine). 2025. 4(89): 32-40. doi: 10.15574/PS.2025.4(89).3240

For citation: Kozlov SM, Kosei NV, Leshchynska NO, Yakovenko NO, Nazarov YaS, Kozlov OS. (2025). Comparative analysis of secondary prophylaxis strategies for esophageal variceal bleeding in clinically manifest portal hypertension: a 12-year single-center study. Paediatric Surgery (Ukraine). 4(89): 32-40. doi: 10.15574/PS.2025.4(89).3240.

Recurrent variceal bleeding remains a leading cause of mortality in patients with portal hypertension (PH). Standard secondary prophylaxis strategies (SP), including non-selective β-blockers (NSBB) and endoscopic injection sclerotherapy (EIS), demonstrate suboptimal anti-recurrence efficacy.

Aim – to determine the optimal SP method for esophageal variceal bleeding (EVB) by comparing long-term outcomes of partial splenic artery embolization (PSE) versus NSBB and EIS-based strategies.

Materials and methods. This prospective and retrospective study included 514 patients who survived at least one EVB episode. SP efficacy was analyzed in three groups: NSBB monotherapy (n=243), EIS+NSBB (n=151), and PSE+NSBB (n=120). The efficacy evaluation criteria were survival and EVB recurrence rates within 12 months.

Results. During the 12-month follow-up, survival was significantly higher in the PSE group (0.87) than in the NSBB (0.73) and EIS (0.64) groups ($p < 0.001$), respectively. Recurrence-free status at one year was achieved in 56.2% of PSE patients, versus 35.0% in EIS and 24.3% in NSBB. All-cause mortality was lowest after PSE (13.3%) and highest in the EIS group (37.1%).

Conclusions. PSE provides significantly higher SP efficacy compared to standard strategies, offering an effective minimally invasive treatment for high-risk patients with clinically significant PH.

The study was conducted in accordance with the principles of the Declaration of Helsinki. The study approved by the Local Ethics Committee. Informed consent was obtained from all patients. The authors declare no conflict of interest.

Keywords: portal hypertension, esophageal varices, variceal bleeding, secondary prophylaxis, partial splenic artery embolization, endoscopic injection sclerotherapy.

Порівняльний аналіз стратегій вторинної профілактики кровотеч із варикозно розширених вен стравоходу при клінічно маніфестній портальній гіпертензії: 12-річне одноцентрове дослідження

С.М. Козлов[1], Н.В. Косей[2], Н.О. Лещинська[3], Н.О. Яковенко[3], Я.С. Назаров[4], О.С. Козлов[3]

[1]Національний медичний університет імені О.О. Богомольця, м. Київ, Україна
[2]ДНУ «Центр інноваційних медичних технологій НАН України», м. Київ
[3]Національний університет охорони здоров'я України імені П.Л. Шупика, м. Київ
[4]КНП «Київська міська клінічна лікарня №12», Україна

Рецидивні варикозні кровотечі (ВК) залишаються однією з провідних причин смертності при портальній гіпертензії. Стандартна вторинна профілактика (ВП) – неселективні β-блокатори (НСББ) та ендоскопічна склеротерапія (ЕСТ) не забезпечують прийнятного протирецидивного ефекту.

Мета – визначити оптимальний метод ВП кровотеч із варикозно розширених вен (ВРВ) стравоходу на основі аналізу віддалених результатів парціальної емболізації селезінкової артерії (ПЕСА) порівняно з НСББ та ЕСТ.

Матеріали і методи. У проспективно-ретроспективному одноцентровому дослідженні взяли участь 514 пацієнтів, які перенесли щонайменше один епізод кровотечі з варикозних вен стравоходу або шлунку. Аналіз ефективності ВП проведено у трьох клінічних групах: монотерапії НСББ (n=243), ЕСТ у комбінації з НСББ (n=151) та ПЕСА у комбінації з НСББ (n=120). Критеріями оцінки ефективності слугували показники виживаності та частота повторних епізодів ВК протягом 12 місяців.

Результати. У період 12-місячного спостереження виживаність була достовірно вищою у групі ПЕСА (0,87) ніж у групах НСББ (0,73) та ЕСТ (0,64) відповідно. Відсутність рецидивів кровотечі протягом року зафіксовано у 56,2% пацієнтів групи ПЕСА, тоді як у групах ЕСТ та НСББ ці показники становили 35% та 24,3%. Загальна летальність була найнижчою у групі ПЕСА+НСББ (13,3%) та найвищою у групі ЕСТ+НСББ (37,1%).

Висновки. ПЕСА забезпечує достовірно вищу ефективність вторинної профілактики варикозних кровотеч при клінічно маніфестній портальній гіпертензії порівняно з монотерапією НСББ та ЕСТ в комбінації з НСББ.

Дослідження виконано відповідно до принципів Гельсінської декларації та схвалено локальною етичною комісією. Всі пацієнти надали інформовану згоду на участь.

Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: портальна гіпертензія; кровотечі з варикозно-розширених вен стравоходу; вторинна профілактика; емболізація селезінкової артерії; ендоскопічна склеротерапія.

Introduction

Portal hypertension (PH) is the central pathophysiological driver of major complications in cirrhosis [13], most notably the development and rupture of gastroesophageal varices (GEV) [3,7]. An initial episode of esophageal variceal bleeding (EVB) marks the transition to decompensated cirrhosis and is associated with a median survival of approximately 1.5–2 years, underscoring the critical importance of effective secondary prophylaxis (SP) [2].

Before the introduction of pharmacologic and endoscopic strategies, the 1-year risk of rebleeding exceeded 60% [5]. Current approaches, including non-selective β-blockers (NSBB) and endoscopic therapies, have reduced this risk to approximately 30–32%, yet these rates remain unacceptably high, particularly in patients with clinically significant portal hypertension (CSPH), marked splenomegaly, and hypersplenism [11].

Early rebleeding within the first 6 weeks continues to be a leading cause of mortality [13].

Transjugular intrahepatic portosystemic shunt (TIPS) represents a potent modern modality for SP [12]. However, the procedure's high cost, technical complexity, and associated complications most notably hepatic encephalopathy limit its widespread adoption in Ukrainian clinical practice. Consequently, endoscopic techniques and pharmacological treatment remain the predominant standard of care.

In this context, the limited efficacy of standard care has prompted growing interest in minimally invasive interventional techniques aimed at modifying splanchnic hemodynamics. PSE offers a dual mechanism of benefit: it reduces excessive arterial inflow to the portal circulation and mitigates hypersplenism, a key contributor to bleeding risk in CSPH [6,9,14]. Therefore, comparing the efficacy of PSE against these traditional prophylactic methods is a crucial step toward optimizing treatment strategies.

The aim of this study was to determine the optimal SP method for EVB by comparing long-term clinical outcomes, specifically survival and rebleeding risks of PSE versus standard pharmacological treatment and endoscopic sclerotherapy.

Materials and methods of the study

Study Design and Patient Population. This prospective and retrospective single-center study included 514 consecutive patients with liver fibrosis or cirrhosis who survived an index episode of EVB. Clinical history and pre-inclusion bleeding data were analyzed retrospectively, whereas post-intervention outcomes were followed prospectively. All patients were managed according to international guidelines (American Association for the Study of Liver Diseases, Baveno VII) and received standardized acute care prior to allocation to a secondary prophylaxis strategy. The treatment groups were well-matched regarding fundamental demographic parameters.

Patients were subsequently assigned to one of three treatment groups:

NSBB – 243 patients;

EIS + NSBB – 151 patients;

PSE + NSBB – 120 patients.

Basic demographic characteristics of the study population (n=514) are summarized in Table 1.

The choice of secondary prophylaxis was individualized, reflecting clinical severity, bleeding risk, hemodynamic profile, and response to initial therapy. PSE was reserved for patients with severe splenomegaly, hypersplenism, morphologic signs of splenic arterial hyperperfusion, or recurrent bleeding despite pharmacological or endoscopic management.

Table
Baseline demographic and clinical characteristics of the study groups

Variable	NSBB (n=243)	EIS + NSBB (n=151)	PSE + NSBB (n=120)	p-value
Age, years (mean ± SD)	50.2±12.9	51.4±12.2	49.5±10.5	>0.05[1,2]
Male sex, n (%)	154 (63.4%)	95 (62.9%)	70 (58.3%)	>0.05[1,2]

Note: statistically significant or non-significant difference relative: 1 – to NSBB group; 2 – to EIS group.

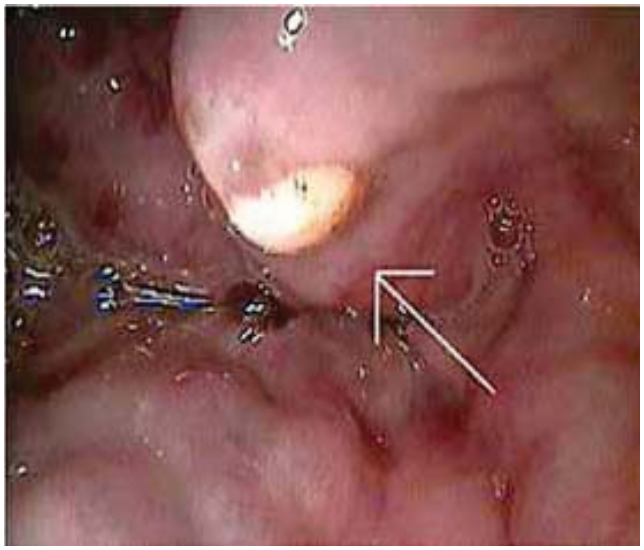

Fig. 1. Endoscopic appearance of an esophageal varix with a «nipple sign». A protruding fibrin plug(«nipple») is seen on the surface of a large esophageal varix (arrow). This finding represents a recent bleeding point and is a well-recognized predictor of high rebleeding risk during acute variceal hemorrhage

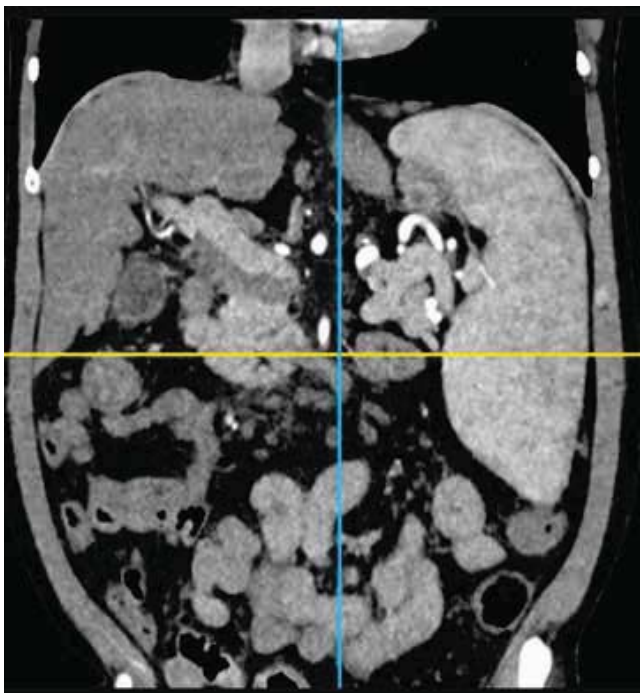

Fig. 2. CT: marked splenomegaly, a nodular liver contour, a patent portal vein, and dilated looping splenic artery segments indicating hyperperfusion

Statistical analysis was performed using EZR (Saitama Medical Center, Jichi Medical University, Saitama, Japan). Continuous variables are expressed as mean ± standard deviation (SD) and were compared using one-way analysis of variance (ANOVA). When ANOVA indicated significant differences, Bonferroni's post-hoc test was applied for pairwise comparisons. Categorical variables were analyzed using the chi^2 test or Fisher's exact test, as appropriate.

To quantify the burden of bleeding, incidence rate ratios (IRR) with 95% confidence intervals (CI) were calculated as the ratio of the total number of bleeding episodes to the number of patients in each group. Survival outcomes (overall and rebleeding-free survival) were estimated using the Kaplan–Meier method, and differences between curves were evaluated with the log-rank test. A two-tailed p-value< 0.05 was considered statistically significant.

Pharmacological Therapy: NSBB. Patients in the NSBB group received propranolol or nadolol according to standard titration protocols. Therapy was initiated after hemodynamic stabilization and titrated to achieve a target heart rate of 55–60 bpm or a 25% reduction from baseline, provided systolic blood pressure remained above 90 mmHg. Patients unable to reach target dosing due to hypotension or intolerance were classified as partial responders, while those requiring discontinuation were defined as non-responders. NSBB monotherapy served as the reference arm for standard pharmacological secondary prophylaxis.

Endoscopic Injection Sclerotherapy (EIS). EIS was performed during the index hospitalization or within 10–14 days of stabilization. During the initial endoscopic assessment, specific stigmata of recent hemorrhage, such as the «nipple sign,» were evaluated as indicators of high rebleeding risk (Fig. 1). Intravariceal injections of sodium tetradecyl sulfate were administered using a standard 23 and 25-gauge sclerotherapy needle in 0.5–1.0 ml aliquots per varix. Sessions were repeated every 10–14 days until variceal obliteration or until the patient was transitioned to maintenance endoscopic therapy.

Complications of EIS were relatively uncommon but clinically relevant. In addition to minor adverse effects such as transient retrosternal discomfort, low-grade fe-

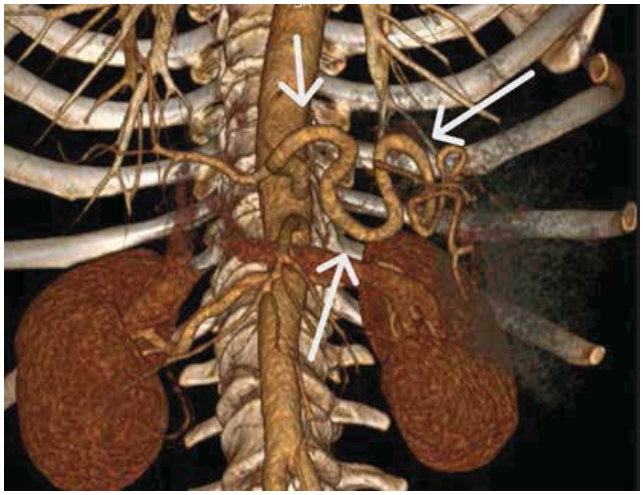

Fig. 3. CT: tortuosity and significant dilation of the splenic artery (white arrows)

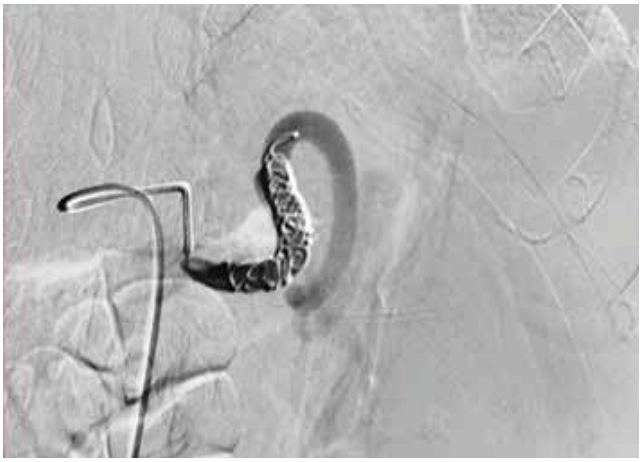

Fig. 4. Intraprocedural angiography of the splenic artery from the same patient demonstrates dense coil packing, contrast reflux from coil clusters, and preserved distal perfusion

ver, or temporary dysphagia, several potentially serious events were observed and are well documented in the literature.

One of the rare but severe complications is the migration of the sclerosant into the systemic or pulmonary circulation. Accidental intravascular injection may allow the sclerosant to enter esophageal perforating veins and subsequently reach the pulmonary arterial branches, where it can cause chemical endothelial injury, segmental vasospasm, or vascular obstruction. In isolated cases, this process may result in focal pulmonary infarction or thromboembolic phenomena, often presenting with pleuritic chest pain and localized consolidation on computed tomography (CT). Mucosal necrosis or ulceration at the injection site is uncommon but can develop when the sclerosant extravasates into submucosal or muscular layers. In more severe cases, this may lead to deep mural injury or esophageal wall necrosis, predisposing to ulceration, delayed strictures, or rarely, microperforation.

Pre-procedural imaging and eligibility assessment for PSE. All patients considered for PSE underwent contrast-enhanced CT as part of the standard pre-procedural evaluation. This imaging modality played a central role in confirming the presence and severity of PH, while simultaneously providing detailed anatomical information necessary for planning the intervention.

CT allowed the assessment of the splenic artery diameter and morphology, quantification of splenic volume, and verification of portal and splenic vein patency. These parameters were essential both for determining procedural feasibility and for anticipating the technical complexity of the embolization (Fig. 2, 3).

Based on CT angiography findings, patients were considered eligible for PSE if they demonstrated radiologic features consistent with significant splenic arterial hyperperfusion. The most important determinants included dilation of the splenic artery to at least 5–7 mm and marked arterial tortuosity, often manifesting as a characteristic corkscrew configuration. Eligibility further required splenic enlargement exceeding 400–500 cm^3, a fully patent portal venous system, and the absence of a proximal stenosis or a celiac trunk obstruction that could compromise inflow dynamics. In addition, the arterial anatomy had to be favorable enough to permit safe and selective catheterization of the intended embolization segment.

Taken together, these imaging criteria ensured that PSE would appropriately target the inflow component of PH, reducing the splenic arterial contribution to portal pressure while preserving adequate splenic venous outflow. This comprehensive pre-procedural assessment was essential not only for procedural safety but also for ensuring a durable long-term hemodynamic response.

Procedural technique. PSE was performed via a transfemoral or transradial approach under fluoroscopic guidance.

After selective catheterization of the splenic artery, a 5F catheter was advanced into the target segment, followed by controlled deployment of metallic coils to reduce arterial inflow (Fig. 4).

Technical success of the procedure was confirmed intraoperatively based on several standardized angiographic criteria. First, a stable catheter position within the target arterial segment was required to ensure controlled deployment of the metallic coils. Successful embolization was characterized by dense and compact coil packing, forming a well-defined occlusion mass. A classic «reflux sign» on post-embolization angiography, with flow attenuation at the coil cluster, served as an essential indicator of adequate inflow reduction. At the same

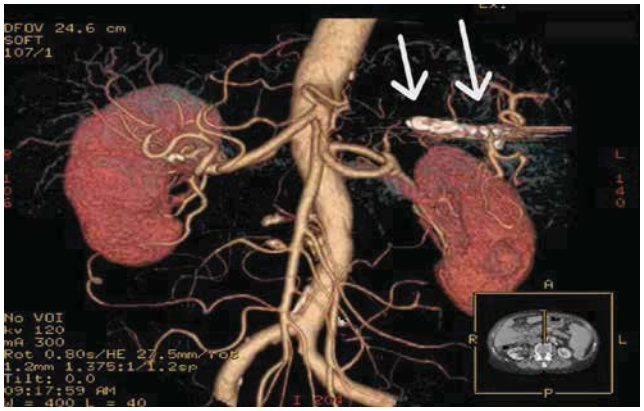

Fig. 5. Representative 12-month post-embolization 3D CT angiography. The image demonstrates a compact, stable cluster of metallic coils (white arrows) with markedly attenuated splenic arterial flow and no evidence of recanalization, consistent with a durable and well-maintained hemodynamic response

time, preserved perfusion of viable splenic parenchyma was mandatory to avoid excessive devascularization. The procedure was considered technically safe when performed without non-target embolization, coil migration, or arterial injury.

Post-embolization imaging and follow-up. The assessment of long-term effectiveness following PSE was performed using a standardized imaging protocol. All patients underwent contrast-enhanced CT at 12 months after the procedure to evaluate the durability of the embolization and confirm a persistent hemodynamic benefit.

The follow-up CT focused on several key parameters. First, a sustained reduction of splenic arterial inflow was assessed by visualizing decreased enhancement of the previously embolized arterial segments. The stability of the metallic coil construct was verified by ensuring that the coil configuration and density remained unchanged, with no evidence of migration or mechanical displacement (Fig. 5).

Additionally, radiologists evaluated the absence of arterial recanalization or collateral reconstitution, which might indicate partial failure or the re-establishment of splenic inflow. The preservation of viable splenic parenchyma was an equally important criterion, confirming that the embolization remained selective rather than organ-ablative. The patency of the portal venous system-including the portal and splenic veins was routinely checked to exclude delayed thrombosis. Clinical endpoints such as the absence of rebleeding and the improvement in cytopenias were assessed in parallel with imaging findings, ensuring a combined anatomical and clinical evaluation of treatment success.

The assessment and usage of all clinical data were approved and permitted before the study by the Ethics Committee of Bogomolets National Medical University. The study protocol conformed to the ethical guidelines of the «World Medical Association (WMA) Declaration of Helsinki – Ethical Principles for Medical Research Involving Human Subjects» adopted by the 18th WMA General Assembly, Helsinki, Finland, June 1964, and amended by the 59th WMA General Assembly, Seoul, South Korea, October 2008. Written informed consent was obtained from all individual participants included in the study.

Table 2
Baseline clinical characteristics of the study population

Variable	NSBB (n=243)	EIS + NSBB (n=151)	PSE + NSBB (n=120)	p-value
Etiology of cirrhosis, n (%):				
- viral (HCV/HBV)	112 (46.1%)	64 (42.4%)	55 (45.8%)	>0.05[1,2]
- alcohol-associated liver disease	89 (36.6%)	57 (37.7%)	41 (34.2%)	>0.05[1,2]
- cryptogenic	42 (17.3%)	30 (19.9%)	24 (20.0%)	>0.05[1,2]
Child-Pugh class, n (%):				
- A	71 (29.2%)	43 (28.5%)	44 (36.7%)	>0.05[1,2]
- B	158 (65%)	100 (66.2%)	67 (55.8%)	>0.05[1,2]
- C	14 (5.8%)	8 (5.3%)	9 (7.5%)	>0.05[1,2]
MELD	7.5±3.6	8.6±3.5	8.7±4.9	<0.05[1]
Platelet count, $\times 10^9$/L	95 ± 51	90.1 ± 23.4	82.7 ± 30.3	<0.05[1,2]
Hemoglobin, g/L	96.5 ± 20.1	93 ± 17	83.3 ± 19.7	<0.01[1,2]
Splenomegaly, n (%)	188 (77.4%)	134 (88.7%)	120 (100%)	<0.01[1,2]
Total episodes of prior bleeding, n (%)	268	243	352	NA
IRR	1.1	1.61	2.93	<0.01[1,2]

Notes: statistically significant or nonsignificant difference relative: 1 – to NSBB group; 2 – to EIS group; MELD – Model for End-Stage Liver Disease; IRR – Incidence Rate Ratio.

Results of the study and discussion

The treatment groups demonstrated a high degree of homogeneity regarding baseline demographic and clinical parameters (Tables 1, 2)

Significant heterogeneity was observed in parameters related to PH severity. The PSE group exhibited markedly different baseline profiles compared to the NSBB and EIS cohorts, specifically regarding MELD (Model for End-Stage Liver Disease) scores (p=0.004), hemoglobin levels (p<0.001), platelet counts (p=0.022), and spleen size (p<0.001). While the incidence of portal vein patency was similar across all groups, the pre-treatment bleeding burden, expressed as IRR, was notably higher in the PSE group (2.93) compared to the NSBB group (1.10).

There were no statistically significant differences across the NSBB, EIS, and PSE groups in terms of age, sex distribution, or the etiology of liver cirrhosis (viral, alcohol-associated liver disease, or metabolic), indicating a homogeneous population structure regarding the underlying liver disease (p>0.05). Furthermore, hepatic synthetic function, as assessed by Child–Pugh classification, was comparable among the groups.

However, significant heterogeneity was observed in parameters related to PH severity. The PSE group exhibited markedly different baseline profiles compared to the NSBB and EIS cohorts, specifically regarding MELD scores (p=0.004), hemoglobin levels (p<0.001), platelet counts (p=0.022), and spleen size (p<0.001). While the incidence of portal vein patency was similar across all groups, the pre-treatment bleeding burden, expressed as IRR, was notably higher in the PSE group (IRR=2.93) compared to the NSBB group (Table 2).

During the 12-month follow-up period, the clinical trajectories of the three secondary prophylaxis groups diverged substantially, allowing a clear comparison of therapeutic effectiveness (Table 3). Although patients in the PSE cohort presented with the most severe baseline profile, including the highest burden of previous bleeding episodes, pronounced splenomegaly, and more severe hypersplenism, their subsequent clinical course was markedly more favorable.

Overall survival analysis using the Kaplan–Meier method demonstrated a pronounced advantage of PSE over EIS and NSBB. The survival curve in the PSE group declined more gradually, reflecting fewer fatal events and more effective long-term control of PH. Twelve-month overall survival rates were 0.87 for PSE, 0.73 for NSBB, and 0.64 for EIS, with statistically significant differences among the curves (log-rank p<0.001).

This pattern was mirrored in the analysis of recurrence-free survival. Patients who underwent PSE maintained the longest interval without rebleeding, whereas

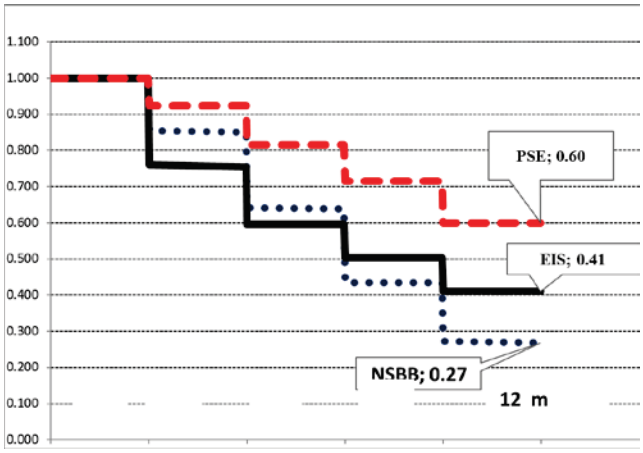

Fig. 6. Recurrence-free survival in the PSE, EIS, and NSBB groups over 12 months

the EIS and NSBB curves exhibited a steep early decline, indicating frequent early recurrences. The probability of remaining free from variceal rebleeding at 12 months was 0.60 in the PSE group, compared with 0.41 and 0.27 in the EIS and NSBB cohorts (Fig. 6), respectively (log-rank p<0.001).

A particularly sensitive indicator of treatment efficacy was the IRR, which reflects the intensity of recurrent bleeding per patient. Despite the initially highest IRR in the PSE group (2.93 at baseline), this cohort achieved the deepest reduction over time, with the IRR decreasing to 0.58 in the first six months and further to 0.39 between months 6–12 – representing a more than 7.5-fold reduction. In comparison, the IRR in the EIS group decreased from 1.61 to 0.56, while NSBB therapy showed the least favorable dynamics, with an initial rise (up to 1.24) followed by a partial decline to 0.68 in the second half of the year.

Between-group comparisons confirmed the superiority of PSE, with IRRs of 1.82 versus EIS and 2.66 versus NSBB (both p<0.001). These findings are consistent with Doppler ultrasound observations demonstrating early stabilization of splanchnic hemodynamics following embolization.

The curve demonstrates substantial differences between treatment strategies. Patients who underwent PSE maintained the highest probability of remaining free from recurrent bleeding (0.60), whereas the corresponding values in the EIS and NSBB groups were 0.41 and 0.27, respectively. The early decline observed in the EIS and NSBB curves reflects the high incidence of early rebleeding episodes, while the prolonged plateau of the PSE curve indicates a stable and durable hemodynamic effect of the intervention. Differences between the curves were statistically significant (log-rank p<0.001).

The frequency of recurrent bleeding episodes also differed markedly across the groups. At least one re-

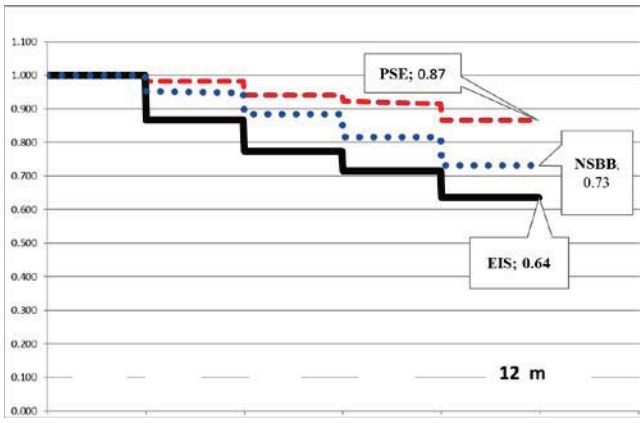

Fig. 7. Twelve-month overall survival in the PSE, EIS, and NSBB groups

bleeding event within 12 months occurred in 43.8% of patients in the PSE group, compared with 65% in the EIS group and 75.7% in the NSBB group. Chi-square analysis confirmed significant differences for all comparisons ($p<0.05$). Importantly, even though the PSE cohort included patients with the most advanced PH, their recurrence rates remained substantially lower than those observed with pharmacological or endoscopic therapy.

Analysis of all-cause mortality further strengthened these observations. Twelve-month mortality was lowest after PSE (13.3%), while the EIS and NSBB groups demonstrated mortality rates of 37.1% and 28.4%, respectively (Fig. 7). The EIS cohort exhibited a higher proportion of non-variceal deaths, including ulcer-related complications, strictures, and septic events, highlighting the potential iatrogenic risks associated with repeated sclerotherapy. Bleeding-related mortality was also lowest in the PSE group (9.1% versus 27.8% and 26.7% for EIS and NSBB).

The PSE cohort demonstrates the highest survival probability (0.87), with a markedly slower decline in the Kaplan-Meier curve compared with NSBB (0.73) and EIS (0.64). These findings indicate superior long-term hemodynamic stability and reduced mortality following PSE (log-rank test, $p<0.001$).

Collectively, these results demonstrate the significant therapeutic advantage of partial splenic artery embolization over both endoscopic injection sclerotherapy and non-selective β-blocker therapy across all clinically meaningful endpoints, including rebleeding frequency, recurrence intensity, recurrence-free survival, and overall survival. Notably, PSE provided the most favorable outcomes despite being reserved for patients with the most severe baseline disease, underscoring its capacity to achieve durable hemodynamic stabilization and long-term clinical benefit in real-world practice.

The present study provides compelling evidence that partial splenic artery embolization (PSE) offers a distinct survival and clinical advantage over standard pharmacological and endoscopic therapies. A notable finding of our investigation is the inverse relationship between baseline severity and clinical outcome: although the PSE cohort comprised patients with the most advanced PH, this group achieved superior long-term hemodynamic stability compared to those treated with NSBB or EIS. This underscores the potential of PSE to effectively interrupt the pathophysiology of variceal bleeding even in the setting of decompensated cirrhosis.

Our observations are consistent with several landmark international studies that have described the physiological and clinical benefits of PSE. The pioneering work of D.G. Spigos et al. demonstrated that partial splenic embolization can reduce portal pressure by 20–30% and significantly decrease the risk of recurrent variceal bleeding [10]. Subsequent investigations, including those by K.G. Koconis et al. and K. Zhu et al., confirmed that PSE not only decompresses the portal venous system but also effectively corrects hypersplenism through improved platelet counts and reduced splenic pooling [8,14]. In their review, K.G. Koconis et al. [8] position PSE as a key tool when standard portal pressure control fails. The correction of cytopenias plays a vital role in reducing bleeding risk, particularly in patients with advanced disease, and this mechanism likely contributed to the favorable outcomes observed in our cohort.

Importantly, our findings support the growing consensus that traditional modalities such as EIS and NSBB are limited by their lack of direct impact on PH. This limitation is well documented in the foundational works of G. Garcia-Tsao et al. and J. Bosch et al., who emphasized that effective secondary prophylaxis requires a sustained reduction of portal pressure, rather than merely a local modification of variceal channels [1,4]. Endoscopic obliteration techniques provide a short-term anatomical benefit but fail to address the underlying hemodynamic drivers of variceal formation, while NSBB therapy may be insufficient in patients with severe hyperdynamic circulation or refractory PH.

In contrast, PSE directly targets the inflow component of PH by reducing splenic arterial perfusion, subsequently decreasing portal venous inflow and improving splanchnic hemodynamics. The magnitude and stability of the IRR reduction observed in our study, particularly the more than seven-fold decrease over 12 months, reflect this physiological mechanism and align with Doppler-based assessments reported in prior literature. Regarding safety, it is important to acknowledge that

post-embolization syndrome (abdominal pain, fever) is a common adverse event following PSE; however, it is transient and medically manageable, standing in contrast to the potentially serious iatrogenic complications associated with repeated EIS.

A noteworthy finding is that despite the unfavorable baseline clinical profile of the PSE group, including the highest IRR and most advanced splenomegaly, these patients ultimately achieved the lowest recurrence rates, the lowest bleeding-related mortality, and the highest overall survival. This observation parallels the conclusions drawn by K. Zhu et al., who reported particularly high efficacy of PSE in patients with pronounced splenomegaly and recurrent bleeding [14].

Our study has certain limitations. First, it was a single-center experience, which may limit the generalizability of the findings. Second, the study design was retrospective and prospective with non-randomized group selection, introducing the potential for selection bias. However, the observed baseline disparities between the treatment groups are not attributable to this bias, but rather reflect the specific clinical indications for each intervention strategy. The PSE cohort represented a patient population with more advanced manifestations of PH, characterized by severe hypersplenism and profound thrombocytopenia. These patients were selected for interventional radiology specifically due to significant hemodynamic compromise or contraindications/failure of standard conservative therapies (NSBB/EIS). Consequently, the lower platelet counts, larger spleen sizes, and higher initial bleeding burden in the PSE group document the appropriate real-world triage of high-risk patients to a more intensive treatment modality.

Overall, the present findings reinforce contemporary international recommendations that advocate for the consideration of PSE in patients with complicated PH, particularly when standard endoscopic or pharmacological strategies fail to achieve sufficient hemodynamic control. Given its ability to modulate both portal pressure and hematologic parameters, PSE represents an effective and physiologically justified modality for secondary prophylaxis, with substantial potential to improve long-term outcomes in high-risk cirrhotic patients.

Conclusions

Partial splenic artery embolization demonstrated superior efficacy compared with NSBB and EIS-based strategies in the secondary prophylaxis of esophageal variceal bleeding. PSE provided the most substantial reductions in rebleeding frequency and intensity, along with the highest overall and recurrence-free survival, even in patients with the most advanced disease at baseline. These findings underscore the ability of PSE to achieve durable hemodynamic improvement and support its integration into treatment algorithms for high-risk patients with complicated PH.

The authors have no conflicts of interest to declare.

Funding. The work was performed in accordance with the plan of research work of the Department of Surgery No 3 of Bogomolets National Medical University: «Innovative algorithms for diagnosis and surgical treatment of acute complications of diseases and neoplastic lesions of the gastrointestinal tract and hepatobiliopancreatic zone». The authors did not receive additional financial support.

References/Література

1. Bosch J, Groszmann RJ, Shah VH. (2015). Evolution in the understanding of the pathophysiological basis of portal hypertension: How changes in paradigm are leading to successful new treatments. J Hepatol. 62; 1 Suppl: S121-S130. doi: 10.1016/j.jhep.2015.01.003.
2. D'Amico G, Garcia-Tsao G, Pagliaro L. (2006). Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. J Hepatol.;44(1):217-231. doi: 10.1016/j.jhep.2005.10.013.
3. De Franchis R, Bosch J, Garcia-Tsao G, Reiberger T, Ripoll C et al. (2022). Expanding consensus in portal hypertension: Report of the Baveno VII consensus workshop: Personalizing care for portal hypertension. J Hepatol. 76(4): 959-974. doi: 10.1016/j.jhep.2021.12.022.
4. Garcia-Tsao G, Abraldes JG, Berzigotti A, Bosch J. (2017). Portal hypertensive bleeding in cirrhosis: Risk stratification, diagnosis, and management: 2016 practice guidance from the American Association for the Study of Liver Diseases. Hepatology. 65(1): 310-335. doi: 10.1002/hep.28906.
5. Graham DY, Smith JL. (1981). The course of patients after variceal hemorrhage. Gastroenterology. 80(4): 800-809.
6. Helaly AZ, Al-Warraky MS, El-Azab GI, Kohla MA, Abdelaal EE. (2015). Portal and splanchnic hemodynamics after partial splenic embolization in cirrhotic patients with hypersplenism. APMIS. 123(12): 1032-1039. doi: 10.1111/apm.12470.
7. Kaplan DE, Ripoll C, Thiele M, Fortune BE, Simonetto DA, Garcia-Tsao G et al. (2024). AASLD Practice Guidance on risk stratification and management of portal hypertension and varices in cirrhosis. Hepatology. 79(5): 1180-1211. doi: 10.1097/HEP.0000000000000647.
8. Koconis KG, Singh H, Soares G. (2007). Partial splenic embolization in the treatment of patients with portal hypertension: a review of the english language literature. J Vasc Interv Radiol. 18(4): 463-481. doi: 10.1016/j.jvir.2006.12.734.
9. Saeki M, Okubo H, Takasaki Y, Nakadera E, Fukuo Y, Fukada H et al. (2023). The Impact of Partial Splenic Embolization on Portal Hypertensive Gastropathy in Cirrhotic Patients with Portal Hypertension. J Clin Med. 12(7): 2662. doi: 10.3390/jcm12072662.
10. Spigos DG, Jonasson O, Mozes M, Capek V. (1979). Partial splenic embolization in the treatment of hypersplenism. AJR Am J Roentgenol. 132(5): 777-782. doi: 10.2214/ajr.132.5.777.
11. Villanueva C, Albillos A, Genescà J, Garcia-Pagan JC, Calleja JL, Aracil C et al. (2019). Beta blockers to prevent decompensation of cirrhosis in patients with clinically significant portal hyperten-

sion (PREDESCI): a randomised, double-blind, placebo-controlled, multicentre trial. Lancet. 393(10181): 1597-1608. doi: 10.1016/S0140-6736(18)31875-0.
12. Yin X, Gu L, Zhang M, Yin Q, Xiao J, Wang Y et al. (2022). Covered TIPS Procedure-Related Major Complications: Incidence, Management and Outcome From a Single Center. Front Med (Lausanne). 9: 834106. doi: 10.3389/fmed.2022.834106.
13. Younossi ZM, de Avila L, Racila A, Nader F, Paik J, Henry L et al. (2025). Prevalence and predictors of cirrhosis and portal hypertension in the United States. Hepatology. 82(5): 1229-1240. doi: 10.1097/HEP.0000000000001243.
14. Zhu K, Meng X, Qian J, Huang M, Li Z, Guan S et al. (2009). Partial splenic embolization for hypersplenism in cirrhosis: a long-term outcome in 62 patients. Dig Liver Dis. 41(6): 411-416. doi: 10.1016/j.dld.2008.10.005.

Відомості про авторів:

Козлов Сергій Миколайович *– к.мед.н., доц. каф. хірургії №3 НМУ ім. О.О. Богомольця. Адреса: м. Київ, бульв. Т. Шевченка, 13. https://orcid.org/0000-0002-2359-8581.*

Косей Наталія Василівна *– д.мед.н., проф., зав. відділу репродуктивного здоров'я ДНУ «Центр інноваційних медичних технологій НАН України». Адреса: м. Київ, вул. Вознесенський узвіз, 22. https://orcid.org/0000-0002-1035-1694.*

Лещинська Наталія Олексіївна *– лікар-інтерн-хірург НУОЗ України ім. П.Л. Шупика. Адреса: м. Київ, вул. Дорогожицька, 9. https://orcid.org/0000-0002-4036-9713*

Яковенко Надія Олексіївна *– лікар-інтерн-хірург НУОЗ ім. П.Л. Шупика. Адреса: м. Київ, вул. Дорогожицька, 9. https://orcid.org/0009-0005-8067-5060.*

Назаров Ярослав Сергійович *– лікар-хірург КНП «Лікарня №12». Адреса: м. Києв, вул. проф. Підвисоцького. 4А. https://orcid.org/0000-0003-4007-3338*

Козлов Олександр Сергійович *– лікар-інтерн-хірург НУОЗ України ім. П.Л. Шупика. Адреса: м. Київ, вул. Дорогожицька, 9. https://orcid.org/0000-0003-1034-0909.*

Стаття надійшла до редакції 13.09.2025 р., прийнята до друку 12.12.2025 р.