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Short-term hemodynamic effects of splenic blood flow modulation after partial splenic artery embolization for secondary prevention of esophageal variceal bleeding

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Partial splenic artery embolization (PSE) is used in the management of portal hypertension to reduce splenic inflow. However, its hemodynamic impact in the secondary prophylaxis of esophageal variceal bleeding requires additional investigation.

Objective – to assess changes in splenic hemodynamics after PSE for secondary prevention of variceal bleeding.

Materials and methods. The study included 90 patients (mean age 49.5 years) with a history of variceal bleeding and splenomegaly (mean volume 781.6 cm^3). Splenic hemodynamics were evaluated using Doppler ultrasound at baseline and 1 month after PSE. Splenic volume and complications were monitored for up to 12 months.

Results. One month after PSE, splenic artery diameter decreased from 5.77 ± 1.20 to 4.72 ± 1.14 mm ($p < 0.001$). Peak systolic velocity declined (152.92 ± 50.35 to 89.77 ± 34.28 cm/s, $p < 0.001$), and end-diastolic velocity decreased (56.76 ± 21.93 to 38.18 ± 15.59 cm/s, $p < 0.001$). Both resistance (0.63 ± 0.08 to 0.58 ± 0.13, $p < 0.05$) and pulsatility indices (1.07 ± 0.24 to 0.95 ± 0.27, $p < 0.01$) reduced significantly. Splenic volume initially increased to 831.7 cm^3 due to edema but significantly decreased to 504.2 ± 209.8 cm^3 by month 6 ($p < 0.001$), with this reduction sustained through month 12. Post-embolization syndrome was managed conservatively in 99% of cases; one instance of splenic abscess occurred. Conversely, the sclerotherapy comparison group showed increased splenic volume.

Conclusions. PSE induces significant short-term attenuation of splenic arterial inflow and venous outflow, followed by a substantial reduction in splenic volume. It is an effective adjunct for secondary prophylaxis with a predictable safety profile. Future comparative studies using unified hemodynamic protocols are required.

Keywords
portal hypertension, esophageal varices, secondary prophylaxis, partial splenic artery embolization, Doppler ultrasound, splenic vein.

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Partial splenic artery embolization (PSE) induces predictable and clinically significant alterations in splenic and portal hemodynamics, which underlie its therapeutic efficacy in patients with portal hypertension complicated by splenomegaly and hypersplenism [2]. Immediately after embolization, a marked reduction in arterial inflow to the spleen is observed, resulting in a significant decrease in splenic perfusion. Doppler ultrasound assessment typically demonstrates reduced peak systolic velocity (PSV) and mean flow velocity in the splenic artery, accompanied by an increase in the resistive index (RI). These changes reflect an effective mechanical limitation of splenic arterial inflow and redistribution of blood volume within the splanchnic circulation. At the venous level, PSE leads to a progressive decrease in splenic venous outflow, which contributes to a reduction in portal venous inflow. Several studies have reported a subsequent decline in portal vein diameter and flow velocity, indicating partial decompression of the portal system. This hemodynamic unloading is considered one of the principal mechanisms by which PSE reduces portal pressure and lowers the risk of variceal bleeding. The extent

of these hemodynamic changes correlates with the embolized splenic volume. Patients undergoing embolization of 50–70 % of the splenic parenchyma exhibit a more pronounced reduction in splenic artery flow and portal venous inflow compared to those with limited embolization, while maintaining an acceptable safety profile [5]. Excessive embolization, however, may result in substantial ischemic changes without providing further hemodynamic benefit.

In the subacute and long-term periods, partial restoration of splenic blood flow is commonly observed due to the development of collateral arterial circulation. Nevertheless, Doppler parameters usually remain significantly lower than baseline values, suggesting sustained modulation rather than complete normalization of splenic hemodynamics. This persistent reduction in splenic inflow explains the durable improvement in hypersplenism parameters, particularly platelet and leukocyte counts.

From a pathophysiological perspective, PSE does not eliminate portal hypertension but instead modifies one of its major contributors–the hyperdynamic splenic circulation. By reducing splenic arterial inflow, PSE decreases splenic sequestration and portal venous inflow, thereby exerting a dual beneficial effect on both systemic hematologic parameters and portal hemodynamics.

Objective – to assess short-term changes in splenic arterial and venous hemodynamics following partial splenic artery embolization performed for secondary prevention of esophageal variceal bleeding.

Materials and methods

Study design and patients

This prospective observational study included 90 patients who underwent PSE as a part of secondary prevention of esophageal variceal bleeding. All patients had a documented history of at least one episode of variceal hemorrhage prior to enrollment, with the number of bleeding episodes ranging from one to seven.

The study cohort consisted of 38 women and 52 men, with a mean age of 49.5 years. Splenomegaly was present in all patients, with baseline splenic volume exceeding 300 cm^3 in every case. The mean baseline splenic volume was 781.6 cm^3, as assessed at the pre-intervention time point (0 months).

Doppler ultrasound assessment

Doppler ultrasound flowmetry was used to assess splenic arterial and venous hemodynamics before intervention and during follow-up. Examinations were performed by experienced operators using standardized protocols. The splenic artery was evaluated at the proximal segment, with measurements including vessel diameter, peak systolic velocity, end-diastolic velocity, time-averaged mean velocity, RI, and pulsatility index (PI). Volumetric blood flow was calculated based on vessel diameter and mean flow velocity (Fig. 1).

The splenic vein was assessed at the hilum or proximal segment, with measurements including vessel diameter, maximal and time-averaged mean flow velocities, and calculated volumetric blood flow.

Baseline Doppler measurements were obtained prior to embolization (0 months), and follow-up assessments were performed at 1 month after the procedure. Additional volumetric and clinical follow-up was conducted up to 12 months post-intervention.

Partial splenic artery embolization technique

Partial splenic artery embolization was performed using a reductional technique under fluoroscopic guidance (Fig. 2, 3). Selective catheterization of the splenic artery was achieved via standard transfemoral access. Embolization was targeted to achieve

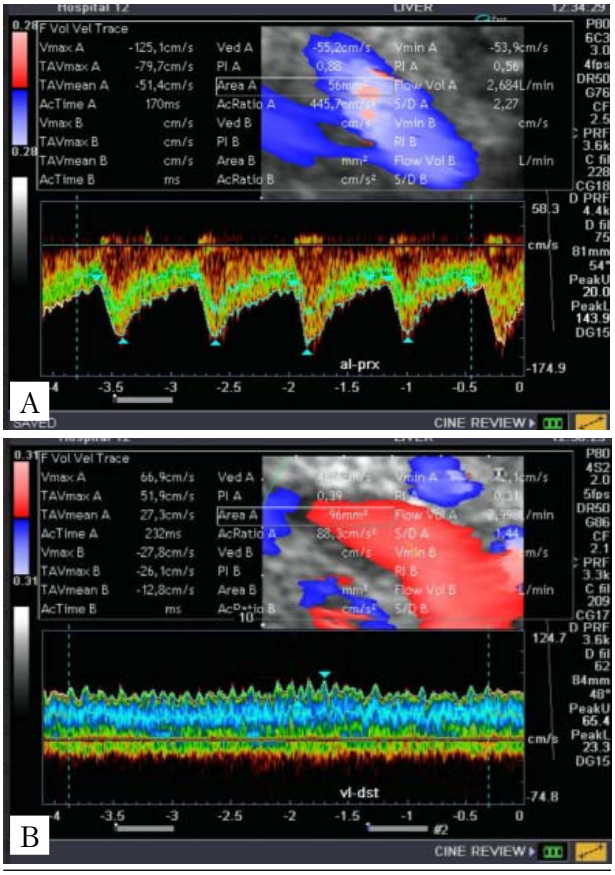

Figure 1. **Doppler ultrasound flowmetry assessment: A. Splenic artery (AL) B. Splenic vein (VL). The image demonstrates the spectral Doppler waveform and the calculation of volumetric blood flow parameters in the splanchnic circulation**

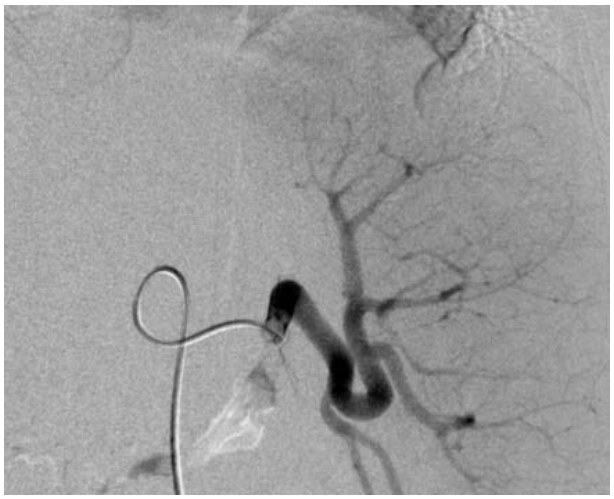

Figure 2. **Selective angiography of the splenic artery. The catheter is positioned in the proximal segment of the vessel prior to embolization**

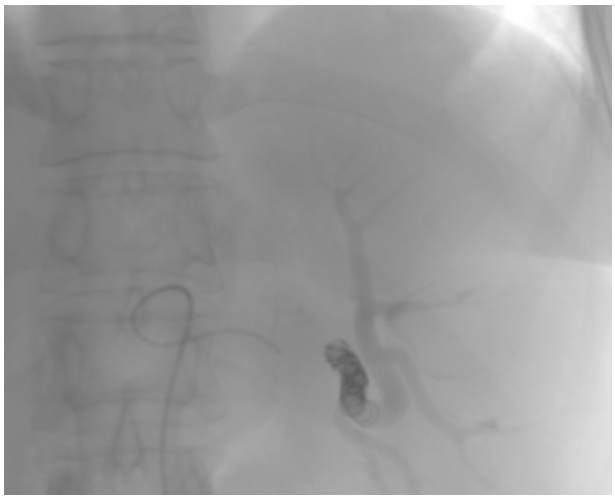

Figure 3. **Angiographic result of partial splenic artery embolization. The image demonstrates the placement of embolization coils in the splenic artery, resulting in reduced distal perfusion**

partial devascularization of the splenic parenchyma while preserving a functioning splenic remnant. The extent of embolization was determined based on angiographic findings and clinical indications.

The extent of embolization was determined based on angiographic findings and clinical indications.

Outcome measures

The primary outcomes of the study were short-term changes in splenic arterial inflow and splenic venous outflow parameters assessed by Doppler ultrasound at 1 month after embolization. Secondary outcomes included changes in splenic volume during follow-up and the incidence of post-embolization syndrome.

Statistical analysis

Continuous variables are presented as mean ± standard deviation. Paired comparisons between baseline and follow-up measurements were performed using appropriate statistical tests. A p-value of < 0.05 was considered statistically significant.

Results

The data presented in Table demonstrate a pronounced hemodynamic effect of reductional PSE on the major vessels of the portal circulation.

At the arterial level, significant changes in splenic artery morphology and flow parameters were observed as early as 30 days after the intervention. Systolic and diastolic splenic artery flow velocities and the effective vessel diameter decreased significantly from baseline values ($p < 0.001$; see Table).

Doppler-derived impedance parameters changed accordingly. Both the PI and RI showed significant reductions, reflecting attenuation of splenic arterial flow intensity following embolization (see Table).

Table. **Changes in splenic artery and vein Doppler parameters after partial splenic artery embolization**

Parameter	Before intervention	1 month after PSE	p
Splenic artery			
Diameter, mm	5.77 ± 1.20	4.72 ± 1.14	< 0.001
Peak systolic velocity, cm/s	152.92 ± 50.35	89.77 ± 34.28	< 0.001
End-diastolic velocity, cm/s	56.76 ± 21.93	38.18 ± 15.59	< 0.001
RI	0.63 ± 0.08	0.58 ± 0.13	< 0.05
PI	1.07 ± 0.24	0.95 ± 0.27	< 0.01
Splenic vein			
Diameter, mm	9.09 ± 2.57	7.15 ± 2.30	< 0.001
Maximal flow velocity, cm/s	34.08 ± 15.04	21.53 ± 7.87	< 0.001

The reduction in arterial inflow to the spleen was followed by a corresponding decrease in venous outflow. As shown in Table, splenic vein hemodynamic parameters demonstrated a statistically significant ($p < 0.001$) and sustained decline throughout the postoperative observation period.

Maximal splenic vein flow velocity decreased from 34.08 cm/s to 21.53 cm/s (–37 %), while time-averaged mean velocity declined from 27.96 cm/s to 17.62 cm/s (–37 %). Simultaneously, a reduction in splenic vein diameter was observed, resulting in a marked decrease in calculated splenic venous volumetric flow from 1.19 L/min to 0.39 L/min (–67.2 %).

For comparison, in the endoscopic sclerotherapy group, in which ultrasound examination was performed using a limited protocol without Doppler flowmetry for vascular velocity assessment, an increase in splenic vein diameter was observed at 1 month after the procedure.

At 1-month follow-up, splenic vein diameter after PSE (7.15 ± 2.30 mm) was numerically smaller than that observed after endoscopic sclerotherapy (8.63 ± 2.79 mm and 9.88 ± 1.45 mm).

As an integral consequence of reduced splenic arterial inflow and venous outflow, dynamic changes in splenic volume and the functioning parenchymal fraction were observed. Splenic volume increased from 781.6 cm^3 prior to embolization to 831.7 cm^3 at 1 month of follow-up. Subsequently, a consistent downward trend was noted up to the 12-month control point, with a statistically significant reduction observed from the 6-month follow-up onward ($p < 0.001$). The transient early increase in splenic volume was attributed to parenchymal edema associated with embolization-induced infarctions and the infarcted areas themselves. During further follow-up, infarct evolution and replacement by fibrous tissue led to a gradual reduction in splenic volume, typically observed between 6 and 12 months after the procedure.

Clinically, splenic infarctions were accompanied by transient fever ranging from 37.5°C to 38.7°C and left upper quadrant pain radiating to the left clavicle, shoulder, or scapular region. These manifestations were interpreted as post-embolization syndrome (PES). In 99 % of cases, PES was successfully managed with nonsteroidal anti-inflammatory drugs in combination with antibiotic prophylaxis administered for 7–12 days. The occurrence of splenic infarctions and PES was not regarded as a postoperative complication, as exclusion of a portion of the functioning splenic parenchyma represents the intended mechanism of PSE. Inadequate antibiotic prophylaxis was associated with secondary infection of splenic infarcts and abscess formation in one patient.

Discussion

In patients treated with endoscopic sclerotherapy, an increase in splenic volume was observed during short-term follow-up without evidence of splenic infarction. Mean splenic volume increased from 724.2 ± 242.6 cm^3 at baseline to 750.8 ± 165.1 cm^3 at 1 month after the procedure. This pattern differed from that observed after PSE.

The observed increase in splenic volume after sclerotherapy may reflect the progression of unfavorable portal hemodynamic changes associated with impaired venous outflow. However, given that ultrasound assessment in the sclerotherapy group was performed using a limited protocol without Doppler flowmetry, this observation should be interpreted with caution. This limitation highlights the need for further studies using a unified hemodynamic assessment protocol across patient groups managed with different secondary prophylaxis strategies.

Although direct studies evaluating splenic hemodynamic changes after endoscopic sclerotherapy are scarce, the relationship between portal hypertension, venous congestion, and splenomegaly is well established in the literature [1, 2, 5, 11]. Endoscopic sclerotherapy effectively controls variceal bleeding at the local level but does not directly modify splanchnic inflow or splenic venous return. As a result, portal venous congestion and splenic enlargement may persist or progress, depending on baseline liver function and collateral circulation patterns [3, 7].

In contrast, PSE has been shown to reduce splenic arterial inflow and splenic venous return, thereby modulating portal hemodynamics [1, 4, 6]. In our cohort, early Doppler flowmetry demonstrated a pronounced reduction in splenic artery velocities and effective vessel caliber at 1 month after PSE. This arterial inflow reduction was paralleled by a concordant decline in splenic venous velocity and diameter, supporting a mechanistic chain whereby reduced splenic arterial inflow leads to decreased splenic venous return and, consequently, a lower portal inflow burden.

From a pathophysiological standpoint, PSE should not be viewed as eliminating portal hypertension but rather as targeting one of its major modifiable components–excessive splanchnic (splenic) inflow–with a plausible downstream effect on portal venous loading. Because direct portal pressure measurements (e.g., hepatic venous pressure gradient) were not performed in the present study, any inference regarding portal pressure reduction should be regarded as hypothesis-generating and requires confirmation in future studies employing standardized pressure-based endpoints [2, 5, 8].

A clinically relevant observation in our study was the dynamic volumetric response of the spleen

after PSE. An initial increase in splenic volume at 1 month was followed by a sustained downward trend with a significant reduction at later follow-up intervals. This pattern is consistent with post-embolization parenchymal edema and infarction evolution, followed by fibrosis and contraction of non-functioning splenic tissue. It supports the concept that the therapeutic effect of PSE is achieved through controlled reduction of functional splenic parenchyma and redistribution of splenic inflow rather than immediate organ shrinkage.

Importantly, the concept of splenic inflow reduction for portal decompression is not limited to endovascular techniques. Tutchenko and colleagues described laparoscopic and laparoscopic-assisted surgical approaches aimed at reducing portal inflow, including clipping or ligation of the splenic artery, often combined with interventions on left gastric vessels, in patients with portal hypertension complicated by variceal bleeding [10]. These authors interpreted splenic artery clipping or ligation as producing a direct, moderate portodecompressive effect by decreasing arterial inflow to the spleen and reducing venous runoff toward the portal vein. More recently, Tutchenko et al. compared splenectomy and selective splenic artery ligation during porto-azygos disconnection and emphasized that splenic flow-reduction strategies are associated with measurable changes in portal system parameters, while also carrying distinct risk profiles for thrombosis, infection, and infarction [9].

Taken together, our short-term Doppler findings support the concept that PSE induces an early and coordinated reduction in both splenic arterial inflow and splenic venous return, changes that are mechanistically consistent with a dicrease in portal inflow burden. Comparison with endoscopic sclerotherapy suggests fundamentally different hemodynamic consequences of these treatment strategies. However, definitive conclusions regarding their relative impact on portal pressure and long-term outcomes require prospective, head-to-head studies with harmonized imaging protocols and pressure-based validation.

Conclusions

Partial splenic artery embolization performed for secondary prevention of esophageal variceal bleeding is associated with significant short-term attenuation of splenic arterial inflow, as evidenced by reductions in splenic artery diameter, systolic and diastolic flow velocities, and Doppler impedance indices.

The reduction of splenic arterial inflow after embolization is accompanied by a concordant decrease in splenic venous outflow, including significant reductions in splenic vein diameter and flow velocity, indicating decreased venous return from the spleen.

These coordinated arterial and venous hemodynamic changes suggest that PSE modulates splenic contribution to portal circulation by reducing portal inflow load. However, direct portal pressure measurements were not performed, and the effect on portal pressure requires further validation.

Splenic volume demonstrates a characteristic short-term increase following embolization, followed by a sustained downward trend with significant reduction during mid-term follow-up, reflecting post-embolization parenchymal changes and controlled reduction of functional splenic tissue.

In contrast, endoscopic sclerotherapy is associated with an increase in splenic volume and splenic vein diameter during short-term follow-up, highlighting fundamentally different hemodynamic consequences compared with splenic inflow–modulating strategies.

The findings support PSE as a hemodynamically active intervention that extends beyond local variceal control; however, comparative assessment of secondary prophylaxis strategies requires future prospective studies using unified hemodynamic assessment protocols and pressure-based validation.

Declaration of interests

The authors have no conflicts of interest to declare.

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Ethical considerations

The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all patients prior to inclusion.

Ethics approval and written, informed consent statements

The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all patients prior to inclusion.

The assessment and use of all clinical data were approved and permitted by the ethics committee of Bogomolets National Medical University before the study. 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.

Authors contributions

S. M. Kozlov: work concept and design, data collection and analysis, responsibility for statistical analysis, and writing the manuscript.

I. V. Kolosovych: work concept and design, critical review, and final approval of the manuscript.

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Безпосередні гемодинамічні результати корекції селезінкового кровоплину після емболізації селезінкової артерії при вторинній профілактиці кровотеч із варикозно розширених вен стравоходу

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Парціальну емболізацію селезінкової артерії (ПЕСА) використовують при портальній гіпертензії для зменшення селезінкового артеріального притоку. Її гемодинамічний вплив у контексті вторинної профілактики кровотеч із варикозно розширених вен стравоходу потребує уточнення.

Мета — оцінити зміни селезінкової гемодинаміки після ПЕСА, виконаної для вторинної профілактики варикозних кровотеч.

Матеріали та методи. У дослідження було залучено 90 пацієнтів (середній вік – 49,5 року) з анамнезом варикозних кровотеч і спленомегалією (середній об'єм – 781,6 см^3). Селезінкову гемодинаміку оцінювали за допомогою допплерівського ультразвукового дослідження (УЗД) до ПЕСА та через 1 місяць. Моніторинг об'єму селезінки та ускладнень проводили протягом 12 міс.

Результати. Через 1 міс після ПЕСА діаметр селезінкової артерії зменшився з (5,77 ± 1,20) до (4,72 ± 1,14) мм ($p < 0,001$). Пікова систолічна швидкість знизилася з (152,92 ± 50,35) до (89,77 ± 34,28) см/с ($p < 0,001$), кінцева діастолічна швидкість – з (56,76 ± 21,93) до (38,18 ± 15,59) см/с ($p < 0,001$), індекс резистентності – з 0,63 ± 0,08 до 0,58 ± 0,13 ($p < 0,05$), індекс пульсації – з 1,07 ± 0,24 до 0,95 ± 0,27 ($p < 0,01$). Об'єм селезінки спочатку збільшився до 831,7 см^3 (набряк), але до 6-го місяця статистично значущо зменшився до (504,2 ± 209,8) см^3 ($p < 0,001$) зі збереженням ефекту до 12-го місяця. Постемболізаційний синдром лікували консервативно в 99 % випадків. Зареєстровано один випадок абсцесу селезінки. У групі порівняння (склеротерапія) відзначено збільшення об'єму селезінки.

Висновки. Парціальна емболізація селезінкової артерії сприяє значному короткостроковому зниженню селезінкового артеріального притоку та венозного відтоку з подальшим суттєвим зменшенням об'єму органа. Це ефективний допоміжний метод вторинної профілактики з прогнозованим профілем безпечності. Необхідно провести порівняльні дослідження з уніфікованими гемодинамічними протоколами.

Ключові слова: портальна гіпертензія, варикозні вени стравоходу, вторинна профілактика, парціальна емболізація селезінкової артерії, допплерографія, селезінкова вена.

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