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Assessment of splenic vascular hemodynamics in patients with established portal hypertension and splenomegaly

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The development of portal hypertension in liver cirrhosis is accompanied by fundamental changes in splanchnic circulation, where the role of the spleen transforms from a passive venous reservoir into an active driver of hemodynamic disturbances. In the present study, based on a comprehensive clinical and instrumental examination of 90 individuals, including 47 patients with liver cirrhosis and established splenomegaly (study group) and 43 practically healthy volunteers (control group), Doppler ultrasonography demonstrated the presence of a pronounced hyperdynamic syndrome in the splenic artery basin. Flowmetry results demonstrate a statistically significant increase in linear blood flow velocities in patients with pathology: Peak Systolic Velocity (PSV) reached 150.8 ± 43.6 cm/s, which is 1.8 times higher than in the control group (85.4 ± 26.7 cm/s), and End-Diastolic Velocity (EDV) nearly doubled to 59.9 ± 19.9 cm/s vs 32.3 ± 10.2 cm/s in healthy individuals. Paradoxically, against the background of a multiple increase in volumetric blood flow, the calculated Resistive Index (RI) in the study group remained stable and did not differ from normal values (0.61 ± 0.08 vs 0.60 ± 0.10), indicating profound vascular wall remodeling and the capacity of the vascular bed to accommodate excessive blood volumes without increasing peripheral resistance. As a consequence of arterial hyperperfusion, a significant increase in maximum velocity in the splenic vein was recorded (34.1 ± 15.6 cm/s vs 23.5 ± 6.2 cm/s in controls). The established strong Spearman correlation between the morphometric volume of the spleen and the velocity of arterial inflow (ρ = 0.616;) confirms the formation of a sustained hyperdynamic type of circulation as a potential mechanism for the development and progression of splenomegaly, and indicates the unconditional predominance of the active splenic component in the pathogenesis of portal hypertension.
Key words: portal hypertension; splenomegaly; Doppler ultrasonography; splenic artery; hemodynamics; liver cirrhosis.

INTRODUCTION

Portal hypertension (PH) remains one of the most critical complications of chronic diffuse liver diseases, significantly affecting patient prognosis and quality of life [1]. Traditionally, the pathophysiology of PH was interpreted through the mechanical theory, which posited that the primary factor was increased intrahepatic vascular resistance due to parenchymal fibrosis and sinusoidal architectural disruption [2, 3].

However, evidence from fundamental physiology and clinical hepatology accumulated over recent decades convincingly demonstrates that mechanical outflow obstruction serves merely as an initiating trigger. The maintenance and progression of hypertension are driven by complex hemodynamic alterations within the splanchnic circulation [4, 5]. While the resistance component (*R*) dominates in early disease stages, established cirrhosis is characterized by hyperdynamic circulatory syndrome - a pathological increase in blood inflow to abdominal organs [6, 7].

This condition involves systemic vasodilation, increased cardiac output, and decreased peripheral vascular resistance, creating a “vicious circle”: compensatory mechanisms intended to counteract portal stasis via increased inflow

paradoxically further elevate portal pressure. In the context of hyperdynamic circulation, the role of the splenic vascular bed warrants particular attention. Splenomegaly was long regarded solely as a passive consequence of venous congestion (congestive splenomegaly). However, clinical and instrumental data suggest the spleen acts not merely as a blood reservoir but as an active contributor to increased portal blood flow [8, 9]. Despite progress in understanding these processes, the correlation between splenic morphometric parameters (volume) and quantitative hemodynamic indicators in the portal basin remains insufficiently elucidated. Specifically, the contribution of splenic arterial inflow to total portal pressure in patients with critical splenomegaly requires clarification. Understanding these patterns is essential for justifying reductive interventions (e.g., splenic artery embolization) as pathogenetically sound approaches to PH management.

METHODS

Study Design and Population. The work was carried out during 2020-2025 at the Department of Surgery with the Abdominal Surgery Course of the Bogomolets National Medical University and approved by the Commission on Bioethical Expertise and Ethics of Scientific Research (Decision No. 131, Date Apr 29, 2020). A comprehensive clinical and instrumental examination of 90 subjects was conducted. Participants were divided into two groups based on the study objectives and the presence of PH signs. The main group comprised 47 patients with a verified diagnosis of liver cirrhosis complicated by PH syndrome and clinically significant splenomegaly. Inclusion criteria were: presence of clinical, laboratory, and instrumental signs of PH; increased spleen volume; and dilation of portal vessels. The comparison (control) group consisted of 43 practically healthy volunteers without history or clinical signs of hepatobiliary disease, matched for age and sex with the study group. All patients provided informed consent in accordance with the Declaration of Helsinki.

There were 42 (46.7%) men (18 (38.3%) in the main group and 24 (55.8%) in the comparison group) and 48 (53.3%) women (29 (61.7%) in the main group and 19 (44.2%) in the comparison group). The average height of the patients was 170.98 ± 8.6 cm (171.28 ± 8.77 cm in the main group and 170.67 ± 8.41 cm in the comparison group), the average weight was 70.5 ± 11.6 kg (74,3 ± 12,5 kg in the main group and 67,4 ± 12,2 kg in the comparison group), the average body mass index (BMI) was 24.87 ± 4,1 kg/m² (25.52 ±3 ,5 kg/m² in the main group and 24.22 ± 4,5 kg/m² in the comparison group). The spleen volume was 905,6 ± 150,4 cm³ in the main group and 136.5 ± 35,5 24 cm³ in the comparison group. Groups were statistically homogeneous regarding age (P > 0.05) and sex (P = 0.151). The study group exhibited significantly higher weight and BMI (P < 0.05), and a critical increase in spleen volume (6.6-fold vs. control; P < 0.001), confirming established splenomegaly. Ultrasound (US) and Doppler flowmetry were performed using a Toshiba Nemio system with a 3.5-5.0 MHz convex probe. Examinations were conducted after fasting. Morphometric parameters (length, thickness, volume) were measured.

Instrumental methods. Ultrasound examination (US) and Doppler were performed on a Toshiba Nemio system with a convex sensor of 3.5-5.0 MHz. Examinations were performed on an empty stomach. Morphometric parameters (length, thickness, volume) were measured. Hemodynamic parameters of the splenic artery (Peak Systolic Velocity (PSV), End-Diastolic Velocity (EDV), Resistive Index (RI), Pulsatility Index (PI) and splenic vein (Maximum Velocity (Vmax) were measured using Pulsed-Wave (PW) Doppler and Color Flow Mapping (CFM) with beam angle correction <60° (Fig. 1).

Statistical analysis. Obtained data were analyzed using EZR (Easy R) software (Saitama Medical Center, Jichi Medical University, Japan). Normality was tested using the Shapiro-Wilk test. Normally distributed data are presented as M ± SD (Student's t-test); non-normally

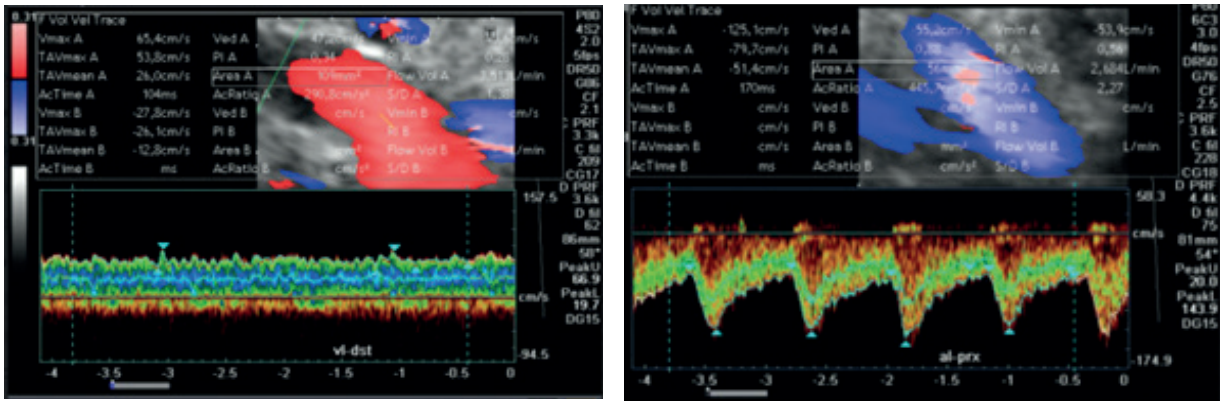

Fig. 1. Methodology of Doppler hemodynamic assessment of splenic vessels: A – assessment of venous blood flow in the splenic vein (measurement of maximum velocity Vmax); B – spectral Doppler of the splenic artery (measurement of PSV and EDV, calculation of RI and PI indices)

distributed data as Me (IQR) (Mann-Whitney U-test). Categorical variables were compared using Pearson's χ^2 test. Spearman's rank correlation coefficient (ρ) was used to assess relationships between variables. A $P < 0.05$ was considered statistically significant.

RESULTS

Doppler assessment revealed significant hemodynamic remodeling in the splenic artery basin in patients with clinically manifest PH, indicating a pronounced hyperdynamic circulation (Table).

Velocity analysis (Fig. 2) demonstrated a significant increase ($P < 0.001$) in linear blood flow velocity in both cardiac cycle phases in the study group.

PSV in the study group was 1.8 times higher than in controls (150.8 cm/s vs 85.4 cm/s), and EDV nearly doubled (59.9 cm/s vs 32.3 cm/s). This velocity elevation correlates with the increased volumetric flow required for hypertrophied spleen. Despite marked velocity changes, resistance indices (Fig. 3) showed no significant differences between groups ($P > 0.05$).

RI in the study group (0.61 ± 0.08) was comparable to controls (0.60 ± 0.10), as was PI. The analysis of hemodynamic parameters revealed a notable difference in the maximum blood flow velocity in the splenic vein (V_max v. lienalis) between the groups. The Study group demonstrated a higher median velocity (34.10 cm/s) compared to the comparison group (23.54 cm/s). The absence of significant differences in RI and PI despite sharply increased PSV and EDV suggests the splenic vascular bed adapts to increased load by proportionally increasing systolic and diastolic flow while maintaining normal vascular tone.

Comparative characteristics of splenic artery hemodynamic parameters (cm/s)

Parameter	Comparison group (n = 43)	Main group (n = 47)
Splenic artery peak systolic velocity	85.43 ± 26.72	150.84 ± 43.57*
Splenic artery end-diastolic velocity	32.26 ± 10.23	59.88 ± 19.96*
Splenic vein maximum velocity	23.54 ± 6.16	34.10 ± 15.55*

Notes: *P < 0,001 by Mann-Whitney U-test.

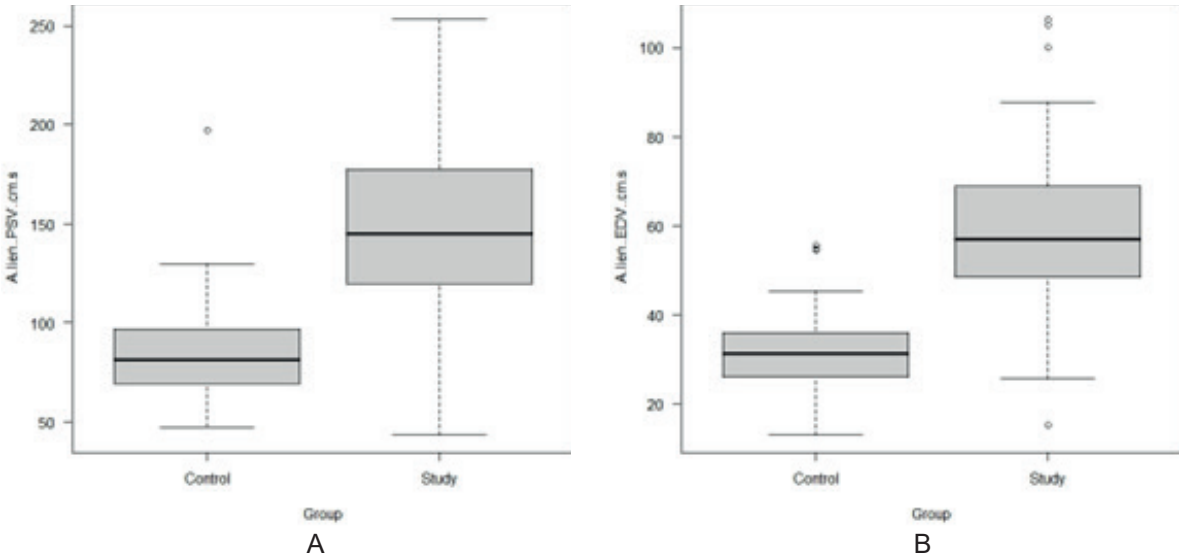

Fig. 2. Comparative characteristics of blood flow velocity parameters in the splenic artery in study groups: A - Peak Systolic Velocity (PSV); B - End-Diastolic Velocity (EDV)

DISCUSSION

Our findings confirm and expand current concepts of PH pathophysiology, emphasizing the importance of splanchnic hyperdynamic syndrome alongside the "hepatic block." The significant increase in splenic artery PSV correlates with Bolognesi et al. [6], who identified celiac trunk vasodilation as a key mechanism maintaining high portal pressure. This "splanchnic steal" phenomenon redirects a substantial portion of cardiac output to abdominal organs, overloading the venous system. Notably, RI remained stable despite increased flow. This aligns with Zipprich [8] and Ghanem, Moustafa [9], who attribute low vascular resistance in cirrhosis

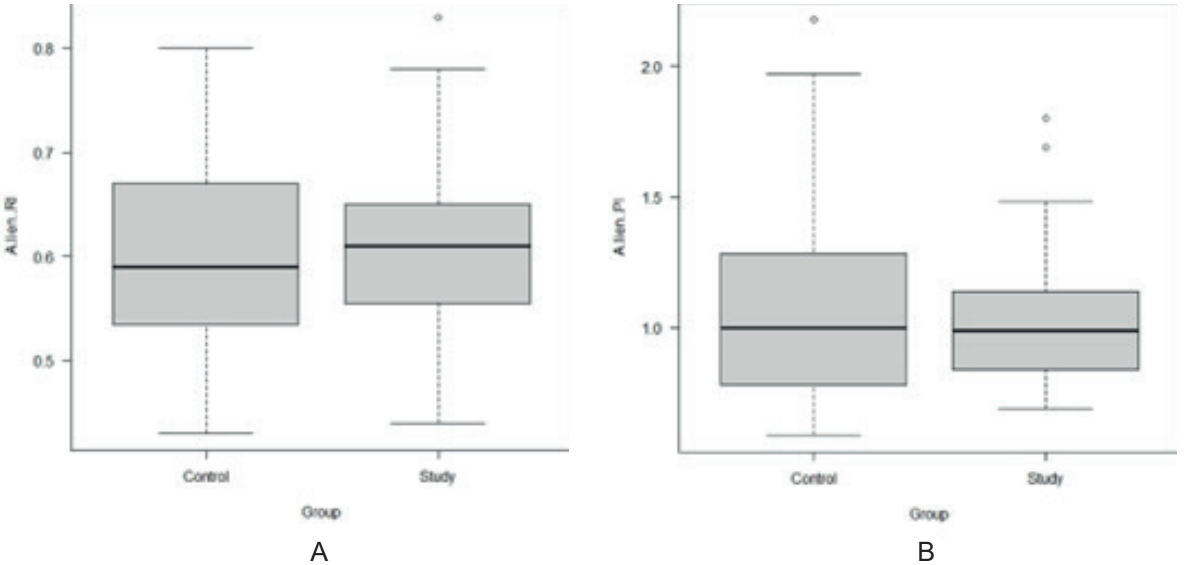

Fig. 3. Comparative characteristics of splenic artery vascular resistance indices in study groups: A - Resistive Index (RI); B - Pulsatility Index (PI)

to excessive endogenous vasodilator production (nitric oxide, glucagon). Such resistance "rigidity" amidst high flow indicates profound vascular remodeling. The correlation between spleen volume and both arterial velocities and venous outflow is shown in Fig. 4.

This confirms that splenomegaly is not a passive outcome of venous stasis but involves active recruitment of additional arterial volume. A "vicious circle" is established: hyperperfusion stimulates angiogenesis and hyperplasia, further increasing spleen volume and venous return, thereby overloading the portal system. According to literature, while intrahepatic shunts maintain portal flow in cirrhosis, effective portal perfusion decreases [9]. PH progression involves splenic artery remodeling – increased diameter and flow velocities. This "flow hypersplenism" causes the splenic vein to drain excessive blood volume into the portal system, which the compromised liver cannot accommodate [10]. The splenic artery functions as a high-output pump, while the splenic vein becomes a high-pressure conduit, obstructing mesenteric flow and promoting variceal formation. This supports the "active splenic component" theory [11]. These data underscore that splenomegaly is an active generator of increased portal pressure. Further research into splanchnic hemodynamics is promising for defining indications for reductive interventions (e.g., splenic artery embolization, Transjugular Intrahepatic Portosystemic Shunt (TIPS) in patients with hyperdynamic circulation [12].

CONCLUSIONS

1.In patients with liver cirrhosis and established portal hypertension, splenomegaly is accompanied by the development of a hyperdynamic splanchnic circulation.

2. This is evidenced by a significant 1.8-fold increase in splenic artery peak systolic velocity and a near doubling of end-diastolic velocity compared to healthy controls.

3. The identified hemodynamic disturbances–excessive splenic inflow with limited portal

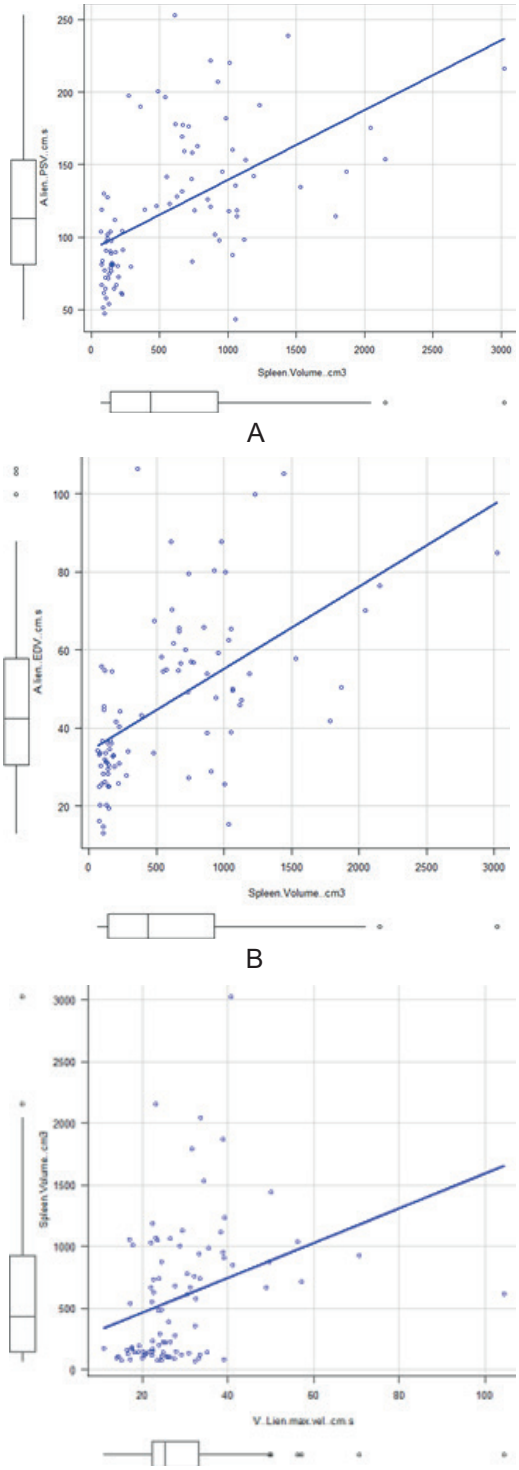

Fig. 4. Correlation between spleen volume and peak systolic (A), end-diastolic velocity (B) (in both cases Spearman P = 0.616, P < 0.001) and maximum velocity in the splenic vein (C) (Spearman P = 0.402; P < 0.001)

outflow – constitute a key pathophysiological mechanism driving the hyperkinetic circulation and portal hypertension.

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ОЦІНКА ГЕМОДИНАМІКИ СУДИН СЕЛЕЗІНКИ У ПАЦІЄНТІВ ЗІ СФОРМОВАНОЮ ПОРТАЛЬНОЮ ГІПЕРТЕНЗІЄЮ ТА СПЛЕНОМЕГАЛІЄЮ

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Розвиток портальної гіпертензії при цирозі печінки супроводжується фундаментальними змінами спланхнічного кровообігу, де роль селезінки трансформується з пасивного венозного резервуара в активний чинник гемодинамічних порушень. У представленому дослідженні на основі комплексного клініко-інструментального обстеження 90 осіб, яке включало 47 пацієнтів із цирозом печінки та сформованою спленомегалією (основна група) та 43 практично здорових добровольців (група контролю), за допомогою ультразвукової допплерографії доведено наявність вираженого гіпердинамічного синдрому в басейні селезінкової артерії. Результати флоуметрії демонструють статистично значуще зростання лінійних швидкостей кровотоку у пацієнтів із патологією: пікова систолічна швидкість (PSV)у 1,8 раза перевищувала показники контрольної групи (85,4 ± 26,7 см/с), а кінцева діастолічна швидкість (EDV) зросла майже вдвічі .Парадоксальним чином, на тлі кратного зростання об›ємного кровотоку, розрахунковий індекс резистентності (RI) в основній групі залишався стабільним і не відрізнявся від норми (0,61 ± 0,08 щодо 0,60 ± 0,10). Це свідчить про глибоке ремоделювання судинної стінки та здатність судинного русла пропускати надмірні об'єми крові без підвищення периферичного опору. Як наслідок артеріальної гіперперфузії, зафіксовано достовірне збільшення максимальної швидкості у селезінковій вені (34,1 ± 15,6 см/с щодо 23,5 ± 6,2 см/с у контролі). Встановлений сильний кореляційний зв'язок за коефіцієнтом Спірмена між морфометричним об'ємом селезінки та швидкістю артеріального притоку (ρ = 0,616) підтверджує формування стійкого гіпердинамічного типу кровообігу як потенційного механізму розвитку та прогресування спленомегалії, а також свідчить про безумовне переважання активного селезінкового компоненту в патогенезі портальної гіпертензії.

Ключові слова: портальна гіпертензія; спленомегалія; доплерографія; селезінкова артерія; гемодинаміка; цироз печінки.

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