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M.I. Tutchenko[1], D.M. Patrakh[1], D.V. Rudyk[1], M.S. Besedinskyi[1], A.V. Lovin[1], I.V. Klyuzko[2]

[1]Bogomolets National Medical University, Kyiv, Ukraine
[2]Vyshhorod Central District Hospital, Vyshhorod, Kyiv region, Ukraine

Mutually aggravating complications in the development of hepatorenal syndrome in decompensated portal hypertension

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Abstract. ***Background.*** *Hepatorenal syndrome (HRS) developing in the setting of decompensated portal hypertension (PH) is characterized by progressive renal failure, elevated serum creatinine levels, and worsening oliguria. HRS occurs in approximately one-third of patients with Child-Pugh class C liver cirrhosis. The purpose was to optimize early diagnosis and treatment, as well as to identify risk factors for the development of HRS and associated mortality.* ***Materials and methods.*** *A total of 126 medical records of patients with decompensated PH treated at the Kyiv City Emergency Hospital and the Department of Surgery, Dental Faculty, Bogomolets National Medical University, between 2022 and 2024 were analyzed. HRS was diagnosed in 51 (40.5 %) cases. Diagnostic criteria included an increase in serum creatinine > 150 μmol/L (1.69 mg/dL) or ≥ 26.5 μmol/L (0.3 mg/dL) within 48 hours in the presence of oliguria and absence of other signs of intrinsic renal injury, particularly hematuria. Patients were divided into two groups. The first group included 24 (47.1 %) patients in whom HRS and community-acquired spontaneous bacterial peritonitis (SBP) were diagnosed within the first 24 hours, and a restrictive transfusion strategy was applied. The second group comprised 27 (52.9 %) participants in whom HRS and nosocomial SBP were diagnosed on days 3–5 of hospitalization, and a liberal transfusion strategy was used. The impact of transfusion strategy, ascites severity, and SBP as mutually aggravating factors in HRS development was assessed.* ***Results.*** *Analysis showed that the combination of large-volume ascites, SBP, and a liberal transfusion strategy forms an unfavorable prognostic complex associated with a significantly increased risk of mortality. HRS development was more frequently associated with large-volume ascites (70.8 %), nosocomial SBP (37.0 %), and a liberal transfusion strategy (52.9 %). Among 126 patients, 27 (21.4 %) underwent surgery. In the postoperative period, HRS developed in 1 (4.2 %) patient in the first group and in 2 (7.4 %) patients in the second group. Overall mortality was 74.5 % (n = 38), with 14 deaths (58.3 %) in the first group and 24 (88.9 %) in the second group (p = 0.03). According to ROC analysis, the model demonstrated high predictive performance for mortality (AUC = 0.909; 95% CI 0.831–0.987), confirming strong discriminative ability for risk stratification in patients with HRS.* ***Conclusions.*** *HRS is a severe complication of decompensated PH associated with high mortality. A liberal transfusion strategy, severe ascites, and SBP increase the risk of its development and progression. Early monitoring of urine output and serum creatinine levels is critical for timely diagnosis, prevention, and management of HRS.*

Keywords: *hepatorenal syndrome; liver cirrhosis; portal hypertension; albumin; variceal bleeding; creatinine; diuresis; spontaneous bacterial peritonitis*

Introduction

Decompensated portal hypertension (PH) represents the terminal stage in the progression of liver cirrhosis (LC), as well as non-cirrhotic forms, including presinusoidal and sinistral PH; among their complications, hepatorenal syndrome (HRS) plays a central role [1, 2]. The pathogenesis of HRS remains incompletely understood [3]. This syndrome is regarded as a functional renal failure developing in profound disturbances of systemic and splanchnic hemodynamics [4, 5]. Its pathogenesis is associated with blood redistribution

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Для кореспонденції: Патрах Денис Михайлович, аспірант, Національний медичний університет імені О.О. Богомольця, бульвар Тараса Шевченка, 13, Київ, 01601, Україна; e-mail: dr.patrakh.denys@gmail.com
For correspondence: Denys Patrakh, PhD student, Bogomolets National Medical University, Taras Shevchenko blvd, 13, Kyiv, 01601, Ukraine; e-mail: dr.patrakh.denys@gmail.com
Full list of authors information is available at the end of the article.

within the splanchnic vascular bed, reduction of the effective arterial blood volume, activation of vasoconstrictor systems (the renin-angiotensin-aldosterone and sympathetic nervous systems), and renal vasoconstriction with a subsequent decrease in glomerular filtration rate [6]. Despite the absence of primary structural kidney damage, progression of HRS without liver transplantation is associated with high mortality rates [7, 8].

The current concept of hepatorenal syndrome-acute kidney injury defines the diagnosis according to the criteria of an increase in serum creatinine ≥ 26.5 μmol/L (0.3 mg/dL) within 48 hours or ≥ 50 % from baseline within the preceding 3 months, in the absence of hypovolemia and other causes of acute kidney injury [9]. Early verification enables timely initiation of vasoconstrictor therapy and albumin infusion aimed at stabilization of renal perfusion [10].

Acute bleeding from esophageal and/or gastric varices is one of the leading triggers of systemic hemodynamic deterioration and the development of renal dysfunction [11]. Excessive infusion therapy promotes splanchnic vasodilation, renal vasoconstriction, and increases the risk of HRS [12].

An equally significant factor is spontaneous bacterial peritonitis (SBP), which is considered a potent trigger of systemic inflammatory response and circulatory dysfunction [13]. SBP is diagnosed when the neutrophil count exceeds 250 cells per 1 mL of ascitic fluid. Community-acquired and nosocomial SBP are distinguished based on differences in pathogen spectrum and antibiotic resistance profiles [14]. Systemic inflammation, endothelial dysfunction, and reduced effective circulating blood volume contribute to the development of HRS [15]. Delayed paracentesis and antibiotic therapy are associated with increased in-hospital mortality [16].

Thus, variceal bleeding, transfusion strategy, and SBP may constitute a pathophysiological complex that necessitates further investigation and the development of preventive and therapeutic approaches.

Materials and methods

A total of 126 medical records of patients hospitalized between 2022 and 2024 for decompensated PH were retrospectively analyzed at the Kyiv City Clinical Emergency Hospital and the Department of Surgery of the Dental Faculty of Bogomolets National Medical University. Renal dysfunction characterized by biochemical blood abnormalities, including serum creatinine > 150 μmol/L (1.69 mg/dL) or an increase in creatinine ≥ 26.5 μmol/L (0.3 mg/dL) within 48 hours in the presence of concomitant oliguria and absence of renal dysfunction manifested by hematuria, was considered indicative of HRS, which was diagnosed in 51 (40.5 %) patients.

The first group included 24 (47.1 %) patients in whom HRS was diagnosed within the first 24 hours after admission. Hemotransfusion support was administered according to a restrictive strategy, using blood components with maintenance of hemoglobin levels within 70 g/L. Diagnostic paracentesis performed during the first hours of hospitalization in 8 (33.3 %) patients revealed > 250 polymorphonuclear neutrophils per 1 mL of ascitic fluid, indicating community-acquired SBP.

The second group consisted of 27 (52.9 %) patients in whom HRS was diagnosed on days 3–5 of hospitalization. Nosocomial SBP was identified in 10 (37.0 %) cases. Hemotransfusion therapy was performed according to a liberal transfusion strategy targeting a hemoglobin level of 100 g/L.

The gender distribution in the first group was as follows: 8 (33.3 %) women and 16 (66.7 %) men. Their mean age was 54.8 ± 13.9 years. In the second group, there were 8 (29.6 %) women and 19 (70.4 %) men, with a mean age of 53.4 ± 13.2 years. No statistically significant differences between groups were observed regarding age or sex ($p > 0.05$).

Sinusoidal PH was observed in 22 (91.7 %) patients in the first group. Among them, viral LC was diagnosed in 6 (25.0 %) patients, alcoholic or toxic LC in 15 (62.5 %), and autoimmune LC in 1 (4.2 %). Presinusoidal PH was identified in 2 (8.4 %) patients, including portal vein thrombosis in 1 (4.2 %) case and sinistral PH in 1 (4.2 %) case.

In the second group, sinusoidal PH was diagnosed in 24 (88.9 %) patients. Viral LC was identified in 7 (25.9 %) cases, alcoholic LC in 16 (59.3 %), and autoimmune LC in 1 (3.7 %). Portal vein thrombosis causing presinusoidal PH was diagnosed in 2 (7.4 %) patients, whereas sinistral PH was observed in 1 (3.7 %) case.

In the first group, moderate ascites was detected in 7 (29.2 %) patients, whereas severe ascites was present in 17 (70.8 %). In the second group, moderate ascites occurred in 8 (29.6 %) patients and severe ascites in 19 (70.4 %). The presence of ascites, indicating decompensation of PH, constitutes an absolute indication for diagnostic paracentesis. A neutrophil count exceeding 250 cells per 1 mL of ascitic fluid is indicative of SBP and requires initiation of empirical antibiotic therapy, microbiological verification, and assessment of antibiotic susceptibility.

Table 1 presents the clinical and etiological characteristics of patients with HRS in the study groups.

Accumulation of ascitic fluid within the abdominal cavity is associated with increased intra-abdominal pressure (IAP) and the development of abdominal compartment syn-

Table 1. Clinical and etiological characteristics of patients with HRS in the study groups, n (%)

Parameters	Group I (n = 24)	Group II (n = 27)
Ascites:		
— moderate	7 (29.2)	8 (29.6)
— severe	17 (70.8)	19 (70.4)
SBP	8 (33.3)	10 (37.0)
Sinusoidal PH:	22 (91.7)	24 (88.9)
— viral LC	6 (25.0)	7 (25.9)
— alcoholic and toxic LC	15 (62.5)	16 (59.3)
— autoimmune LC	1 (4.2)	1 (3.7)
Presinusoidal PH:	2 (8.4)	3 (11.1)
— portal vein thrombosis	1 (4.2)	2 (7.4)
— sinistral PH	1 (4.2)	1 (3.7)

drome, which, in turn, impairs renal perfusion. In cases of IAP > 10–15 mmHg measured in the supine position via a Foley urinary catheter using the Kron method, therapeutic paracentesis was performed with evacuation of ascitic fluid until IAP decreased to < 7 mmHg. This approach constituted an essential component in reducing the risk of abdominal compartment syndrome and, consequently, HRS development.

Terlipressin was administered to 5 (20.8 %) patients in group I and 6 (22.2 %) patients in group II. In the treatment of HRS, this agent demonstrated a dual effect by exerting a beneficial influence on splanchnic circulation, thereby improving renal function.

Results

Diagnosis, prevention, and treatment of HRS required identification and elimination of pathogenetic factors contributing to the development of hepatorenal dysfunction.

In group I, a restrictive transfusion strategy was implemented. In 7 (29.2 %) cases, bleeding was controlled conservatively using hemostatic agents. Interventional endoscopic treatment by means of variceal ligation was performed in 5 (20.8 %) patients, whereas placement of a Sengstaken-Blakemore tube was required in 12 (50.0 %) cases.

The median volume of packed red blood cell (PRBC) transfusion per patient was 349.5 mL (interquartile range 303.5–649.5). Under the restrictive strategy, 5 (20.8 %) patients in group I received transfusion of 3 units of PRBCs, 2 (8.4 %) patients received 2 units, and 17 (70.8 %) patients received 1 unit, allowing achievement of a mean hemoglobin level of 69.9 ± 6.1 g/L.

Hemotransfusion support according to a liberal strategy was performed targeting stabilization of hemoglobin levels at approximately 100 g/L.

In group II, under the liberal transfusion strategy, 1 (3.7 %) patient required transfusion of 1 PRBC unit, 4 (14.8 %) patients received 2 units, 11 (40.7 %) received 3 units, 7 (25.9 %) received 4 units, and 4 (14.8 %) received 5 units. The mean volume of transfused PRBCs per patient in this group was 1149.0 ± 421.6 mL. Endoscopic variceal ligation was performed in 6 (22.2 %) patients, whereas tamponade of bleeding varices using a Sengstaken-Blakemore tube was applied in 16 (59.3 %) cases. In 5 (18.5 %) patients, stable hemostasis was achieved pharmacologically. The mean post-transfusion hemoglobin level in group II was 97.96 ± 4.61 g/L.

The graph illustrating the mean volume of PRBC transfusion in patients of both groups is presented in Fig. 1.

Intra-abdominal hypertension > 15 mmHg necessitated therapeutic paracentesis in 13 (54.2 %) patients in group I and 18 (66.7 %) patients in group II, followed by intravenous administration of 6–8 g of 20% albumin solution per liter of ascitic fluid removed during paracentesis in order to maintain oncotic pressure.

Terlipressin was prescribed to 5 (20.8 %) patients in group I and 6 (22.2 %) patients in group II; however, positive clinical dynamics, manifested by increased diuresis and attenuation of serum creatinine elevation, were observed in only 1 (4.2 %) patient in group I.

Among 126 patients with decompensated PH hospitalized for bleeding from esophageal and/or gastric varices, 27 (21.4 %) underwent surgical intervention. In the early postoperative period, HRS developed in 1 (4.2 %) patient in group I following esophagogastric devascularization, stapled esophageal transection, and splenectomy, and in 2 (7.4 %) patients in group II who underwent esophagogastric devascularization, gastric fundus resection, and splenectomy.

In group I, 14 (58.3 %) patients died, including 7 (50.0 %) with community-acquired SBP, whereas in group II, 24 (88.9 %) people died, including 10 (41.7 %) with nosocomial SBP. Overall mortality was 74.5 % (n = 38) and was statistically significantly higher in group II (p = 0.03)

Patients with a fatal outcome demonstrated a higher volume of blood transfusion therapy compared to those who recovered (Fig. 2).

As shown in Fig. 2, patients with a fatal outcome had a higher median transfusion volume and a wider distribution of values compared to patients who recovered.

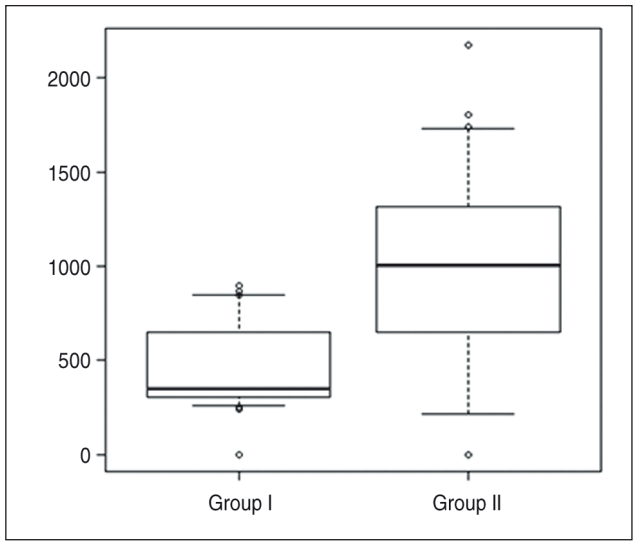


Figure 1. Mean volume of PRBC transfusion in patients of groups I and II, mL

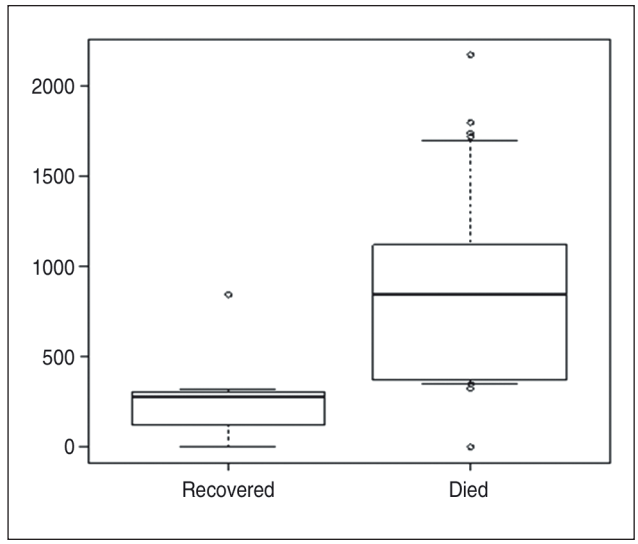


Figure 2. Blood transfusion volume in patients with HRS according to treatment outcome

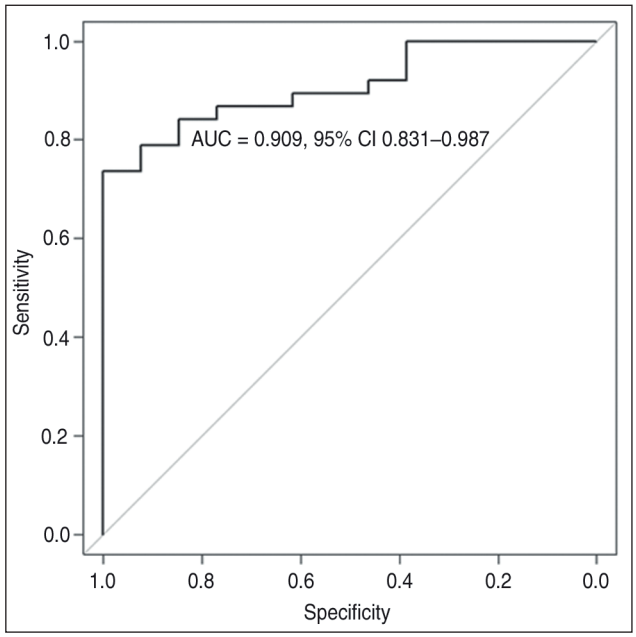


Figure 3. ROC analysis of the prognostic value of SBP, massive ascites, and hemotransfusion volume for mortality

Fig. 3 presents the results of receiver operating characteristic (ROC) analysis performed to evaluate the prognostic value of SBP, massive ascites, and hemotransfusion volume in relation to mortality in patients of the study groups.

According to logistic regression analysis, a higher volume of hemotransfusion showed a statistically significant association with mortality (odds ratio (OR) = 1.00, 95% confidence interval (CI) 1.00–1.01; p = 0.027), indicating its potential prognostic value.

Moderate ascites was associated with a reduced risk of mortality compared to massive ascites (OR = 0.072, 95% CI 0.010–0.494; p = 0.007), suggesting its stronger prognostic significance in patient stratification.

SBP demonstrated a tendency toward increased mortality risk (OR = 12.2, 95% CI 0.84–176; p ≈ 0.066), although this association did not reach statistical significance.

The prognostic model evaluating the combined effect of interacting complications in HRS development in decompensated PH showed statistical significance ($\chi^2 = 24.707$, df = 3, p < 0.0001). The discriminative ability assessed by ROC analysis confirmed high predictive accuracy (AUC = 0.909, 95% CI 0.831–0.987).

These findings indicate the potential prognostic relevance of the studied factors and may be used for mortality risk stratification in patients with HRS; however, further studies on a larger cohort are required to validate these results.

Thus, HRS develops in the context of progressive complications of decompensated PH. Its prevention depends on the effectiveness of ascites management, while infection of ascitic fluid — resulting from increased intestinal permeability and not representing a primary surgical problem — is not always diagnosed in a timely manner. Liberal transfusion strategy with a mean volume of 1149.0 ± 421.6 mL is associated with increased mortality.

Overall, management of HRS, due to the complexity of its pathophysiological mechanisms, remains highly challenging, is associated with high mortality, and requires further optimization of therapeutic approaches.

Discussion

Decompensated PH is a complex syndrome in which complications develop in an interrelated manner and mutually reinforce each other. Ascites is a key marker of PH progression, indicating transition to the decompensated stage and worsening prognosis [17]. Its development reflects portal hemodynamic impairment, systemic vasodilation, and neurohumoral dysregulation, creating conditions for SBP and HRS [18].

An important role is played by increased IAP due to ascites [19], which reduces abdominal perfusion pressure and impairs organ perfusion, particularly renal blood flow [20]. In combination with systemic vasodilation and activation of vasoconstrictor systems, this contributes to functional renal failure. Thus, ascites represents not only a manifestation of PH but also a driver of systemic hemodynamic derangements [21].

Infection of ascitic fluid further aggravates the pathological process. SBP develops as a result of bacterial translocation and is accompanied by a systemic inflammatory response [22]. In the setting of cirrhosis-associated immune dysfunction and reduced opsonic activity of ascitic fluid, conditions are created for bacterial proliferation [23], while cytokine and vasodilator release exacerbates circulatory disturbances and renal dysfunction [24].

HRS develops in the context of systemic vasodilation with reduced vascular resistance and redistribution of blood flow, leading to renal hypoperfusion. Cardiac dysfunction (cirrhotic cardiomyopathy) may also contribute by reducing cardiac output [25]. Decreased effective arterial blood volume activates the renin-angiotensin-aldosterone system and sympathetic nervous system, resulting in pronounced renal vasoconstriction [26].

Variceal bleeding from esophageal and/or gastric varices is a major destabilizing factor, as acute blood loss induces hemodynamic fluctuations and may lead to acute kidney injury [27]. At the same time, excessive fluid resuscitation increases portal pressure and the risk of rebleeding, necessitating a balanced therapeutic approach [11].

Current management includes vasoactive agents, endoscopic ligation, balloon tamponade when indicated, and portosystemic shunting techniques [28–31].

An essential component is transfusion strategy. A restrictive approach (Hb ≈ 70 g/L) helps avoid volume overload and increases in portal pressure, whereas a liberal strategy (Hb ≈ 100 g/L) is reserved for severe anemia. Evidence suggests superiority of restrictive transfusion strategies in reducing rebleeding risk and improving survival [32].

Treatment of HRS is aimed at correcting vasodilation and restoring effective arterial blood volume. The most effective approach involves vasoconstrictors combined with albumin infusion. Terlipressin improves renal perfusion by reducing splanchnic blood flow [33, 34], although it requires caution due to the risk of ischemic complications [35, 36].

In summary, ascites, its infection, bleeding, and hemodynamic disturbances form an interconnected pathological cascade promoting HRS development. This underscores the

need for a comprehensive approach with close monitoring of hepatic, renal, and systemic hemodynamics and early correction of complications.

Conclusions

Diagnosis, prevention, and treatment of HRS, identified in 51 (40.5 %) patients with decompensated PH, require a comprehensive approach including elimination of infectious factors (SBP), optimization of renal perfusion, and careful control of transfusion therapy. Early initiation of treatment based on diuresis and serum creatinine monitoring is essential.

Variceal bleeding, massive ascites with increased intra-abdominal pressure, and SBP form a self-perpetuating pathogenetic cycle leading to circulatory dysfunction and progression of renal failure. The highest mortality is observed in cases where these factors coexist.

Ethical compliance

Ethical approval for the study was obtained from the Bioethics Committee of Bogomolets National Medical University, Ministry of Health of Ukraine. All patients provided written informed consent for participation in the study in accordance with the recommendations of Biomedical Research Ethics Committees, the healthcare legislation of Ukraine, the 2000 version of the Declaration of Helsinki, and European Community Directive 86/609/EEC on human participation in biomedical research.

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Information about authors

Mykola Tutchenko, Bogomolets National Medical University, Kyiv, Ukraine; https://orcid.org/0000-0002-5050-6494
Denys Patrakh, PhD student, Bogomolets National Medical University, Kyiv, Ukraine; e-mail: dr.patrakh.denys@gmail.com; https://orcid.org/0000-0001-9724-0470
Diana Rudyk, Bogomolets National Medical University, Kyiv, Ukraine; https://orcid.org/0000-0001-9736-3614
Mykola Besedinskyi, Bogomolets National Medical University, Kyiv, Ukraine; https://orcid.org/0009-0009-8618-1458
Andrii Lovin, Bogomolets National Medical University, Kyiv, Ukraine; https://orcid.org/0000-0003-1109-6581
Ivan Klyuzko, Vyshhorod Central District Hospital, Vyshhorod, Kyiv region, Ukraine; https://orcid.org/0000-0001-8253-9349

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Тутченко М.І.[1], Патрах Д.М.[1], Рудик Д.В.[1], Беседінський М.С.[1], Льовін А.В.[1], Клюзко І.В.[2]
[1]Національний медичний університет імені О.О. Богомольця, м. Київ, Україна
[2]КНП «Вишгородська центральна районна лікарня», м. Вишгород, Україна

Взаємообтяжуючі ускладнення в розвитку гепаторенального синдрому при декомпенсованій портальній гіпертензії

Резюме. ***Актуальність.*** Гепаторенальний синдром (ГРС), що виникає при декомпенсованій портальній гіпертензії (ПГ), характеризується розвитком ниркової недостатності, зростанням рівня креатиніну крові, прогресуванням олігурії. У третини пацієнтів із цирозом печінки класу С за Child — Pugh розвивається ГРС. ***Мета:*** оптимізувати ранню діагностику й лікування, а також визначити фактори ризику розвитку ГРС і пов'язаної з ним летальності. ***Матеріали та методи.*** Проаналізовано 126 історій хвороб пацієнтів із декомпенсованою ПГ, яких лікували в КНП «Київська міська клінічна лікарня швидкої медичної допомоги» та на базі кафедри хірургії стоматологічного факультету НМУ імені О.О. Богомольця у 2022–2024 роках. ГРС виявлено в 51 (40,5 %) випадку. Критеріями встановлення діагнозу були підвищення рівня креатиніну > 150 мкмоль/л (1,69 мг/дл) або > 26,5 мкмоль/л (0,3 мг/дл) протягом 48 год за наявності олігурії без супутніх ознак ураження нирок, зокрема

гематурії. Пацієнтів розподілено на дві групи. До першої групи увійшло 24 (47,1 %) особи, у яких ГРС та позагоспітальний спонтанний бактеріальний перитоніт (СБП) діагностовано впродовж першої доби і при проведенні гемотрансфузійної підтримки дотримувались рестриктивної тактики. До другої — 27 (52,9 %) хворих, у яких ГРС та нозокоміальний СБП встановлено на 3-тю — 5-ту добу лікування, а гемотрансфузійну підтримку виконували з дотриманням ліберальної тактики. Оцінено роль гемотрансфузійної підтримки, тяжкості асциту й СБП у формуванні ГРС як взаємообтяжуючих чинників. ***Результати.*** Аналіз отриманих даних показав, що поєднання великого асциту, СБП та ліберальної гемотрансфузійної тактики формує несприятливий прогностичний комплекс, пов'язаний зі значним підвищенням ризику смерті. Розвиток ГРС частіше асоціювався з великим асцитом (70,8 %), нозокоміальним СБП (37,0 %) та ліберальною гемотрансфузійною тактикою (52,9 %). Серед 126 обстежених пацієнтів прооперовано 27 (21,4 %), а ГРС у післяопераційному періоді розвинувся в 1 (4,2 %) хворого першої групи та у 2 (7,4 %) — другої. Загальна летальність становила 74,5 % (n = 38), у першій групі померло 14 (58,3 %) осіб, у другій — 24 (88,9 %), p = 0,03. За результатами ROC-аналізу модель продемонструвала високу прогностичну здатність щодо летальності (AUC = 0,909; 95% довірчий інтервал 0,831–0,987), що підтверджує її добру дискримінаційну силу для стратифікації ризику в пацієнтів із ГРС. ***Висновки.*** ГРС є тяжким ускладненням декомпенсованої ПГ із високою летальністю. Ліберальна трансфузійна тактика, виражений асцит і СБП підвищують ризик його розвитку та прогресування. Ранній моніторинг діурезу й рівня креатиніну є ключовим для своєчасної діагностики, профілактики та лікування ГРС.

Ключові слова: гепаторенальний синдром; цироз печінки; портальна гіпертензія; альбумін; варикозна кровотеча; креатинін; діурез; спонтанний бактеріальний перитоніт