



RESEARCH ARTICLE



The role of intra-abdominal pressure in the evolution of ascitic syndrome complications in decompensated liver cirrhosis

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ABSTRACT

Introduction. The clinical value of admission intra-abdominal pressure in patients with decompensated liver cirrhosis and refractory ascites remains insufficiently defined, particularly in relation to the different phenotypes of ascitic fluid infection.

Material and methods. An exploratory observational analysis was performed on a structured dataset that included 100 patients with decompensated liver cirrhosis, refractory ascites, and a Child-Pugh class C score. All patients had the results of the diagnostic paracentesis performed at admission and the transvesical measurement of intra-abdominal pressure available. Ascitic fluid phenotypes were defined on the basis of the polymorphonuclear cell count and the ascitic culture result: spontaneous bacterial peritonitis, bacterascites, and sterile ascites. Intra-abdominal hypertension was defined as an intra-abdominal pressure ≥ 12 mmHg. The primary objective was the comparison of admission intra-abdominal pressure among phenotypes. The secondary objectives consisted of examining the associations between admission intra-abdominal pressure and acute kidney injury, sepsis, acute-on-chronic liver failure, and the need for intensive care.

Results. The cohort included 22 patients with spontaneous bacterial peritonitis, 10 with bacterascites, and 68 with sterile ascites. Median admission intra-abdominal pressure differed significantly among phenotypes, being highest in spontaneous bacterial peritonitis and lower in bacterascites and sterile ascites: 16.9 (15.2–17.9), 13.4 (11.7–15.4), and 13.4 (12.1–14.6) mmHg, respectively ($p < 0.001$). Intra-abdominal hypertension was present in all patients with spontaneous bacterial peritonitis, in 60.0% of those with bacterascites, and in 75.0% of those with sterile ascites. In adjusted logistic models, each 1-mmHg increase in admission intra-abdominal pressure was independently associated with spontaneous bacterial peritonitis (odds ratio [OR] 3.06; 95% confidence interval [CI] 1.56–6.02), any infected ascites (OR 1.56; 95% CI 1.20–2.02), sepsis (OR 2.12; 95% CI 1.44–3.12), acute-on-chronic liver failure (OR 1.43; 95% CI 1.09–1.86), and the need for intensive care (OR 1.40; 95% CI 1.06–1.83), but not with acute kidney injury (OR 0.91; 95% CI 0.73–1.14).

Conclusions. Higher admission intra-abdominal pressure was associated with a higher probability of spontaneous bacterial peritonitis and with a more severe in-hospital course. Admission intra-abdominal pressure did not show an independent association with acute kidney injury and appears to reflect mainly infectious and general clinical severity.

Keywords: intra-abdominal pressure, intra-abdominal hypertension, liver cirrhosis, refractory ascites, spontaneous bacterial peritonitis, bacterascites.

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Key messages

What is not yet known on the issue addressed in the submitted manuscript

The clinical significance of admission intra-abdominal pressure in decompensated liver cirrhosis with refractory ascites remains insufficiently clarified, particularly in relation to ascitic fluid phenotypes defined by cytological and microbiological criteria. Compar-

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ative data on the distribution of intra-abdominal pressure among spontaneous bacterial peritonitis, bacterascites, and sterile ascites are limited.

The research hypothesis

We hypothesized that elevated admission intra-abdominal pressure is associated with a higher probability of spontaneous bacterial peritonitis and with a more severe course in patients with Child-Pugh class C decompensated liver cirrhosis and refractory ascites. We further hypothesized that this association is more pronounced for indicators of infectious and systemic severity than for isolated renal dysfunction.

The novelty added by the manuscript to the already published scientific literature

The present work provides a comparative analysis of intra-abdominal pressure among three ascitic fluid phenotypes: spontaneous bacterial peritonitis, bacterascites, and sterile ascites. The results support the role of intra-abdominal pressure as a complementary severity marker, associated with infected ascites, sepsis, and acute-on-chronic liver failure, but not with acute kidney injury.

Introduction

Decompensated liver cirrhosis represents the advanced clinical stage of chronic liver disease and is characterized by ascites, bacterial infections, hepatic encephalopathy, variceal hemorrhage, renal dysfunction, and progressive multiple organ failure [1, 2]. Among these complications, ascites is particularly important because it marks the transition to a higher-risk clinical state, increases the need for repeated hospitalizations, and predisposes patients to spontaneous bacterial peritonitis, hepatorenal dysfunction, and acute-on-chronic liver failure [1-3]. Systemic inflammation and cirrhosis-associated immune dysfunction further amplify the vulnerability to infectious decompensation and organ failure in this population [4].

Intra-abdominal pressure is a measurable mechanical determinant of abdominal organ function. According to the World Society of the Abdominal Compartment Syndrome, intra-abdominal hypertension is defined as a sustained or repeated increase in intra-abdominal pressure to 12 mmHg or more, whereas abdominal compartment syndrome requires a pressure above 20 mmHg associated with newly developed organ dysfunction [5]. In patients with cirrhosis and tense or refractory ascites, increased intra-abdominal pressure may impair venous return, reduce abdominal perfusion, worsen renal perfusion, and compromise respiratory mechanics [5-7]. Previous physiological studies in cirrhosis have shown that decompression of tense ascites can improve hemodynamic and renal parameters, supporting the biological plausibility of pressure-related clinical deterioration [6, 7].

Ascitic fluid infection remains one of the most relevant acute complications of advanced cirrhosis. Current guidelines define spontaneous bacterial peritonitis by an ascitic polymorphonuclear cell count ≥ 250 cells/mm³, with or without a positive culture, in the absence of a surgically

treatable intra-abdominal source [2, 3]. Bacterascites is defined by a positive ascitic culture with a polymorphonuclear cell count < 250 cells/mm³, whereas sterile ascites is characterized by a negative culture and a polymorphonuclear cell count < 250 cells/mm³ [8-10]. Although these phenotypes are well recognized diagnostically, their relationship with admission intra-abdominal pressure has not been sufficiently clarified.

The available literature indicates that intra-abdominal hypertension is frequent in critically ill patients with cirrhosis and ascites and is associated with unfavorable outcomes, including infection, acute-on-chronic liver failure, and mortality [11, 12]. However, most published cohorts originated from intensive care units, emphasized serial or peak pressures rather than admission values, and did not stratify patients in detail according to ascitic infection phenotypes [11, 12]. The renal implications of abdominal pressure in cirrhosis may also depend on abdominal perfusion pressure and systemic hemodynamics rather than on the pressure itself [13-15].

In this context, the present study aimed to evaluate the association between admission intra-abdominal pressure, ascitic fluid phenotype, and early in-hospital course in patients with Child-Pugh class C decompensated liver cirrhosis and refractory ascites. We hypothesized that higher admission intra-abdominal pressure would be associated with a higher probability of spontaneous bacterial peritonitis and with a more severe in-hospital course.

Materials and methods

Study design and data source. The present work represents an exploratory observational analysis of a structured analytical dataset, developed within a postdoctoral research project focused on refractory ascites in decompensated liver cirrhosis, which included 100 patients. The data-

set was compiled within the Department of Surgery No. 2 of the *Nicolae Testemițanu* State University of Medicine and Pharmacy and the General Surgery Department no. 2 of the *Holy Trinity* Municipal Clinical Hospital, Chișinău, Republic of Moldova.

The dataset was analyzed after verifying the availability of the variables required for the objectives of the present work. The data were derived from the clinical, biological, microbiological, and hemodynamic evaluation of the included patients, with no additional interventions performed for the purpose of the present analysis. Reporting followed the STROBE recommendations applicable to observational studies.

Study population and eligibility criteria. The study included consecutive patients admitted to the General Surgery Department No. 2 of the *Holy Trinity* Municipal Clinical Hospital, Chișinău, Republic of Moldova, between January 2023 and December 2024.

The inclusion criteria were: a confirmed diagnosis of decompensated liver cirrhosis, refractory ascites, a Child-Pugh class C score, availability of the results of the diagnostic paracentesis performed at admission, and availability of the transvesical measurement of intra-abdominal pressure at admission.

The exclusion criteria were: ascites of non-cirrhotic etiology (cardiac, tuberculous, peritoneal carcinomatosis, or other non-cirrhotic causes), associated active oncological disease, and the absence of any of the mandatory parameters for inclusion.

Refractory ascites was defined according to the EASL and International Ascites Club criteria as ascites that cannot be mobilized or whose early recurrence cannot be prevented despite sodium restriction and maximal diuretic therapy (spironolactone 400 mg/day and furosemide 160 mg/day for at least one week), or ascites associated with diuretic-induced complications that preclude the use of effective doses [2, 3].

Following the application of these criteria, 100 patients were included in the final analysis.

Definitions of ascitic fluid phenotypes. Classification of the ascitic fluid was performed on the basis of the polymorphonuclear (PMN) cell count determined by cytology of the ascitic fluid obtained by diagnostic paracentesis at admission and the ascitic culture result, in accordance with the diagnostic criteria of the EASL 2018 and AASLD 2021 guidelines [2, 3, 8-10].

Spontaneous bacterial peritonitis was defined by an ascitic PMN cell count $\geq 250/\text{mm}^3$, irrespective of the culture result, in the absence of a surgically treatable intra-abdominal source of infection [2, 3]. Bacterascites was defined by a positive ascitic culture and a PMN cell count $< 250/\text{mm}^3$ [8-10]. Sterile ascites was defined by a negative culture and a PMN cell count $< 250/\text{mm}^3$ [2, 3, 8].

Applying this classification, the cohort comprised 22 patients with spontaneous bacterial peritonitis, 10 with bacterascites, and 68 with sterile ascites.

Measurement of intra-abdominal pressure. Intra-abdominal pressure was measured by the indirect transvesical

method, the standard method recommended by the World Society of the Abdominal Compartment Syndrome for the clinical assessment of intra-abdominal pressure [5].

The measurement technique was as follows: the patient was positioned in the complete supine position (0°), with relaxed abdominal musculature. The urinary bladder was emptied completely through the existing Foley catheter. The drainage tubing was clamped distal to the sampling port. Through the sampling port of the Foley catheter, 25 mL of sterile 0.9% saline solution was instilled aseptically into the urinary bladder. After instillation, a graduated manometric column positioned vertically was connected. The zero-reference point of the manometric column was set at the intersection of the midaxillary line with the iliac crest, according to the WSACS recommendations [5]. The height of the fluid column was read at the end of normal expiration, in the absence of abdominal muscle contractions, and was recorded in cmH_2O .

The values obtained in cmH_2O were converted to mmHg using the standard conversion factor established by the WSACS: $P(\text{mmHg}) = P(\text{cmH}_2\text{O})/1.36$ [5]. The values expressed in mmHg were used for all analyses.

Intra-abdominal hypertension was defined as an intra-abdominal pressure ≥ 12 mmHg, according to the consensus criteria of the World Society of the Abdominal Compartment Syndrome [5].

Outcomes. The primary objective of the study was the analysis of the distribution of admission intra-abdominal pressure among the three ascitic fluid phenotypes: spontaneous bacterial peritonitis, bacterascites, and sterile ascites.

The secondary objectives addressed the evaluation of the independent association between admission intra-abdominal pressure and early in-hospital clinical outcomes, grouped as follows.

Infectious outcomes: spontaneous bacterial peritonitis, defined by an ascitic PMN cell count $\geq 250/\text{mm}^3$ irrespective of the culture result, and any infected ascites, defined as spontaneous bacterial peritonitis or bacterascites [2, 3].

Renal outcome: acute kidney injury, defined according to the revised criteria of the International Club of Ascites as an increase in serum creatinine ≥ 0.3 mg/dL from baseline within 48 hours, or an increase of ≥ 1.5 times from baseline within 7 days [13].

Systemic severity outcomes: sepsis, defined according to the international consensus Sepsis-3 criteria as life-threatening organ dysfunction caused by a dysregulated host response to infection, with an increase in the SOFA score of ≥ 2 points [16] – a definition confirmed and maintained by the Surviving Sepsis Campaign 2021 guideline [17]; acute-on-chronic liver failure, defined according to the EASL-CLIF Consortium criteria [14]; and the need for intensive care, established on the basis of standard clinical criteria for transfer to the intensive care unit.

Acute kidney injury was evaluated separately from other systemic severity outcomes, given its multifactorial nature in advanced cirrhosis and its dependence on hemodynamic,

inflammatory, and exposure-duration factors, beyond the isolated value of intra-abdominal pressure [13-15].

Statistical analysis. Data were analyzed using IBM SPSS Statistics 29.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro-Wilk test and reported as medians (interquartile ranges); categorical variables were reported as counts and percentages. Between-group comparisons were made using the Kruskal-Wallis test for continuous variables and the chi-square or Fisher's exact test for categorical variables. Correlations between admission intra-abdominal pressure and severity markers were evaluated using Spearman's rank correlation coefficient. Associations with clinical outcomes were examined by multivariable logistic regression and expressed as adjusted odds ratios (95% confidence intervals). A p value <0.05 (two-tailed) was considered statistically significant.

Ethics approval and informed consent. The study was approved by the Research Ethics Committee of the *Nicolae Testemițanu* State University of Medicine and Pharmacy, Chișinău, Republic of Moldova (approval no. 1 of 12.05.2022, following the review of application no. 8 of 10.01.2022). The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants.

Results

The cohort comprised 22 patients with spontaneous bacterial peritonitis, 10 with bacterascites, and 68 with sterile ascites. The baseline characteristics of the cohort, stratified by ascitic fluid phenotype, are presented in Table 1. Across the entire group, the median Child-Pugh score was 11.0 (11.0–12.0), the median MELD-Na score was 26.0 (23.0–27.0), and the median admission intra-abdominal pressure was 13.9 (12.4–15.5) mmHg.

Table 1. Baseline characteristics of the study cohort

Variable	SBP (n=22)	Bacterascites (n=10)	Sterile ascites (n=68)	p value
<i>Demographics</i>				
Age, years — median (IQR)	58 (52–66)	59 (51–67)	57 (50–64)	0.742
Male sex, n (%)	15 (68.2)	7 (70.0)	44 (64.7)	0.919
BMI, kg/m ² — median (IQR)	27.0 (24.5–30.6)	26.8 (24.2–29.8)	26.4 (23.8–29.2)	0.841
<i>Etiology of cirrhosis, n (%)</i>				
Alcoholic liver disease	12 (54.5)	5 (50.0)	32 (47.1)	0.993
Hepatitis B virus	3 (13.6)	1 (10.0)	8 (11.8)	0.993
Hepatitis C virus	4 (18.2)	2 (20.0)	14 (20.6)	0.993
Mixed or other etiology	3 (13.6)	2 (20.0)	14 (20.6)	0.993
<i>Liver disease severity</i>				
Child-Pugh score — median (IQR)	12 (11–12)	11 (11–12)	11 (11–12)	0.047
MELD-Na score — median (IQR)	27 (25–29)	26 (24–28)	25 (23–27)	0.018
<i>Vital signs at admission</i>				
Heart rate, beats/min — median (IQR)	105 (96–116)	96 (88–104)	86 (78–94)	<0.001
Mean arterial pressure, mmHg — median (IQR)	73 (68–79)	78 (72–84)	82 (76–89)	0.001
Body temperature, °C — median (IQR)	38.0 (37.5–38.5)	37.4 (36.9–38.0)	36.9 (36.6–37.3)	<0.001
<i>Laboratory values at admission</i>				
White blood cell count, $\times 10^3/\mu\text{L}$ — median (IQR)	12.8 (10.5–15.9)	9.8 (7.6–12.1)	7.2 (5.8–9.0)	<0.001
C-reactive protein, mg/L — median (IQR)	84 (56–126)	48 (28–76)	24 (12–45)	<0.001
Procalcitonin, ng/mL — median (IQR)	1.20 (0.55–3.20)	0.45 (0.20–1.10)	0.12 (0.05–0.28)	<0.001
Serum creatinine, $\mu\text{mol/L}$ — median (IQR)	102 (80–137)	95 (76–124)	98 (75–130)	0.874
Serum lactate, mmol/L — median (IQR)	2.3 (1.7–3.4)	1.8 (1.3–2.6)	1.5 (1.1–2.0)	0.009

Note. SBP, spontaneous bacterial peritonitis; IQR, interquartile range; BMI, body mass index; MELD-Na, Model for End-Stage Liver Disease–sodium. Continuous data are presented as median (IQR); categorical data as number (percentage). Comparison among groups: Kruskal-Wallis test for continuous variables; chi-square test for categorical variables. A two-sided p value <0.05 was considered statistically significant.

Intra-abdominal pressure according to ascitic fluid phenotype. Admission intra-abdominal pressure differed significantly among the three phenotypes, with the highest values in the spontaneous bacterial peritonitis group (Table 2). Two patients, both in the spontaneous bacterial peritonitis group, had an admission intra-abdominal pressure >20 mmHg.

Correlations with severity markers. Spearman's rank correlations revealed positive associations between admission intra-abdominal pressure and C-reactive protein ($\rho=0.45$; $p<0.001$), procalcitonin ($\rho=0.41$; $p<0.001$), white

blood cell count ($\rho=0.33$; $p<0.001$), heart rate ($\rho=0.42$; $p<0.001$), body temperature ($\rho=0.31$; $p=0.002$), and serum lactate ($\rho=0.20$; $p=0.046$), as well as an inverse association with mean arterial pressure ($\rho=-0.25$; $p=0.012$). No significant correlation was identified with serum creatinine ($\rho=0.07$; $p=0.517$).

Early clinical outcomes. Early in-hospital outcomes differed significantly among phenotypes for the need for intensive care, sepsis, and acute-on-chronic liver failure, but not for acute kidney injury (Table 3).

Table 2. Admission intra-abdominal pressure according to ascitic fluid phenotype

Ascitic fluid phenotype	n	IAP, median (IQR), mmHg	IAH, n (%)
Spontaneous bacterial peritonitis	22	16.9 (15.2–17.9)	22 (100.0)
Bacterascites	10	13.4 (11.7–15.4)	6 (60.0)
Sterile ascites	68	13.4 (12.1–14.6)	51 (75.0)

Note. IAP, intra-abdominal pressure; IAH, intra-abdominal hypertension; IQR, interquartile range. IAP is presented as median (IQR), in mmHg. IAH was defined as IAP ≥12 mmHg and is expressed as number (percentage). Comparison of IAP among the three phenotypes: Kruskal–Wallis test, $p<0.001$. A two-sided p value <0.05 was considered statistically significant.

Table 3. Early in-hospital outcomes according to ascitic fluid phenotype

Outcome	SBP (n=22)	Bacterascites (n=10)	Sterile ascites (n=68)	p value
Need for intensive care, n (%)	6 (27.3)	2 (20.0)	3 (4.4)	0.007
Acute kidney injury, n (%)	4 (18.2)	1 (10.0)	9 (13.2)	0.784
Sepsis, n (%)	9 (40.9)	2 (20.0)	3 (4.4)	<0.001
Acute-on-chronic liver failure, n (%)	10 (45.5)	3 (30.0)	10 (14.7)	0.010

Note. SBP, spontaneous bacterial peritonitis. Data are presented as number (percentage). Comparison of proportions among phenotypes: chi-square test. A two-sided p value <0.05 was considered statistically significant.

Multivariable analysis. In multivariable logistic regression models, each 1-mmHg increase in admission intra-abdominal pressure was independently associated with spontaneous bacterial peritonitis, any infected ascites, sepsis, acute-on-chronic liver failure, and the need for intensive care, but not with acute kidney injury (Table 4).

Table 4. Multivariable logistic regression models for admission intra-abdominal pressure as a predictor of selected outcomes

Outcome	Adjusted OR per 1 mmHg IAP	95% CI	p value
Spontaneous bacterial peritonitis*	3.06	1.56–6.02	0.001
Any infected ascites**	1.56	1.20–2.02	<0.001
Acute kidney injury	0.91	0.73–1.14	0.430
Sepsis	2.12	1.44–3.12	<0.001
Acute-on-chronic liver failure	1.43	1.09–1.86	0.009
Need for intensive care	1.40	1.06–1.83	0.017

Note. OR, odds ratio; CI, confidence interval; IAP, intra-abdominal pressure; PMN, polymorphonuclear; MELD-Na, Model for End-Stage Liver Disease–sodium. The models were adjusted for mean arterial pressure, serum creatinine at admission, and MELD-Na. *Spontaneous bacterial peritonitis defined by a PMN cell count $\geq 250/\text{mm}^3$, irrespective of the culture result. **Any infected ascites defined as spontaneous bacterial peritonitis or bacterascites. A two-sided p value <0.05 was considered statistically significant.

Discussion

The distribution of admission intra-abdominal pressure among ascitic fluid phenotypes – spontaneous bacterial peritonitis, bacterascites, and sterile ascites – remains insufficiently elucidated in the literature, with a lack of direct comparative data, particularly in patients evaluated outside

intensive care units [11, 12]. The present analysis demonstrates that admission intra-abdominal pressure differs significantly among the three phenotypes, that elevated values are independently associated with markers of infectious and systemic severity, and that there is no independent association with acute kidney injury.

The independent association between admission intra-abdominal pressure and spontaneous bacterial peritonitis has not previously been quantified in cohorts stratified by ascitic infection phenotypes. The present analysis demonstrates that each 1-mmHg increase in admission intra-abdominal pressure is independently associated with spontaneous bacterial peritonitis, with an adjusted odds ratio of 3.06 (95% CI 1.56–6.02). This association is biologically coherent – the neutrophilic inflammatory response that defines spontaneous bacterial peritonitis, with PMN $\geq 250/\text{mm}^3$ according to the EASL and AASLD criteria [2, 3], generates an increase in the protein content of the ascitic fluid, abdominal wall edema, and accelerated fluid accumulation – mechanisms that contribute directly to the increase in intra-abdominal pressure [5].

The abdominal mechanical profile of bacterascites relative to spontaneous bacterial peritonitis and sterile ascites has not previously been characterized through the measurement of intra-abdominal pressure. The present analysis demonstrates that bacterascites presents with a median intra-abdominal pressure of 13.4 mmHg – identical to that of sterile ascites and significantly lower than in spontaneous bacterial peritonitis – and intra-abdominal hypertension was present in 60.0% of patients with bacterascites compared with 100.0% in spontaneous bacterial peritonitis. Ascitic culture positivity in the absence of a neutrocytic response does not produce the same abdominal mechanical load as spontaneous bacterial peritonitis. Although previous studies have suggested that bacterascites may approach spontaneous bacterial peritonitis in severity and prognosis [9, 10], the data from the present analysis add an abdominal mechanical perspective, demonstrating that the two entities are distinct in terms of intra-abdominal pressure.

Previously published studies have demonstrated that intra-abdominal hypertension in critically ill cirrhotic patients is associated with organ dysfunction and increased mortality [11, 12]; however, these studies did not evaluate the predictive value of a single admission measurement outside the intensive care unit, stratified by ascitic phenotype. The present analysis demonstrates that a single measurement of admission intra-abdominal pressure – prior to any therapeutic intervention – is independently associated with sepsis (adjusted odds ratio 2.12; 95% CI 1.44–3.12), acute-on-chronic liver failure (adjusted odds ratio 1.43; 95% CI 1.09–1.86), and the need for intensive care unit admission (adjusted odds ratio 1.40; 95% CI 1.06–1.83), after adjustment for mean arterial pressure, serum creatinine, and MELD-Na.

The relationship between admission intra-abdominal pressure and acute kidney injury in this specific population has not previously been analyzed in adjusted models.

The present analysis demonstrates the absence of an independent association (adjusted odds ratio 0.91; 95% CI 0.73–1.14; $p=0.430$). Physiological studies have shown that large-volume paracentesis improves renal function [6, 7]; however, these studies evaluate the effect of a therapeutic intervention, not the predictive value of a parameter at a fixed time point. Acute kidney injury in decompensated cirrhosis results from the interaction among splanchnic vasodilation, activation of the renin-angiotensin-aldosterone system, and a reduction in effective circulating volume [13–15] – factors that cannot be captured by a single admission pressure value. The non-significant Spearman's correlation with serum creatinine ($\rho=0.07$; $p=0.517$), in contrast to the significant correlations with C-reactive protein ($\rho=0.45$), procalcitonin ($\rho=0.41$), heart rate ($\rho=0.42$), and mean arterial pressure ($\rho=-0.25$), confirms that admission intra-abdominal pressure is a marker of infectious and systemic severity, not of isolated renal risk.

From a practical standpoint, admission intra-abdominal pressure, measured by the standardized transvesical technique [5], is an accessible, non-invasive, and reproducible parameter that can be integrated into the initial evaluation of patients with decompensated liver cirrhosis and refractory ascites. Interpreted together with clinical and biological data, it contributes to the early identification of cases at an increased risk of spontaneous bacterial peritonitis and a severe clinical course. Intra-abdominal pressure does not replace diagnostic paracentesis and the cytological and microbiological interpretations of the ascitic fluid [2, 3], but rather complements them in the initial evaluation.

The observational design and the small size of the bacterascites subgroup ($n=10$) limit the generalizability of the results, which require confirmation in prospective studies.

Conclusions

Elevated admission intra-abdominal pressure was associated with a higher probability of spontaneous bacterial peritonitis and with a more severe in-hospital course in patients with decompensated liver cirrhosis and refractory ascites. It was independently associated with infected ascites, sepsis, acute-on-chronic liver failure, and the need for intensive care, but not with acute kidney injury, reflecting a predominantly infectious and systemic severity. Measurement of intra-abdominal pressure by the standardized transvesical method may complement the initial evaluation of these patients, contributing to the early stratification of severity. Intra-abdominal pressure does not replace diagnostic paracentesis and the cytological and microbiological interpretations of the ascitic fluid, and the present findings require confirmation in prospective studies.

Competing interests

None declared.

Authors' contributions

GA and SP conceived the study and developed the analytical framework. GA, SP, OC, TZ, GL, and LC collected the data. GA, SP, and TZ analyzed and interpreted the data. SP

drafted the manuscript. GA and SP critically reviewed the manuscript for important intellectual content. All authors approved the final version of the manuscript.

Ethics approval

The study was approved by the Research Ethics Committee of *Nicolae Testemițanu* State University of Medicine and Pharmacy, Chișinău, Republic of Moldova (approval no. 1 of 12.05.2022). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patient consent

Obtained.

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The authors used generative artificial intelligence tools for linguistic and editorial assistance in the process of writing and revising the manuscript. No artificial intelligence tool was used to generate, modify, or analyze the study data. The authors critically reviewed, edited, and approved the final manuscript and take full responsibility for its scientific content.

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