ORIGINAL ARTICLE

CONTENTS

Prediction of abdominal compartment syndrome in patients with severe acute pancreatitis

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ABSTRACT

Aim: To identify factors associated with the risk of developing abdominal compartment syndrome (ACS) in patients with severe acute pancreatitis (SAP).

Materials and Methods: A retrospective single-center cohort study of 106 patients with SAP, complicated by ACS (n=32) and without the given complication (n=74). The first stage included an intergroup comparison of 28 clinical-laboratory indicators recorded during the early stages of the disease. The second and third stages consisted of univariate and multivariate logistic regression analyses of the variables selected over the first stage, developing a prediction model, and evaluating its characteristics.

Results: Statistically significant difference between the groups was found for the following 14 indicators: BMI, Glasgow Coma Scale score, visceral obesity index, CRP level, radiological signs of paresis, pleural effusion, fluid collections in the retroperitoneal space, daily fluid balance, pain intensity according to the NPRS scale, the total score on the Marshall and BISAP scales, early persistent SIRS, APP and RFG levels ($p<0.05$). The given factors were identified as independent risk factors for ACS development: daily fluid balance, pain intensity (NPRS), retroperitoneal fluid collections, early persistent SIRS, and elevated WHR. The developed logistic model demonstrated high discriminatory ability: AUC = 0.92, sensitivity – 94%, specificity – 81%.

Conclusions: The developed model can enable patient stratification and targeted correction of potentially modifiable ACS risk factors in the early stages of SAP.

KEY WORDS: acute severe pancreatitis, abdominal compartment syndrome, early prediction, logistic regression

Wiad Lek. 2025;78(9):1751-1757. doi: 10.36740/WLek/212506 DOI

INTRODUCTION

Abdominal compartment syndrome (ACS) is characterized by sustained intra-abdominal pressure (IAP) exceeding 20 mmHg and organ failure (OF). Regardless of the etiology, it requires prompt decision-making, intensive therapy and urgent surgical intervention when conservative methods are ineffective [1]. Acute pancreatitis (AP) is accompanied by intra-abdominal hypertension (IAH) in 50-60% of cases, and in 15-30% of these cases, it progresses to ACS [2, 3]. The systemic and local manifestations of severe acute pancreatitis (SAP) worsen with the development of ACS, a rare complication that significantly worsens the course and prognosis, with mortality rates increasing to 75% [4, 5].

The course, management, and prevention of intra-abdominal hypertension (IAH) progression in severe acute pancreatitis (SAP) are actively being researched. However, knowledge on the risk factors for ACS in SAP still remains limited [6-9]. Hence, the critically important need for the review of approaches to patient stratification in SAP and IAH, depending on the response to complex therapeutic measures, within the concept of preventing irreversible ACS outcomes [10, 11]. In the given study, we analyzed information from medical records of SAP patients to study the risk factors for ACS development, followed by the systematization of prediction.

AIM

To identify factors associated with the risk of developing abdominal compartment syndrome in patients with severe acute pancreatitis.

MATERIALS AND METHODS

A retrospective non-randomized single-center cohort study was conducted among 106 patients treated at the General Surgery Department No. 1 of Bogomolets National Medical University, between 2018 and 2024, with a diagnosis of SAP (K-85). Patients were divided into two groups:

- Group A – Patients with SAP complicated by ACS, n=32;
- Group B – Patients with SAP not complicated by ACS, n=74.

The diagnosis and severity of AP were established based on the revised Atlanta criteria (2012). The modified

Table 1. Intergroup comparison of variables (First stage of the study)

№	Variable, units of measurement	Group A (SAP + ACS) N = 32	Group B (SAP - ACS) N = 74	p-value
1.	Sex	Male 21(65.6%)	Male 52(71.6%)	0.653[X]
2.	Age, years	52.84 ± 9.79	55.01 ± 9.33	0.283[T]
3.	BMI, kg/m^2	31.28 ± 2.99	26.64 ± 2.64	<0.001[T]
4.	Onset, hours	6(5 - 7)	5(5 - 6)	0.159[U]
5.	Etiology	A - 13(40.6%)	23(31.1%)	0.824[F]
		B - 12(37.5%)	27(36.5%)	
		H - 3(9.4%)	9 (12.2%)	
		P - 1(3.1%)	4(5.4%)	
		I - 3(9.4%)	11(14.9%)	
6.	Charlson Index, points	2(1 - 3)	2(1 - 3)	0,725[U]
7.	Glasgow Coma Scale, points	14(13 - 14)	15(14 - 15)	<0.001[U]
8.	Waist-to-Hip Ratio, %	96(90.5 - 100)	92.5(87 - 97)	0.01[U]
9.	Platelets, 10^9/l	269.5 ± 66.15	273.7 ± 71.97	0.780[T]
10.	Amylase, U	969.9 ± 532.6	1025 ± 495	0.611[T]
11.	C-reactive protein, mg/l	102(93 - 124)	98(88 - 104)	0.03[U]
12.	Albumin, g/l	30.75 ± 4.32	30.9 ± 3.68	0.865[T]
13.	AST, U/l	44.3(29.4 - 54.35)	49.65(31.2-62.1)	0.184[U]
14.	Creatinine, μmol/l	89.28 ± 12.65	93.97 ± 13.67	0.1[T]
15.	Glucose, mmol/l	8.1(6.65 - 9.4)	8(6.8 - 9.5)	0.880[U]
16.	Radiological signs of paresis	Yes - 18(56.3%)	Yes - 20(27.8%)	0.007[X]
17.	Pleural effusion	Yes - 21(65.6%)	Yes - 29(39.2%)	0.02[x]
18.	Inferior vena cava	19.2 ± 3.84	18.9 ± 3.5	0.742[T]
19.	Retroperitoneal fluid collections	Yes - 20(62.5%)	Yes - 28(37.8%)	0.03[x]
20.	Daily fluid balance, l	2.76 ± 0.45	2.35 ± 0.38	<0,001[T]
21.	Diuresis, l	1.2(0.95 - 1.4)	1.25(1.05 - 1.45)	0.364[U]
22.	NPRS scale, max points	8.3 ± 0.99	7.1 ± 1.1	<0.001[T]
23.	BISAP, points	3.3 ± 0.73	2.9 ± 0.86	0.04[T]
24.	Marshall, points	2.2±0.81	1.9±0.75	0,02[T]
25.	Early persistent SIRS	Yes - 21 (65.6%)	Yes - 31 (41.9%)	0.03[F]
26.	MAP, mmHg	93.59±5.58	94.43±5.98	0.498[T]
27.	APP, mmHg (3rd day)	77.79±5.18	82.14±6.9	<0.001[T]
28.	RFG, mmHg (3rd day)	61.99±5.1	69.84±8.1	<0.001[T]

T - Student's t-test, U - Mann-Whitney U test, X - Chi-square (χ^2), F - Fisher's Exact Test

Source: compiled by the authors of this study

Marshall score was used to assess the severity of organ failure (OF), [12, 13]. ACS was diagnosed with IAP ≥20 mmHg and progression of organ failure occurred.

Inclusion Criteria:

- Persistent organ failure (>48 hours) and local complications of SAP;
- Hospitalization to the intensive care unit (ICU) within the first 72 hours from the onset of symptoms;
- Written informed consent for data processing.

Demographic, epidemiological, and clinical-laboratory data from the first seven days of hospitalization or less (for deceased patients) were collected from the medical records. The study included sex, age, body mass index (BMI), and the time in hours from the onset of symptoms. Upon admission, data on the etiology of AP, Charlson comorbidity index, Glasgow Coma Scale score, and Bedside Index of Severity in Acute Pancreatitis (BISAP) score [14] were collected. On the first day of hospitalization, all patients underwent anthropometric measurement of visceral obesity index using the Waist-to-Hip Ratio (WHR) method – waist circumference (cm) divided by hip circumference (cm) – with the result recorded in percentages for convenience in calculations.

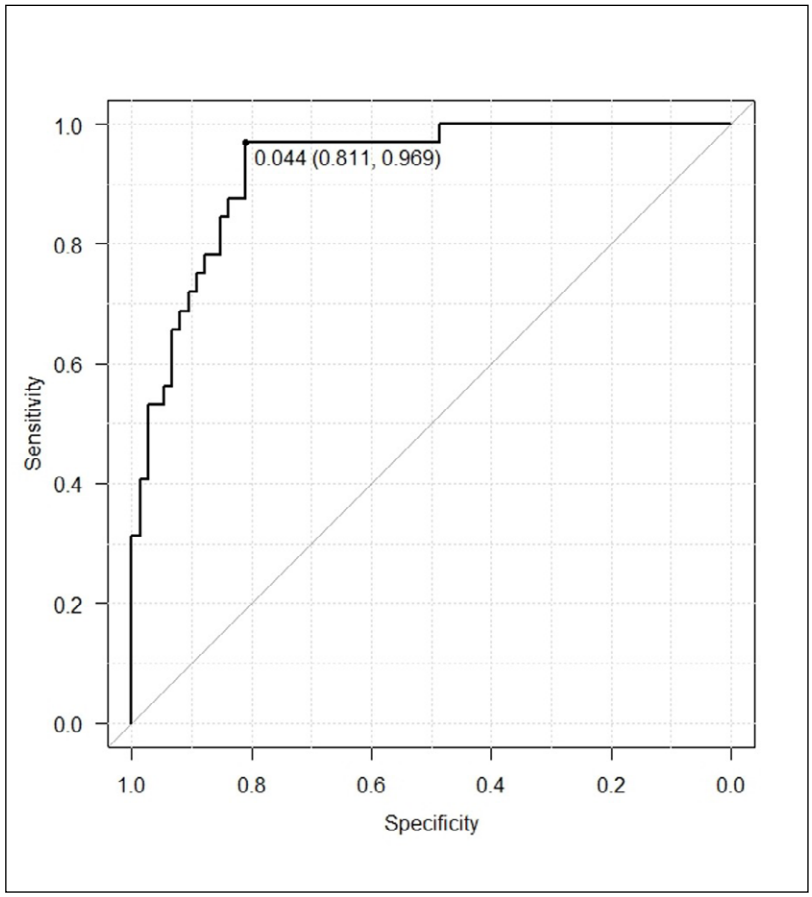

Fig. 1. ROC curve for predicting the risk of ACS in patients with SAP
Picture taken by the authors

The laboratory indicators selected for the first day included the following: platelet count, serum amylase, C-reactive protein (CRP) level, albumin, aspartate aminotransferase (AST) level, creatinine, and venous blood glucose level.

After visual patient's examination over the first day, the following signs were also recorded: evidence of paresis (marked pneumatosis or the presence of single horizontal fluid levels) on abdominal radiography and pleural effusion detected during ultrasound (US) examination. The maximum diameter of the inferior vena cava (IVC) during ultrasound examination of the abdominal organs and the presence of retroperitoneal fluid collections were noted during the first two days after hospitalization.

After one day of treatment, daily fluid balance and diuresis (in liters) were recorded. Pain intensity, assessed by the patient using the Numeric Pain Rating Scale (NPRS), a simplified version of the Visual Analog Scale, was also recorded. For calculations, the maximum score obtained during the first day of hospitalization was used. Data on daily intra-abdominal pressure (IAP) measurements via transurethral catheter in mmHg, mean arterial pressure (MAP), abdominal perfusion pressure (APP), and renal filtration gradient (RFG) were also recorded. The presence of persistent early systemic inflammatory response syndrome (SIRS) diagnosed within the first 24 hours and lasting for more than 24 hours was included in the study.

For presenting quantitative data, the mean ± standard deviation was used for normal distribution, while for non-normal distribution it was, the median and interquartile range (IQR) were presented. For categorical data, the absolute number and percentage were provided. Continuous variables were compared using the Student's t-test and the Mann-Whitney U test, while categorical data were analyzed using Fisher's exact test and the chi-square test. A univariate logistic regression analysis was conducted to evaluate the association of selected variables with the development of ACS. Odds ratios with 95% confidence intervals were calculated. Variables showing statistical significance were considered potential predictors and were included in the multivariate analysis. To assess the discriminatory ability of the constructed multivariate logistic model, the area under the ROC curve (AUC) was used. Results were considered statistically significant with $p < 0.05$. Data were processed using the standard Microsoft Excel 365 package. For calculations and analysis, MedStat v.5.2 and EZR (R-statistics) [15] were used.

RESULTS

In Group A, there were 21 male patients (65.6%), with an average age of 52.84 ± 9.79 years, and 25 (78%) patients, who died due to complications of the primary disease.

Table 2. Results of univariate regression analysis

№	Variable, units	OR (95% CI)	p-value
1.	BMI, kg/m^2	3.32 (1.94 - 5.68)	<0.001
2	Glasgow Coma Scale, points	0.21 (0.10 - 0.44)	<0.001
3.	Waist-to-Hip Ratio, %	1.08 (1.01 - 1.16)	0.01
4.	C-reactive protein, mg/l	1.03 (1.01 - 1.06)	0.001
5.	Radiological signs of paresis	3.47 (1.46 - 8.26)	0.005
6.	Pleural effusion	13.2 (4.9 -35.5)	<0.001
7.	Retroperitoneal fluid collections	2.74 (1.16 - 6.45)	0.02
8.	Daily fluid balance, ml	12.4 (3.5 - 44.1)	<0.001
9.	Max NPRS scale, points	2.77 (1.72 - 4.46)	<0.001
10.	BISAP, first day, points	1.66 (0.99 - 2.76)	0.05
11.	Marshall, first day, points	1.82 (1.05 - 3.16)	0.03
12.	Early persistent SIRS	2.65 (1.12 - 6.28)	0.02
13.	APP on the 3rd day, mmHg	0.9 (0.83 - 0.96)	0.003
14.	RFG on the 3rd day, mmHg	0.85 (0.78 - 0.92)	<0.001

Source: compiled by the authors of this study

Table 3. Coefficients of the final model for predicting ACS risk in patients with SAP – five independent predictors

Variable	Coefficient, b ± mb	p-value	OR (95% CI)
Const	-31.42 ± 6.86	<0.001	-
Fluid balance, day 1	2.4 ± 0.91	0.008	11.1 (2.0 - 62.1)
Max NPRS, day 1	1.24 ± 0.35	<0.001	3.5 (1.82 - 6.73)
Retroperitoneal fluid collections	1.98 ± 0.74	0.007	7.65 (1.88 - 31.2)
Early persistent SIRS	2.34 ± 0.83	0.004	11.1 (2.33 - 53.2)
Waist-to-Hip Ratio, %	0.11 ± 0.04	0.01	1.13 (1.03 - 1.23)

Source: compiled by the authors of this study

In Group B, there were 52 male patients (71.6%), with an average age of 55.01 ± 9.33 years, and 28 (38%), patients who died. The patients did not differ in age, sex, time from the onset of symptoms, comorbidities, or etiology ($p > 0.05$). When performing the first stage of intergroup comparison of selected variables, a statistically significant difference was found among 14 indicators (Table 1).

In the second stage, a series of univariate regression analyses was conducted for the variables that showed statistically significant differences in the intergroup comparison. In the univariate logistic regression analysis, all 14 variables demonstrated statistically significant relevance (Table 2). When selecting variables for the third stage, the potential modifiability of the variables was considered, and variables with multicollinearity or a high risk of overfitting the predictive model were excluded.

In the third stage of the study, a multivariate logistic regression model was constructed using the variables selected during the previous stage. The analysis revealed a relationship between the risk of developing ACS and five factor characteristics: fluid balance, maximum NPRS score during the first day of treatment, presence of retroperitoneal fluid collections during the first 48 hours of observation, early persistent SIRS, and the visceral obesity index WHR. The model assessment coefficients are presented in Table 3.

The obtained mathematical model for predicting the risk of abdominal compartment syndrome can be expressed by the following formula:

logit (P) = -31.42 + 2.4X1 + 1.24X2 + 1.98X3 + 2.34X4 + 0.11*X5,

- logit(p) = p / (1-p) – the log odds of ACS development;
- X1 – fluid balance during the first day of treatment, l;
- X2 – maximum NPRS score during the first day of treatment;
- X3 – presence of retroperitoneal fluid accumulation (0/1);
- X4 – presence of early persistent SIRS (0/1);
- X5 – visceral obesity index using the Waist-to-Hip Ratio method, percentage.

The suggested test allows to predict the risk of ACS with AUC = 0.92 (95% CI 0.88 – 0.97) (Fig. 1). To choose

the optimal threshold value for the test, the Youden Index calculation method was used. The optimal decision threshold was $P_m = 0.044$: for a patient with SAP, if the value is equal to or greater than P_m, the risk of ACS is predicted, and for a patient with a value below P_m, the risk of ACS is not indicated. At this decision threshold, the sensitivity of the test was 94% (95% CI 79-99), the specificity of the test was 81% (95% CI 70-89), the positive predictive value (PPV) of the test was 68% (95% CI 52-81), and the negative predictive value (NPV) of the test was 97% (95% CI 88-99).

DISCUSSION

In the given study, we developed and analyzed a predictive model for the risk of ACS in patients with SAP. The proposed model includes five available clinical factors that showed a statistically significant association with the development of this complication. Among these five predictors, at least three – infusion load, pain control, and modification of the systemic inflammatory response -can be actively corrected in the early days of the disease.

The risks of fluid overload during infusion therapy in AP are associated with the development and progression of intra-abdominal hypertension (IAH) [16-20]. Aggressive infusion has logical justifications. However, it can exacerbate the movement of intravascular fluid into the third space, initiated by the action of pro-inflammatory cytokines, which potentiates IAH. On the other hand, positive fluid balance in the early period is limited by diuresis and can be explained by excretory capacity, which is often compromised in SAP [21, 22].

Pain intensity on the NPRS scale during the first day was included in our study as a potentially modifiable predictor of ACS. A systematic review and meta-analysis on pain management in the early stages of SAP (2024) evaluated the effect of seven drugs based on dynamics using the visual analog scale, and in our study a simplified derivative was used [23].

The role of pathological pressure increase in the retroperitoneal space initiated by SAP was considered, highlighting retroperitoneal fluid collections as an independent predictor of ACS. The updated definitions of the World Society of Abdominal Compartment Syndrome (WSACS) emphasized the complexity of ACS mechanisms, referring to the retroperitoneum, pelvis, and omentum as additional compartments among the four main compartments involved in polycompartment syndrome [24]. In previous studies based on a limited sample, we examined the relationship between IAH and the volume of necrosis [25].

Systemic Inflammatory Response Syndrome (SIRS) has been established as an independent predictor of the severity of AP [26] and is utilized as a component of prognostic scales [27, 28]. The duration of SIRS precedes organ failure (OF), and when present, it increases the risk of progression to OF and death [29, 30]. SIRS during the first day is capable of predicting the severity of the course of AP [31].

In 2024, a retrospective study of more than a million patients with AP was published, identifying obesity and blood transfusions as independent risk factors for ACS [32]. Obese patients with AP have been proven to have a worse prognosis [33]. Visceral obesity significantly impacted the outcomes and severity of hypertriglyceridemic pancreatitis [34, 35]. A high level of visceral adipose tissue is an independent negative prognostic marker of AP [36].

CONCLUSIONS

1. The group of patients with severe acute pancreatitis and abdominal compartment syndrome in the studied sample exhibited worse clinical-laboratory indicators compared to the group with reversible or absent intra-abdominal hypertension.
2. In the studied sample of patients with severe acute pancreatitis, the development of abdominal compartment syndrome was associated with the following five independent risk factors: daily fluid balance, pain intensity on the NPRS scale, retroperitoneal fluid collections, early persistent SIRS, and high visceral obesity index (WHR).
3. The developed logistic model demonstrated high discriminatory ability with a decision threshold for predicting the event at 0.044: AUC = 0.92 (95% CI 0.88–0.97), sensitivity 94%, specificity 81%.
4. The practical value of the model lies in the ability to stratify patients and target the correction of potentially modifiable risk factors in the early stage of the disease.
5. Further external validation on independent samples is required to assess universal degree of the model.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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RECEIVED: 06.04.2025
ACCEPTED: 25.08.2025