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The influence of intraabdominal pressure on the mortality rate of patients with acute pancreatitis

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Background/aim: Intraabdominal hypertension (IAH) is a common clinical finding in patients with acute pancreatitis and is associated with poor prognosis. This study aimed to determine the impact of intraabdominal pressure (IAP) on the mortality rate in patients with acute pancreatitis in an intensive care unit.

Materials and methods: A total of 50 patients with acute pancreatitis were included in this prospective cohort study. Based on the obtained values of IAP, the patients were divided into two groups: those with normal IAP (n = 14) and increased IAP (n = 36). Mean values of IAP were compared with examined variables.

Results: The mortality rate of the study group was 40%. Comparing the IAP and treatment outcomes, it was proved that there were statistically highly significant differences (P = 0.012). Increasing the value of IAP increased the mortality rate. Deceased patients in the IAH group had greater statistical significance of APACHE II score (P = 0.016), abdominal perfusion pressure (P = 0.048), lactate (P = 0.049), hematocrit (P = 0.039), Ranson's criteria on admission (P = 0.017), Ranson's criteria after 48 h (P = 0.010), Sequential Organ Failure Assessment score (P = 0.014), and body mass index (P = 0.012) compared to the surviving patients.

Conclusion: IAP has an impact on the increase of mortality rates in patients with acute pancreatitis.

Key words: Intraabdominal pressure, intraabdominal hypertension, abdominal compartment syndrome, acute pancreatitis, organ dysfunction

1. Introduction

The mortality of patients with acute pancreatitis is very high. Most commonly it occurs as a result of multiple organ damage, the development of necrotizing pancreatitis, and the presence of bacterial infection. It was also observed that patients who die of acute pancreatitis have developed intraabdominal hypertension (IAH). The massive fluid resuscitation in the early stages of acute pancreatitis combined with inflammatory processes in the retroperitoneum results in visceral edema, which leads to an increase in intraabdominal pressure (IAP) (1–4). It was observed that patients who die of acute pancreatitis due to a sudden increase in IAP can have developed IAH, which leads to early organ damage (5–7).

Until the World Society of Abdominal Compartment Syndrome (WSACS) conference (8–10), IAH and

abdominal compartment syndrome (ACS) incidences were different in the reports of various hospitals. After the WSACS conference it was concluded that the incidence of IAH in acute pancreatitis was 60%. On admission, 70% of patients already have IAH, and if there is no IAH on admission it develops during the first days or the IAP will increase in value. In patients who die the IAP values are increased, while in others they are gradually reduced (6,11–13). IAH is defined according to the WSACS recommendations (10) as the persistent or repeated pathological increase in IAP to ≥ 12 mmHg (10). The diagnostic criteria for acute pancreatitis are defined according to the American College of Gastroenterology guidelines (14) as the presence of two out of three of the following: typical abdominal pain, a threefold increase of

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serum amylase or lipase, and verification of findings for unclear cases of acute pancreatitis on CT examination (14). The aim of this study is to determine the impact of IAP on the mortality rate of patients with acute pancreatitis in an intensive care unit (ICU).

2. Materials and methods

This prospective cohort study was conducted from January 2011 to December 2013 in the ICU of Clinical Hospital Center "Zvezdara", Belgrade. A total of 50 patients with acute pancreatitis were included in the study. The diagnostic criteria for acute pancreatitis were defined according to the American College of Gastroenterology guidelines (14).

The study was approved by the local medical ethics committee (Clinical Ctr Zvezdara IRB 1-Medical, No IRB00003818, Approved by Federalwide Assurance – FWA00006109). All study participants or their legal guardians provided informed written consent prior to study enrollment and there was no conflict of interest.

Based on the WSACS recommendations (10), observed patients were divided into two groups according to the mean values of IAP: a group with normal values of IAP (<12 mmHg) and a group with high levels of IAP. Furthermore, patients were divided according to the outcome of treatment in each group separately and were followed until exiting the ICU.

IAP measurement was performed according to the recommendations of the WSACS (8). All patients had a urinary catheter placed into the bladder and the other end was connected with a three-way stopcock to the urine bag, and part of the hose from the infusion set was connected to a measuring ruler marked in centimeters. While measurements were conducted, the urine bag was taken off and the hose of an infusion set was attached, through which 25 mL of sterile saline was injected into the empty urinary bladder, and was then pinched. The measurement was performed with the patient in a horizontal position, comfortably on the back, at the end of the expiratory flow 30–60 s after the injection solution. The zero point was at the level of the middle axillary line at the iliac crest. The values obtained were expressed in mmHg, using a correction factor (1 mmHg = 1.36 cmH₂O). Measurements were performed every 12 h until discharge from the ICU.

Mean values of IAP were compared with studied variables (age, sex, body mass index (BMI), Acute Physiology, Age and Chronic Health Evaluation (APACHE) II score on admission, Sequential Organ Failure Assessment (SOFA) score, Ranson's criteria on admission and after 48 h, abdominal perfusion pressure (APP), urea, lactate, number of days of treatment).

2.1. Statistical analysis

Analysis of the survey data was performed using SPSS 19.0 for Windows (IBM Corp., Armonk, NY, USA). The description of numerical characteristics was performed by methods of descriptive statistics: arithmetic mean and a measure of variability (standard deviation), as well as the minimum and maximum values of the characteristics. In analyzing the results, we used the Student t-test for comparison between means of two unequal samples. To analyze the relationship between variables, we used the Pearson correlation coefficient for homogeneous data. Simple linear regression was performed for each independent variable that influenced the dependent variable for IAP groups with high and normal values of IAP for deceased patients. For the prediction of mortality we used a receiver operating characteristics (ROC) curve, which shows the area under the curve (AUC) for the mean values of IAP, APACHE II score, APP, and Ranson's criteria on admission and after 48 h. In all applied statistical methods, the level of significance was $P < 0.05$, and the level of high statistical significance was $P < 0.01$.

3. Results

This study included 50 patients with acute pancreatitis, including 14 with normal values of IAP and 36 with high levels of IAP. Mortality in patients with acute pancreatitis in this study was 40%. In the group of patients with normal pressure the mortality rate was 35.7% (5 deaths out of 14 patients), whereas in the group with high levels of IAP the mortality rate was 41.6% (15 deaths out of 36 patients). Comparing the IAP and treatment outcomes, we have proved that there are statistically highly significant differences ($P = 0.012$).

Examined variables of patients with IAH and patients with normal IAP values are shown in Tables 1 and 2.

Regression analysis showed a significant association between the mean values of IAP and the examined variables in groups of deceased patients with normal values of IAP and IAH (Tables 3 and 4). In the group of deceased patients with normal IAP, regression analysis did not confirm a significant strength of the relation between the tested variables of urea or Ranson's criteria on admission and after 48 h. In the group of deceased patients with IAH the examined variables of age, sex, hematocrit, and urea did not show statistical significance.

The ROC curve and AUC is shown in the Figure for prediction of mortality. The Figure shows the AUC for the mean values of IAP of 0.220 mmHg (95% CI: 0.411–0.985), APACHE II score of 0.897 (95% CI: 0.131–0.828), APP of 0.302 mmHg (95% CI: 0.342–0.991), and Ranson's criteria for admission of 0.197 (95% CI: 0.410–1.00) and after 48 h of 0.045 (95% CI: 0.603–1.00).

Table 1. Examined variables of patients with IAH.

| Variables | Deceased patients (n=15) | Surviving patients (n=21) | P-value |
|--------------------------|--------------------------|---------------------------|---------|
| IAP (mmHg) | 18.33 ± 4.18 | 14.42 ± 2.69 | 0.001 |
| APACHE II score | 27.5 ± 6.13 | 18.3 ± 4.19 | 0.016 |
| APP (mmHg) | 75.4 ± 16.1 | 82.7 ± 15.1 | 0.048 |
| Lactate (mmol/L) | 2.3 ± 2.2 | 2.1 ± 1.9 | 0.049 |
| Hematocrit (%) | 18.3 ± 4.19 | 35.3 ± 5.78 | 0.039 |
| Ranson's at admission | 3.0 ± 1.06 | 1.94 ± 0.93 | 0.017 |
| Ranson's at 48 h | 3.8 ± 0.94 | 2.7 ± 1.12 | 0.010 |
| SOFA score | 18.3 ± 4.19 | 16.3 ± 2.58 | 0.014 |
| BMI (kg/m ²) | 18.3 ± 4.19 | 24.2 ± 2.62 | 0.012 |

IAP: Intraabdominal pressure; APACHE II: Acute Physiology, Age and Chronic Health Evaluation; APP: abdominal perfusion pressure; SOFA: Sequential Organ Failure Assessment; BMI: body mass index.

Table 2. Examined variables of patients with normal IAP values.

| Variables | Deceased patients (n = 5) | Surviving patients (n = 9) | P-value |
|--------------------------|---------------------------|----------------------------|---------|
| IAP (mmHg) | 10.0 ± 1.00 | 7.02 ± 2.12 | 0.01 |
| APACHE II score | 23.0 ± 8.37 | 10.0 ± 1.0 | 0.014 |
| Ranson's at 48 h | 4.4 ± 0.55 | 2.6 ± 1.13 | 0.033 |
| SOFA score | 16.4 ± 1.82 | 12.3 ± 1.5 | 0.022 |
| BMI (kg/m ²) | 25.9 ± 3.7 | 22.2 ± 3.46 | 0.016 |

IAP: Intraabdominal pressure; APACHE II: Acute Physiology, Age and Chronic Health Evaluation; SOFA: Sequential Organ Failure Assessment; BMI: body mass index.

Table 3. Correlations between the mean values of IAP and the studied variables in deceased patients with normal IAP values.

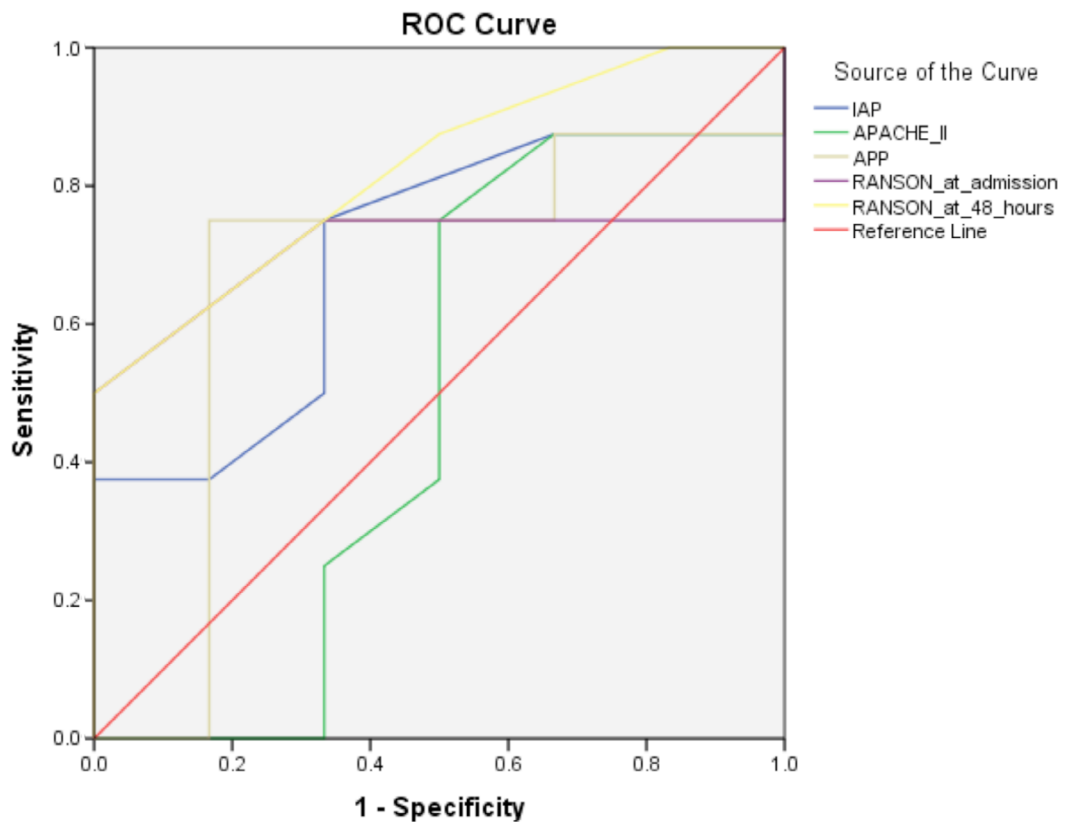
| Variables | R | t | 95% CI | P-value |
|--------------------------|-------|--------|---------------|---------|
| Age (years) | 0.941 | -4.8 | -27.44; -5.56 | 0.017 |
| Sex | 0.45 | -0.889 | -1.14; -0.645 | 0.44 |
| BMI (kg/m ²) | 0.582 | -1.24 | -7.75; -3.405 | 0.303 |
| Lactate (mmol/L) | 0.696 | 1.67 | -0.23; -0.753 | 0.023 |
| Hematocrit (%) | 0.616 | 1.35 | -5.53; -13.75 | 0.509 |
| APACHE II score | 0.807 | -2.365 | -15.83; -2.33 | 0.421 |
| SOFA score | 0.638 | 0.241 | -3.05; -3.55 | 0.951 |

BMI: Body mass index; APACHE II: Acute Physiology, Age and Chronic Health Evaluation; SOFA: Sequential Organ Failure Assessment.

Table 4. Correlations between mean values of IAP and the studied variables in deceased patients with IAH.

| Variables | R | t | 95% CI | P-value |
|--------------------------|--------|--------|----------------|---------|
| BMI (kg/m ²) | 0.565 | -0.23 | -0.414; -0.334 | 0.414 |
| Lactate (mmol/L) | -0.58 | 2.57 | 0.043; -0.483 | 0.023 |
| APP (mmHg) | -0.524 | -2.220 | -6.19; -0.84 | 0.045 |
| APACHE II score | 0.625 | 0.831 | -0.52; -1.18 | 0.421 |
| SOFA score | -0.518 | -0.064 | -0.38; -0.359 | 0.950 |
| Ranson's at admission | 0.699 | 1.569 | -0.038; -0.242 | 0.14 |
| Ranson's at 48 h | 0.744 | 1.323 | -0.049; -0.204 | 0.209 |

BMI: Body mass index; APP: abdominal perfusion pressure; APACHE II: Acute Physiology, Age and Chronic Health Evaluation; SOFA: Sequential Organ Failure Assessment.



Diagonal segments are produced by ties.

Figure. Receiver operating curve (ROC) for IAP, APACHE II, APP, and Ranson's criteria at admission and at 48 h.

4. Discussion

According to the latest guidelines (2,6,7,10,14,16,18,19) for acute pancreatitis it is noted that in recent years admission to hospitals for acute pancreatitis has increased from 40

to 70 per 100,000. Mortality due to acute pancreatitis still remains high. Given that a large number of patients with acute pancreatitis, as part of the pathophysiological mechanisms and the development of complications,

develop IAH, with this study we tried to examine how IAP contributes to the mortality rate in patients with acute pancreatitis.

Mortality in patients with acute pancreatitis in this study was 40%. In the group of patients with normal IAP the mortality rate was 35.7%, whereas in the group with IAH the mortality rate was 41.6%. The incidence of IAH was 72%, which is slightly more than in the last WSACS report (15). According to Keskinen et al. (16), the incidence of IAH was 84% and the mortality rate with increasing IAP ranged from 10% to 50%. In a recent study conducted by Kim et al. (17), which examined the impact of IAP on the mortality rate in critically ill patients in the ICU, it was concluded that the IAP had no effect on the increase in mortality rates if they cured the underlying condition for which the patient was admitted to the ICU, because increased IAP appeared as a consequence of the primary disease. The incidence of IAH in that study was 38%, which is much lower than in our study, but it also differs because it involves critically ill patients, especially patients with acute pancreatitis. Another study, which dealt with patients with acute pancreatitis, showed that the value of IAP on admission is an equal indicator of mortality as the SOFA score but a better indicator compared to the APACHE II score; mortality in that study was 53% (18).

Deceased patients in the study group with IAH had significantly higher IAP, APACHE II score, APP, lactate levels, hematocrit, Ranson's criteria on admission and after 48 h, SOFA score, and BMI compared to surviving patients of the same group, while the average age, number of days of treatment, and value of urea did not show statistical significance. In contrast to them, patients with normal IAP had a significant association only with the IAP, Ranson's criteria after 48 h, and SOFA score, while the APP, lactate levels, hematocrit, Ranson's criteria on admission, average age, number of days of treatment, and value of urea did not show statistical significance in this group. In this study it was observed that there was a statistically significant difference between the values of IAP of deceased patients with normal IAP and IAH (10.0 ± 1.00 mmHg vs. 18.33 ± 4.18 mmHg, $P = 0.035$).

In the study group of patients, there was an increase in IAP, thereby increasing Ranson's criteria after 48 h and SOFA score, but this study shows the mean value of the IAP and not the maximum value. Patients who died in this group had a mean value of marginal IAP, which were separated from the group with IAH. Given that there was

a significant correlation with other parameters, IAP had an impact.

Both groups of deceased patients had the same increased variables, and those variables distinguished them from patients who survived. These variables could affect the mortality rate, because it is exactly these scoring systems used to assess the probability of death. In other studies, it has been proven that patients with acute pancreatitis had higher values of IAP, APACHE II score, SOFA score, lactate, and BMI (10,15,19–21).

Deceased patients with IAH had a significant correlation of IAP with the values of APACHE II score, Ranson's criteria on admission and after 48 h, SOFA score, APP, lactate levels, and BMI, which was confirmed by linear regression analysis. Given that the high statistical significance of IAP with these variables differentiated them from surviving patients, we can say that IAP contributed to the increase in mortality rates. Deceased patients with normal IAP had lower values of APACHE II score on admission, they were younger and had lower BMI, and Ranson's criteria were not relevant, but the SOFA score was significantly associated with IAP.

At the WSACS conference in 2013 (10), there was debate about whether APP is a better indicator of visceral perfusion and restitution compared to the mean arterial pressure. APP had a significant association with IAP in the deceased group of patients with IAH. Those patients had reduced values of APP and increased IAP values while APP was declining. Deceased patients with normal IAP showed no correlation with APP, but they also had lower APACHE II scores and other variables examined, and therefore it had no impact on the outcome. In one recent study, it was shown that IAP is a better indicator of the severity of acute pancreatitis compared to APP (22).

The significance of this study lays in the following: it was conducted according to the WSACS recommendations with patients with acute pancreatitis who were immediately admitted to the ICU, the limit for the possible occurrence of IAH is the value of IAP ≥ 12 mmHg, and the volume of fluid used for performing the method is 25 mL. The limitation of the study is the small number of subjects.

Taking into account all these factors, patients with acute pancreatitis who develop IAH have a statistically high correlation with organ dysfunction because the IAH itself can worsen and lead to death. IAP has an impact on the increase in mortality rates in patients with acute pancreatitis.

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