ID Design Press, Skopje, Republic of Macedonia Open Access Macedonian Journal of Medical Sciences. https://doi.org/10.3889/oamjms.2019.228 eISSN: 1857-9655 *Clinical Science* 



# Abdominal Compartment Syndrome in Critically III Patients

Mohsen Sadeghi<sup>1</sup>, Arda Kiani<sup>2</sup>, Kambiz Sheikhy<sup>3</sup>, Kimia Taghavi<sup>1</sup>, Mohsen Farrokhpour<sup>1</sup>, Atefeh Abedini<sup>1\*</sup>

<sup>1</sup>Chronic Respiratory Diseases Research Center, National Research Institute of Tuberculosis and Lung Diseases (NRITLD), Shahid Beheshti University of Medical Sciences, Tehran, Iran; <sup>2</sup>Tracheal Diseases Research Center, National Research Institute of Tuberculosis and Lung Diseases (NRITLD), Shahid Beheshti University of Medical Sciences, Tehran, Iran; <sup>3</sup>Lung Transplantation Research Center, National Research Institute of Tuberculosis and Lung Disease (NRITLD), Shahid Beheshti University of Medical Sciences, Tehran, Iran

#### Abstract

Citation: Sadeghi M, Kiani A, Sheikh K, Taghavi K, Farrokhpour M, Abedini A. Abdominal Compartment Syndrome in Critically III Patients. Open Access Maced J Med Sci. https://doi.org/10.3889/oamjms.2019.228

Keywords: Intra-Abdominal Hypertension; Incidence; Critical Illness; Intensive Care Units; Prognosis; Glasgow Coma Scale; Systemic Inflammatory Response Syndrome; APACHE

"Correspondence: Atefeh Abedini. Chronic Respiratory Diseases Research Center, National Research Institute of Tuberculosis and Lung Diseases (NRITLD), Shahid Beheshii University of Medical Sciences, Tehran, Iran. E-

mail: dr.abedini110@sbmu.ac.ir Received: 27-Jan-2019; Revised: 03-Apr-2019; Accepted: 04-Apr-2019; Online first: 13-Apr-2019

Copyright: © 2019 Mohsen Sadeghi, Arda Kiani, Kambiz Sheikhy, Kimia Taghavi, Mohsen Farrokhpour, Atefeh Abedini. This is an open-access article distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License (CC BY-NC 4.0) Funding: This research did not receive any financial

support Competing Interests: The authors have declared that no competing interests exist **BACKGROUND:** Abdominal compartment syndrome patients suffer severe obstacles such as kidney failure and shock. To evade further complications, identifying the abdominal compartment syndrome (ACS) and Intraabdominal hypertension (IAH), in critically ill individuals and hospitalised in the intensive care unit (ICU) is obligated.

AIM: The current study intended to study the abdominal compartment syndrome and the concomitant risk factors among hospitalised patients in ICU, by using the Intra-abdominal pressure test.

**MATERIAL AND METHODS:** One hundred and twenty-five hospitalised patients at ICU entered the current survey. Abdominal pressure was measured by standard intravesical technique. The SPSS 21 analysed the preoperative and intraoperative factors such as demographic records and comorbidities.

**RESULTS:** Seventy-three (58.4%) participants were males, and 52 (41.6%) were women in the mean age of 55.1  $\pm$  18.3 years. Eighty-nine patients (71.2%) showed normal intra-abdominal pressure since 31 patients (24.8%), and 5 patients (4%) developed IAH and ACS. The intra-abdominal pressure (IAP) applied to Glasgow Coma Scale (GCS), Acute Physiology, shock, Systemic Inflammatory Response Syndrome (SIRS), central venous oxygen saturation and Chronic Health Evaluation (APACHE II) score (P < 0.05). Patients with high IAP have shown a higher mortality frequency, compared to others (P < 0.05).

**CONCLUSION:** Current findings showed a correlation between IAP hospitalised patients in ICU and shock, SIRS, APACHE II, central venous oxygen saturation and GCS. Intra-abdominal pressure test, as a valuable prognosis test for the abdominal compartment syndrome (ACS) and Intra-abdominal hypertension (IAH), may offer better results when added to the routine medical checkup of ICU patients.

## Introduction

Despite improved survival reports following the laparotomy method, the abdominal compartment syndrome (ACS) is quite an expanding matter [1]. Increased intra-abdominal pressure (IAP) points to intra-abdominal hypertension (IAH) that influences the body function in critically ill patients and cause abdominal compartment syndrome (ACS). As stated by the World Society of the Abdominal Compartment Syndrome (WSACS), IAH is defined above 12 mmHg intra-abdominal pressure (IAP). The low-grade IAH is two types. IAP 12–15 mmHg specifies the IAH Grade I and IAP 16–20 mmHg specifies the IAH Grade II [2]. ACS is also defined as an IAP > 20 mmHg with proven signs of failure in new organs, for instance, kidney failure or increasing complications in ventilation [3]. ACS is an intra-abdominal pathology proven from an extra-abdominal source. IAH and ACS happen following reduced abdominal wall compliance and/or enlarged intra-abdominal capacities [2].

The importance of IAP detection in susceptible patients to an IAH and ACS is well known. Intravesical pressure (IVP) measurement is now the gold standard for indirect diagnosis of IAP [4]. IAH is reported in 32.1% of critically ill patients. IAH is also a predictor for mortality and is seen in 30-50% of intensive care hospitalised patients [5].

IAH and ACS develop in critically ill patients, caused by several risk factors such as abdominal

surgery, hypoalbuminemia, trauma, hypoalbuminemia, and high-volume resuscitation [6].

Proper diagnostic techniques can accelerate future researches in evaluating the pathophysiological mechanism of IAH/ACS [5]. Several types of research have been conducted to increase the accuracy of the diagnosis of IAH/ACS, such as the new porcine model of ACS, which was introduced by Shah et al., [7].

Interestingly, there have been several reports on the growing prevalence of ACS reports in the intensive care unit (ICU) and medical ICUs (10.5%) [8]. Therefore, a rapid and right tool to rapid and exact determining the IAH is trustworthy [9].

Based on the literature, IAH characterises a severe disorder with a high incidence in ICU (18%-81%) [6]. However, because of the insidious origination and nonspecific signs of IAH, it has not been accurately studied. Hence, the object of the current study was to test abdominal compartment syndrome and the concomitant risk factors among hospitalised patients in ICU, using the Intra-abdominal pressure test.

## Material and Methods

#### Study design

Patients who referred to the ICU of a tertiary hospital in Tehran because of surgical or non-surgical problems, included in the current study. The mean age for the patients would be > 18 years old. Demographic indexes including age, sex, Glasgow Coma Scale (GCS), BMI, AQA Acute Physiology and Chronic Health Evaluation score (APACHE II) were collected. The exclusion criteria comprised the presence urinary of contraindications for catheterisation (especially in trauma patients) and age < 18. One hundred and twenty-five patients in ICU joined current study [10]. In the current study, disease severity was measured based on APACHE II score (calculated at the time of IAP measurement) and a 12 routine physiologic point score, age, and history of health status that shows the severity of the disease. We considered an increasing score (range 0 to 71) as a risk case of hospital death [11].

#### Intra-abdominal pressure

The abdominal pressure was measured using the bladder catheter by a standard intravesical method. The catheter clamped and then using a portal aspiration, 25 ccs of hygienic saline was inoculated to the bladder via an attached catheter by an 18-gauge needle to the pressure manometer. Zero of manometer located on mid-maxillary line at the level of the umbilicus. IAH was recorded in Supine and end duration [10]. IAP of  $\geq$  12 mmHg was determined as hypertension. Also, the IAP of  $\geq$  12 mmHg + intraabdominal dysfunctions with/without APP < 60 mmHg were used as ACS. The participants completed and signed informed consent. Each participant was informed about the benefits of the study and personal information kept as secret. The Ethical Committee received.

#### Data and analysis

Statistical analyses performed by Statistical Package for the Social Sciences (SPSS) (ver. 22.0; SPSS Inc. Chicago, IL, USA) software. The nonparametric Mann-Whitney U test determined the between treatments comparisons. Also, a Student's ttest calculated the differences in the mean, considering a p-value of < 0.05 meant for a significant value.

### Results

Tables 1 show demographic data of the study subjects.

#### Table 1: Anthropometric indexes of participants

	Male	Female
Sex	73 (58.4%)	52 (41.6%)
Age	54.5 ± 18.8	56 ± 17.7

Table 2 shows the body mass index of the study subjects.

#### Table 2: Body mass index of the study subjects

Underweight	Normal	Overweight	Obese
4.8%	26.4%	52%	16.8%

As seen in Table 3, 73 patients (58.4%) referred to the hospital for surgery while 52 individuals (41.6%) hospitalised for medical problems.

#### Table 3: The cause of hospitalising in the ICU

Diagnosis	Frequency	Per cent	Diagnosis	Frequency	Per cent
Abdominal mass	2	1.6	Cervix cancer	1	0.8
AKI	3	2.4	CHF	2	1.6
Amputation	2	1.6	Cholangitis	1	0.8
Ascitis	2	1.6	Cholecystectomy	2	1.6
Brain tumor	7	5.6	Cirrhosis	1	0.8
Bronchiectasis	1	0.8	Colectomy	3	2.4
Cerebral aneurysm	1	0.8	COPD	3	2.4
CVA	7	5.6	Electrical injury	1	0.8
DAI	4	3.2	Empyema	1	0.8
DKA	2	1.6	Encephalitis	1	0.8
EDH	1	0.8	Femur FX	6	4.8
Gastric cancer	2	1.6	Intoxication	1	0.8
GIB	3	2.4	LOC	2	1.6
ICH	3	2.4	MI	1	0.8
Intestinal Obstruction	2	1.6	MT	13	10.4
Myasthenia Gravis	2	1.6	Pelvic FX	5	4.0
OSA, OHS	1	0.8	Peritonitis	6	4.8
Ovarian cancer	1	0.8	Pneumonia	7	5.6
Pancreatitis	3	2.4	PTE	2	1.6
Rectal cancer	1	0.8	Spondylodiscitis	1	0.8
SAH	4	3.2	Status Épilepticus	2	1.6
SDH	3	2.4	TTP	1	.8
SLE	1	0.8	Urosepsis	2	1.6
Splenectomy	1	0.8	Vasculitis	1	0.8

Acute Kidney Injury (AKI), Cerebrovascular accident (CVA), Diffuse axonal injury (DAI), Diabetic ketoacidosis (DKA), Subdural hematoma (SDH), chronic obstructive pulmonary disease COPD, Pulmonarv Thromboendarterectomy (PTE). Thrombotic Thrombocytopenic Purpura (TTP). Thirtynine patients (31.2%) had trauma while 86 persons (68.8%) had no earlier trauma. Also, only 34 of the patients (27.2%) had a history of bone fracture. Thirtynine patients had normal ventilation (31.2%) while 71 patients (56.8%) used a mechanical ventilator and the 15 of them (12%) had the tracheostomy. Table 4 presents the result of blood products transfused for the participants. Those with IAP, IAH, and ACS received  $1.3 \pm 4$ ,  $2.3 \pm 2.5$  and  $7.6 \pm 3.2$  units of blood products (P < 0.001).

#### Table 4: Blood analysis report in participants

Blood products	Frequency	Per cent
NO	64	51.2
PC	41	32.8
FFP	2	1.6
PLT	3	2.4
CRYO	2	1.6
PC, FFP	9	7.2
PC, FFP, PLT	3	2.4
PC, FFP, PLT	1	0.8
NO: No blood Products, PC: Packed cell, FFF	: Fresh frozen plasma, PLT: Platelet	, CRYO: Cryoprecipitate.

As stated by the records, 11 patients had a shock, and 92 experienced none shock. Also, 11 patients among 22 SIRS-positive patients had a shock. At the first visit, patients in the ICU underwent for monitoring the IAH, IAP, and ACS. Table 5 presents the data.

Table 5: The per cent of IAH, IAP and ACS and APACHE II and time duration of hospitalising of the patient in the ICU

	Per cent of patients	APACHE II >	20 APACHE II < 20	the time duration
				of hospitalise (day)
IAH	9.52	81	8	14 ± 9.7
IAP	90.48	9	22	18.5 ± 5.9
ACS	-	-	5	29 ± 6
ACS: Abdomina hypertension.	al compartment syndrome,	IAP: Increased	intra-abdominal pressure,	IAH: Intra-abdominal

Table 6 displays the correlation between disease history and IAP in the patients. Based on the results, the concomitant disease had no significant correlation with the IAP (P = 0.09).

Table 6: The correlation between disease history and IAP in the patients

Comorbidity		IAP		Total
	Normal	IAH	ACS	
AF	3	1	0	4
Asthma	2	0	0	2
Cerebral Palsy, Seizure	0	1	0	1
CHF	1	0	0	1
Colon cancer	1	1	0	2
COPD	2	0	0	2
DM	7	5	0	12
DM, Cirrhosis, Rectum adenocarcinoma	0	1	0	1
DM, HLP, Hypothyroidism	1	0	0	1
HBV	1	1	0	2
HLP	1	2	0	3
HTN	7	3	0	10
HTN, DM	5	3	1	9
HTN, DM, CVA	0	1	0	1
HTN, DM, HLP	0	1	0	1
HTN, HLP, BPH, CKD, AF	2	0	0	2
HTN, HLP, DM, IHD	5	0	3	8
IHD	2	0	0	2
MVR, AF	1	0	0	1
NO	45	9	1	55
Osteporosis	1	0	0	1
Polyp	0	1	0	1
PUD	2	1	0	3
Total	89	31	5	125
F: Atrial fibrillation, CHF: Congestive heart fa	ilure DM <sup>.</sup> Diabete	s mellitus	HLP: hyperlip	idemia HB

AF: Atria infinitation, CHF: Congestive near failure, DM: Diabetes mellitus, HLF: hyperipidemia, HBV: Hepatitis B, HTN: Hypertension, BPH: Benign prostatic hyperplasia, CKD: Chronic kidney disease, IHD: Ischemic heart disease, MVR: Mitral Valve Repair, Cerebrovascular accident (CVA), NO: No blood Products. As shown in Figure 1, the IAP of patients with trauma was  $10.04 \pm 5.08$  mmHg while it was  $9.0504 \pm 5.08$  mmHg in non-traumatic patients (P = 0.19). Also, the IAH and ACS incidences were 30 and 2% in trauma patients.

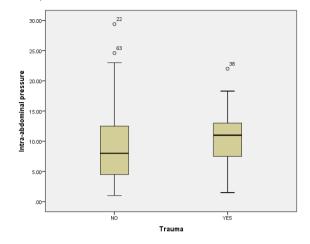


Figure 1: The IAP in patients with or without trauma (IAP: Increased intra-abdominal pressure)

The mean intra-abdominal pressure was  $8.3 \pm 5$  mmHg in non-ventilated patients and  $9.4 \pm 5.6$  mmHg in patients with tracheal intubation. Patients with tracheostomy had an IAP of  $11.3 \pm 6.3$  mmHg (P = 0.15).

There was no significant correlation between positive end-expiratory pressure (PEEP) and intraabdominal pressure, so that in patients without PEEP, the mean intra-abdominal pressure was  $8.4 \pm 4.9$  mm. In PEEP, three centimetres of intra-abdominal pressure was  $4.4 \pm 7.9$  mm Hg, and at a pressure of 5 cm water, the pressure inside the abdomen was  $5.9 \pm$ 9.9 mm Hg and at 7 cm water pressure, intraabdominal pressure was  $4.5 \pm 7.7$  mm Hg (P = 0.15).

Received fluid in patients with normal intraabdominal pressure was  $1.6 \pm 2.2$  litres. While the significant different received fluid volume in patients with IAH and patients with ACS was  $1.5 \pm 2.4$  litres and  $0.4 \pm 4.3$  litres respectively (P = 0.002) (Figure 2).

Of the patients with normal intra-abdominal pressure, six patients did not take fluids. While, 33, 16, one, six, 22, one, three and one patients took normal saline, Dextrose saline, 5% dextrose, half-saline, normal saline/dextrose saline, normal saline/5% dextrose, normal saline/half saline, normal saline/5% dextrose, monophagous in turn.

Ten of patients with IAH took normal saline, five took dextrose saline, three took the half-saline, 10 took normal/ dextrose saline, and one patient took normal/half-saline/2% dextrose water/5% amino fusion. Three and two of patients with ACS took normal saline and normal saline/dextrose saline (P = 0.89).

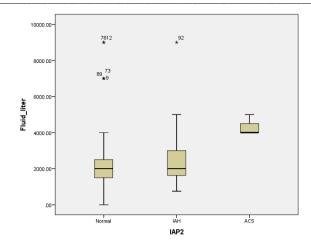


Figure 2: Volume of daily IV fluid in patients (IV: Intravenous)

A significant correlation was documented between homeostasis disorders and IAP in patients (P < 0.001).

 Table 7: Correlation between intra-abdominal pressure and homeostasis disorders in patients

		IAP		Total
Hemostasis	Normal	IAH	ACS	
Normal	53	11	0	64
Abnormal PT	14	11	0	25
Abnormal PTT	7	1	0	8
Thrombocytopenia	1	6	2	22
Abnormal PT, PTT	0	2	1	3
PT and Low Plt	1	0	1	2
PT, PTT, Low Plt	0	0	1	1
Total	89	31	5	125

PTT: Partial Thromboplastin Time, PT: Prothrombin Time, PLT: Platelets.

There was no significant correlation between culture types in patients with intra-abdominal pressure (Figure 3) (P = 0.07).

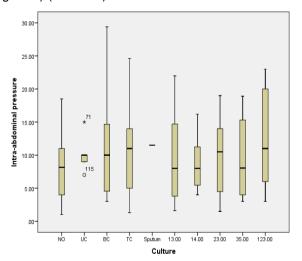


Figure 3: Correlation between culture types in patients with intraabdominal pressure

In the current study, 13% of the patients with normal intracranial pressure (n = 11) had acidosis, 12% of patients with IAH (n = 4) exhibited acidosis, and 100% of patients with ACS (n = 5) had acidosis (P

= 0.001). As presented in Table 8, there was no significant correlation between the type of antibiotic received and intra-abdominal pressure (P = 0.46).

#### Table 8: Correlation between antibiotic administration and IAP

		IAP		Total
Antibiotics	Normal IAP	IAH	ACS	-
Acyclovir	0	1	0	1
Amikacin, Meropenem	2	0	0	2
Cefazoline	2	1	0	3
Cefazoline, Ceftriaxone, Clindamycin	1	0	0	1
Cefotaxime	0	1	0	1
Ceftriaxone, Metronidazole	1	1	0	2
Ciprofloxacine	4	3	0	7
Clindamycin	1	0	0	1
Colistin, Meropenem	3	1	0	4
Colistin, Tazocin	2	0	0	2
Gentamycin	3	1	0	4
Gentamycin, Ciprofloxacin	0	1	0	1
Imipenem, Targocid, Metronidazole	3	2	2	7
Meropenem	6	3	0	9
Metronidazole	5	0	0	5
Metronidazole, Ciprofloxacin	1	0	0	1
NO	32	11	0	43
Vancomycin, Ciprofloxacin, Tazocin	1	0	0	1
Vancomycin, Imipenem, Ciprofloxacin	3	0	0	3
Vancomycin, Meropenem	6	3	0	9
Vancomycin, Meropenem, Ampicillin	1	0	0	1
Vancomycin, Meropenem, Ciprofloxacin	8	1	2	11
Vancomycin, Meropenem, Ciprofloxacin,	1	0	0	1
Colistin, Tazocin				
Vancomycin, Meropenem, Colistin	2	0	0	2
Vancomycin, Meropenem, Metronidazole	1	1	1	3
Total	89	31	5	125

The SCVO2 in normal IAP patients was 76.8%  $\pm$  8.5 while in IAH and ACS patients were 74.6  $\pm$  1 and 59.8%  $\pm$  0.8 (P = 0.001). Table 9 displays the correlation between IAP and the identification of the disease. According to the results, a significant correlation detected between IAP and diagnosis of the disease (P = 0.02).

Table 9: Correlation between IAI	P and diagnosis of the disease
----------------------------------	--------------------------------

	Normal IAP	IAH	ACS		Normal IAP	IAH	ACS
Abdominal	1	0	1	Bronchiectasis	1	0	0
mass							
AKI	0	3	0	Cerebral	1	0	0
				aneurysm			
Amputation	2	0	0	Cervix cancer	1	0	0
Ascitis	1	1	0	CHF	2	0	0
Brain tumor	6	1	0	Cholangitis	0	0	1
Cholecystectom	2	0	0	Colectomy	1	2	0
у							
Cirrhosis	1	0	0	COPD	3	0	0
CVA	6	1	0	Empyema	1	0	0
DAI	4	0	0	Encephalitis	1	0	0
DKA	2	0	0	Femur FX	2	4	0
EDH	1	0	0	Gastric cancer	2	0	0
Electrical injury	1	0	0	GIB	3	0	0
ICH	3	0	0	MT	8	4	1
Intestinal	0	2	0	Myasteni	2	0	0
Obstruction				Gravis			
Intoxication	1	0	0	OSA,OHS	0	1	0
LOC	2	0	0	Ovarian	0	1	0
				cancer			
MI	1	0	0	Pancreatitis	0	3	0
Pelvic FX	2	3	0	SAH	4	0	0
Peritonitis	1	4	1	SDH	2	1	0
Pneumonia	7	0	0	SLE	1	0	0
PTE	2	0	0	Splenectomy	1	0	0
Rectal cancer	0	0	1	Spondylodisciti	1	0	0
				S			
Status	2	0	0	TTP	1	0	0
Epilepticus							
Urosepsis	2	0	0	Vasculitis	1	0	0
ICH: Intracrania				f Consciousness	s, MI: My	ocardial	Infarction,

PTE: Pulmonary ThromboEmbolism.

As observed in figure 4, the IAP in normal patients detected in 89 patients and among them, 84 survived, but 5 passed away. Furthermore, in IAH patients, 21 survived, and 10 expired. Entire ACS

patients died (P = 0.001). The mean IAP of expired patients was  $15.5 \pm 6.7$  mmHg while in endured patients the IAP was  $8.1 \pm 4.5$  mmHg.

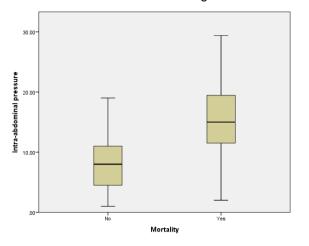


Figure 4: IAP pressure in expired or survived patients (IAP: Increased intra-abdominal pressure)

## Discussion

One of the main challenges in Iran is managing the patients with an open abdomen admitted to ICUs. The open abdomen stays at high risk of several complications and organising a trusty closure starts right after hernia. The qualified management in ICU wards controls the feasibility of closure an open abdomen. For that reason, further studies are favourable on the modality of reaching this target. Therefore, the current study intended to test the frequency of the abdominal compartment syndrome and the concomitant risk factors among hospitalised patients in ICU, using the Intra-abdominal pressure test.

Several approaches diminished IAH and to avoid development to the ACS [12]. IAH affects not only abdominal organs but also several organ organisations. It influences lungs, hemodynamics, and cerebral perfusion systems. IAH is the main determining compliance of the mechanically ventilated patients [13]. As observed in this study, shock, SIRS, APACHE II, central venous oxygen saturation and GCS were associated with the IAP. The mortality frequency was higher in patients with advanced IAP. Our findings suggest IAP measurement is necessary for ICU patients to find their prognosis and right intervention. IAH is a source of organ dysfunctions, and ACS is a catastrophic disruption of body physiology that needs urgent treatments [14].

In this study, 39 patients (31.2%) had trauma while 86 persons (68.8%) had no earlier trauma. In this study, the IAP of the patients with normal

ventilation was 8.3  $\pm$  5 mmHg. As observed in this study, the IAP for patients with the endotracheal tube and tracheostomy were 9.4  $\pm$  5.6 and 11.3  $\pm$  6.3 mmHg (P = 0.15).

Nowadays IAP is performed as a safe, reliable and reproducible technique [14]. IAH and ACS left significant influences on blood factors of the participants in the current study. To avoid overloading of massive fluid, starting resuscitation is mandatory [15]. IAH takes place in about 50% of critical care patients, 32.1% of which showed IAH and 4.2% develop ACS within the first day of admitting to ICU [16].

Patients with ACS succeed with pharmacological, practical and medical trials [16]. Significant differences detected between antibiotic administration and IAP in the current observation was comparable with this. IAH and ACS are serious threats in ill patients. Nurses in the ICU train to diagnose the IAH and ACS and do the right interventions.

Nursing training has to focus on evidencebased training strategies. Nurses should run standard care dealing with patients at risk of IAH and ACS [17]. Hence, prompt screening is essential to find patients who may show IAH and ACS [18]. IAH links to elevated SOFA scores, high APACHE II, high APACHE III, a further need for mechanical ventilation and insufficient PaO2: FiO2 ratios at admission time. Longer durations of the need for mechanical ventilation and lengths of stay at ICUs in such patients reaffirm the pathophysiological damages of raised ICP [19].

Also, our result was matching the reports that shock, SIRS, APACHE II, central venous oxygen saturation and GCS associated with IAP. The mechanical ventilation is independent predisposing to develop IAH with applying PEEP [20]. Based on the observation in the current study, we think IAP measure in ICU patients may help to define a proper prognosis and complete the intervention. We think further research needed to find new methods for direct investigation of IAH in the patients in ICU.

In conclusion, the pathological features of IAH and ACS are appropriate markers to report systemic disorders and mortality in the ICU. Screening the IAH to monitor the signs of ACS is an economical and valuable way can discover complications in the ICU and may adjust treatment outcomes and reduce hospitality overheads.

## Acknowledgement

We thank all the hospital cooperators for helping during this study.

## References

1. Hunt L, Frost SA, Hillman K, Newton PJ, Davidson PM. Management of intra-abdominal hypertension and abdominal compartment syndrome: a review. J Trauma Manag Outcomes. 2014; 8(1):2. <u>https://doi.org/10.1186/1752-2897-8-2</u> PMid:24499574 PMCid:PMC3925290

2. Harhangi BS, Kompanje EJ, Leebeek FW et al. Coagulation disorders after traumatic brain injury. Acta Neurochir. 2008; 150(2):165-75. <u>https://doi.org/10.1007/s00701-007-1475-8</u> PMid:18166989

3. Krinsley JS. Association between hyperglycemia and increased hospital mortality in a heterogeneous population of critically ill patients. Mayo Clin Proc. 2003; 78(12):1471-1478. https://doi.org/10.4065/78.12.1471 PMid:14661676

4. Rossaint R, Bouillon B, Cerny V, Coats TJ, Duranteau J, Fernández-Mondéjar E, etal. Management of bleeding following major trauma: an updated European guideline. Crit Care. 2010; 14(2):R52. <u>https://doi.org/10.1186/cc8943</u> PMid:20370902 PMCid:PMC2887168

5. Gavrilovska-Brzanov A, Nikolova Z, Jankulovski N, Sosolceva M, Taleska G, Mojsova-Mijovska M, et al. Evaluation of the effects of elevated intra-abdominal pressure on the respiratory mechanics in mechanically ventilated patients. Maced J Med Sci. 2013; 6(3):261-5.

6. Herlitz J, Thuresson M, Svensson L, Lindqvist J, Lindahl B, Zedigh C, etal. Factors of importance for patients' decision time in acute coronary syndrome. Int J Cardiol. 2010; 141(3):236-42. https://doi.org/10.1016/j.ijcard.2008.11.176 PMid:19136167

7. Nilsson G, Mooe T, Söderström L, Samuelsson E. Pre-hospital delay in patients with first time myocardial infarction: an observational study in a northern Swedish population. BMC Cardiovasc Disord. 2016; 16(1):93. <u>https://doi.org/10.1186/s12872-016-0271-x</u> PMid:27176816 PMCid:PMC4866271

8. Ladwig KH, Meisinger C, Hymer H, Wolf K, Heier M, von Scheidt W, etal. Sex and age specific time patterns and long term time trends of pre-hospital delay of patients presenting with acute ST-segment elevation myocardial infarction. Int J Cardiol. 2011; 152(3):350-5. <u>https://doi.org/10.1016/j.ijcard.2010.08.003</u> PMid:20813416

9. Braha B, Mahmutaj D, Maxhuni M, Neziri B, Krasniqi S. Correlation of procalcitonin and C-reactive protein with intraabdominal hypertension in intra-abdominal infections: their predictive role in the progress of the disease. Open Access Maced J Med Sci. 2018; 6(3):479.

https://doi.org/10.3889/oamjms.2018.112 PMid:29610604 PMCid:PMC5874369

10. Shafiepour M, Kiani A, Taghavi K, Seifi S, Rezaie MS,

Hashemian SM, etal. A Rare Report of Lung Metastasis of the Common Non-Melanotic Skin Cancer. Tanaffos. 2018; 17(1):62. PMid:30116282 PMCid:PMC6087531

11. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med. 1985; 13(10):818-29. <u>https://doi.org/10.1097/00003246-198510000-</u> 00009 PMid:3928249

12. Morejón CD, Barbeito TO. Effect of mechanical ventilation on intra-abdominal pressure in critically ill patients without other risk factors for abdominal hypertension: an observational multicenter epidemiological study. Ann Intensive Care. 2012; 2(1):S22. https://doi.org/10.1186/2110-5820-2-S1-S22 PMid:23281625 PMCid:PMC3527157

13. Van Hee R. Historical highlights in concept and treatment of abdominal compartment syndrome. Acta Clin Belg. 2007; 62(sup1):9-15.

14. Katsios C, Ye C, Hoad N, Piraino T, Soth M, Cook D. Intraabdominal hypertension in the critically ill: Interrater reliability of bladder pressure measurement. J Crit Care. 2013; 28(5):886-e1. https://doi.org/10.1016/j.jcrc.2013.04.003 PMid:23726386

15. Mahmutaj D, Krasniqi S, Braha B, Limani D, Neziri B. The Predictive Role of Procalcitonin On the Treatment of Intra-Abdominal Infections. Open Access Maced J Med Sci. 2017; 5(7):909. <u>https://doi.org/10.3889/oamjms.2017.194</u> PMid:29362617 PMCid:PMC5771293

16. Mentula P, Leppäniemi A. Prophylactic open abdomen in patients with postoperative intra-abdominal hypertension. Crit Care. 2010; 14(1):111. <u>https://doi.org/10.1186/cc8207</u> PMid:20156323 PMCid:PMC2875490

17. Halstrom S, Price P, Thomson R. Environmental mycobacteria as a cause of human infection. Int J Mycobacteriol. 2015; 4(2):81-91. <u>https://doi.org/10.1016/j.ijmyco.2015.03.002</u> PMid:26972876

18. Strang SG, Van Imhoff DL, Van Lieshout EM, D'Amours SK, Van Waes OJ. Identifying patients at risk for high-grade intraabdominal hypertension following trauma laparotomy. Injury. 2015; 46(5):843-8. <u>https://doi.org/10.1016/j.injury.2014.12.020</u> PMid:25805553

19. Abedini A, Kiani A, Taghavi K, Khalili A, Fard AJ, Fadaizadeh L, etal. High-Frequency Jet Ventilation in Nonintubated Patients. Turk Thorac J. 2018; 19(3):127. https://doi.org/10.5152/TurkThoracJ.2018.17025

20. Jacobs RE, Gu P, Chachoua A. Reactivation of pulmonary tuberculosis during cancer treatment. Int J Mycobacteriol. 2015; 4(4):337-40. <u>https://doi.org/10.1016/j.ijmyco.2015.05.015</u> PMid:26964818