Clinical warning signs for intra-abdominal hypertension in septic shock patients

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Abstract

Background: The latest World Society of the Abdominal Compartment (WSACS) guideline published in 2013 states that risk factors are the most reliable predictors for the diagnosis of intra-abdominal hypertension (IAH) and the bottom line to guide propaedeutic and clinical practice. The objective of this study is to search for clinical, laboratory, and ventilator-associated factors in order to warn medical staff for prompt IAH diagnosis in septic shock patients beyond risk factors simply.

Methods: This is a prospective, observational study, involving all admitted intensive care unit septic shock patients of a single teaching hospital between April and October 2016. All enrolled patients met Sepsis III and Surviving Sepsis Campaign diagnostic criteria. Patients with primary abdominal conditions were excluded, in order to avoid possible bias. Intra-abdominal pressure (IAP) was measured every 6 hours in accordance with WSACS guidelines.

Results: 25 sequential patients were included and followed for 10 days after admission. Median age was 51.13 ± 16.52 years old, 64% male. Pulmonary infection was the most frequent etiology of sepsis, representing 76% of the cases. Elevated IAP correlated with higher central venous pressure (CVP) ($P = 0.0421$); positive end-expiratory pressure (PEEP) ($P = 0.0056$); elevated airway pressure ($P = 0.0015$); accumulated fluid balance ($P = 0.0273$), and elevated SOFA ($P = 0.0393$) in all septic patients. Reduction of acidosis ($P = 0.0096$) and increase of serum bicarbonate ($P = 0.0247$) correlated with lower IAP values.

Conclusions: Elevated CVP, PEEP, SOFA, airway pressure and accumulated fluid balance are correlated with elevated IAP in septic shock patients. Acidosis correction appears to decrease the risk for IAH. Multicentric randomized studies are needed to confirm this hypothesis in a large population.

Key words: intra-abdominal hypertension, intra-abdominal compartment syndrome, sepsis, critical care, intensive care.

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Abdominal compartment syndrome (ACS) is defined as a sustained intra-abdominal pressure > 20 mm Hg (> 3 kPa), with or without an abdominal perfusion pressure < 60 mm Hg (< 8 kPa), that is associated with new organ dysfunction or failure [1]. It leads to decreased organ perfusion, tissue ischemia, organ failure and death if not identified and adequately treated [2].

Although the effects of elevated intra-abdominal pressure have been known since the late 19th century [3], it was not until the early 1980s that the term ACS was first used by Kron *et al*. to describe the pathophysiology of intra-abdominal hypertension (IAH) secondary to aortic aneurysm surgery [4, 5]. In the last two decades clinical awareness of the impact of ACS and IAH in the critical patient has increased [6, 7], probably due to improvements in diagnostic methods and changing paradigms in the treatment of patients sustaining traumatic injuries and the critically ill [8, 9].

There are many known risk factors for developing IAH or ACS, and it is commonly categorized for didactic purposes [3]. However, in the critically ill patient intra-abdominal hypertension is commonly multifactorial and aggravated by some treatments used routinely in intensive care units (ICU), such as aggressive fluid resuscitation and elevated positive end-expiratory pressure (PEEP) [10–13]. In normal

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conditions, the IAP is atmospheric in spontaneously breathing animals [13–17]. In mechanically ventilated patients, the increased intrathoracic pressure is transmitted to the abdominal cavity, aggravating underlying conditions [10, 18].

The objective of this study is to search for clinical, laboratory, and ventilator-associated factors in order to warn medical staff for prompt IAH diagnosis in septic shock patients beyond risk factors simply, in which it may go unnoticed, and to analyze the impact of these parameters on overall mortality.

METHODS

This study was submitted and approved by the Research Ethics Committee under the number 17031113.0.0000.5404 protocol.

This is a prospective, observational study, involving all admitted intensive care unit septic shock patients of a single teaching hospital between April and October 2016. All enrolled patients met Sepsis III [19] and Surviving Sepsis Campaign [20] diagnostic criteria. Patients with primary abdominal conditions were excluded, in order to avoid possible bias. Also excluded in this study were patients admitted to another hospital ward other than the ICU, patients admitted for longer than 48 hours and patients with no urinary catheter placed. In addition, at the moment of bladder catheter removal the patient's participation in the study was withdrawn.

Intra-abdominal pressure (IAP) was measured every 6 hours (AbViser, ConvaTec), in accordance with the World Society of the Abdominal Compartment (WSACS) guideline, at the end of expiration, in a supine position and the zero reference at the level of the medium axillary line. Clinical and ventilatory parameters were also evaluated every 6 hours. Laboratory tests were performed daily during the morning period.

Descriptive variables were summarized in frequencies and percentages and presented the continuous variables as mean or median and standard deviation, depending on the distribution. Regressive analysis was applied to find correlations between variables and look for time influence through generalized estimating equations (GEE). The statistical significance adopted in this study was 5%.

TABLE 1. Inclusion criteria

	Inclusion criteria
A	Septic shock according to SEPSIS-3 Systemic inflammatory response syndrome with infection SBP < 90 mm Hg after 30 mL kg^{-1} of crystalloids + Septic shock according to surviving sepsis q-SOFA ≥ 2 with infection SBP < 90 mm Hg or lactate > 2 mmol L^{-1}
B	Less than 48 hours of hospitalization
C	Admission from the emergency department
D	Absence of abdominal pathologies
E	Presence of bladder catheter
F	18 years of age or older
G	Adequately sedated (RASS –4 or –5) + Not exhibiting abdominal respiratory muscle activity

SBP – systolic blood pressure

RESULTS

Between April and October 2016, a total of 201 consecutive patients were admitted to the Medical ICU with twenty-five consecutive patients included and followed by 10 days after admission with 436 IAP measures. Median age was 51.13 ± 16.52 years old, 64% males. Pulmonary infection was the most frequent primary diagnosis (76%). Other infection sites were blood stream infection (12%), skin infection (8%), and ictero-hemorrhagic fever. Overall mortality in the study was of 52%. 68% (n = 17) of the studied patients developed IAH and 28% (n = 7) developed ACS. Five of these expired, with mortality up to 71.42%.

When looking at clinical parameters and IAH incidence there was found a correlation between high IAP and accumulated fluid balance, central venous pressure, abdominal perfusion pressure and SOFA score, as observed in Table 2.

In the same way, when looking at ventilatory parameters and the presence of IAH, a correlation

TABLE 2. Correlation between clinical parameters and intra-abdominal hypertension

Parameter	Mean ± SD	Intra-abdominal hypertension	
		Correlation, *P*-value	Time influence, *P*-value
Accumulated fluid balance (mL)	10 875 ± 11 008.6	0.0273	0.2281
CVP (cm H_2O)	19.5 ± 8.8	0.0421	0.3440
APP (mm Hg)	80.0 ± 16.2	0.0056	0.2667
Norepinephrine dose (μg kg^{-1} min^{-1})	0.1 ± 0.291	0.8059	0.3126
SOFA score	7.4 ± 2.5	0.0393	0.2069

CVP – central venous pressure, APP – abdominal perfusion pressure

between intra-abdominal pressure and PEEP and Pmax was observed as demonstrated in Table 3.

When comparing arterial blood gas (ABG) values and electrolytes with IAH incidence, a correlation between the consumption of bicarbonate and pH reduction was found and is shown in Table 4.

Comparison of the death outcome group with the survival group was performed. Intra-abdominal pressure, accumulated water balance, SOFA score, central venous pressure and MIP were significantly higher in the death group. On the other hand, pH and serum bicarbonate were significantly lower in the death group, as shown in Table 5.

TABLE 3. Correlation between ventilatory parameters and intra-abdominal hypertension

Parameter	Mean ± SD	Intra-abdominal hypertension	
		Correlation *P*-value	Time influence *P*-value
PEEP (cm H_2O)	9.4 ± 2.9	0.0056	0.2445
P_{max} (cm H_2O)	24.0 ± 4.9	0.0015	0.3105
Tidal volume (mL)	444 ± 116.5	0.1117	0.3586
Respiratory rate (per min)	18.5 ± 4.6	0.9806	0.3628

PEEP – positive end-expiratory pressure

TABLE 4. Correlation between arterial blood gas values and intra-abdominal hypertension

		Intra-abdominal hypertension	
Parameter	Mean ± SD	Correlation *P*-value	Time influence *P*-value
HCO_3^- (mEq L^{-1})	20.1 ± 4.1	0.0247	0.2108
pH	7.34 ± 0.12	0.0421	0.3440
Hb (g dL^{-1})	8.8 ± 1.94	0.5307	0.2921
Ht (%)	27.25 ± 5.83	0.5511	0.2967
Lactate (mmol L^{-1})	1.7 ± 0.08	0.1938	0.2988
K (mEq L^{-1})	4.2 ± 1.24	0.0836	0.2846
Na (mEq L^{-1})	137 ± 5.21	0.6266	0.2651
PaO_2/FiO_2	218 ± 104	0.4613	0.1912

TABLE 5. Comparison between parameters assessed by death outcome

Parameter	Death (*n* = 13) Mean ± SD	Survival (*n* = 12) Mean ± SD	*P*-value
Accumulated fluid balance (mL)	15,165.4 ± 12,719.2	6194.5 ± 6517.1	0.0127
IAP (mm Hg)	14.1 ± 4.2	9.4 ± 2.0	0.0019
APP (mm Hg)	76.8 ± 18.2	83.5 ± 13.6	0.0694
SOFA	8.6 ± 2.6	6.1 ± 1.8	0.0193
CVP (mm Hg)	23.0 ± 9.3	13.8 ± 3.4	0.0029
PEEP (cm H_2O)	10.2 ± 3.3	8.5 ± 2.1	0.165
P_{max} (cm H_2O)	26.3 ± 5.2	21.5 ± 3.2	0.0337
pH	7.3 ± 0.1	7.4 ± 0.1	0.001
HCO_3^- (mmol L^{-1})	17.6 ± 3.1	22.8 ± 3.4	0.0015

IAP – intra-abdominal pressure, APP – abdominal perfusion pressure, CVP – central venous pressure, PEEP – positive end-expiratory pressure

DISCUSSION

This is a prospective observational study, which included all septic shock patients of a single teaching hospital admitted to the ICU. IAP measurement is still neglect, mostly in clinical ill patients in many services, including ours. When informally questioned why, physicians usually answer that they do not believe that IAH could play an important and decisive role in patients' evaluation and for that reason it is not frequently measured. In a recent study Wise *et al*. [21] demonstrated that although most physicians have stated that they were familiar with IAH and ACS, knowledge of the definitions published in the WSACS consensus, measurement and clinical treatment techniques are inconsistent and inadequate.

The rationale here is to evaluate all non-surgical critical patients admitted to this service and measure the IAP in order to observe how frequent IAH is in this population and how it could affect their clinical progress. To avoid bias all patients with any related abdominal diagnosis were excluded. In other words, only patients with septic shock correlated with extra-abdominal ICD were included.

This study showed IAH prevalence similar to worldwide literature, with 68% of the patients presenting with IAH of any grade [22]. In 2004 in the first epidemiological multicentric study on IAH in a mixed population with 58.8% prevalence, 8.2% of them met criteria for ACS [23]. Reintam-Blaser *et al*. [24] investigated 563 patients in one of the largest studies on IAH in mechanically ventilated patients admitted to the ICU. In this study the authors found that 32.3% of IAH and 1.1% of the total patients developed ACS. Many published studies have demonstrated similar outcomes with prevalence of IAH ranging from 30% to 85% and ACS 5% to 50% [25–33].

When looking at patients with extra-abdominal pathologies exclusively, septic patients demonstrated IAH incidences of up to 80% and patients submitted to coronary artery bypass grafting between 30% and 50% [34]. In the present study, we found an IAH prevalence of 68% and 28% of ACS. Also, in agreement with current literature, we found that ACS is associated with higher ICU mortality. A study published in 2018, conducted in a mixed ICU, showed that patients who developed IAH were 3 times more likely to die, independently of other disease severity indexes [35].

IAH originally was described as a complication that presented itself in patients with underlying abdominal conditions, such as

major abdominal surgery, abdominal trauma, and pancreatitis [3]. However, in the last decade many studies have shown IAH in patients with nonsurgical conditions admitted to mixed ICUs [35–38].

In a study of 264 patients in an intensive care unit Reintam-Blaser *et al*. [36] found that patients with IAH had a higher age, higher BMI, greater fluid gain, and higher disease severity scores. Dalfino *et al*. [28] studied 123 patients with ICU hospitalization longer than 24 hours and observed that IAH was associated with age, accumulated fluid balance, shock, sepsis, and abdominal surgery, but only the first three were found to be independent risk factors. Another multicenter study, which analyzed 358 patients from 39 ICUs who included patients requiring mechanical ventilation for more than 6 hours, showed that 22% of patients without additional risk factors had IAH [39].

For one reason or another medical staff do not follow the WSACS Guidelines in Latin America. Usually they rely on clinical signs and physical examination, not on risk factors. The sensitivity of physical examination in the presence of ACS varies between 40% and 61% and its positive predictive value varies between 45% and 76%. So the chances of ACS being diagnosed by physical examination alone are the same (or lower) as throwing a coin upwards, betting on one side, that is 50% (or less) [40, 41]. When the diagnosis is made, it is usually too late. It was therefore necessary to find clinical warning signs that could act as alarms. In fact, ACS hardly led a patient to death alone. The presence of long-term IAH in a sustained way in patients already with perfusion disorder undoubtedly ends up raising ICU LOS. Either due to prolonged ventilation, consequence of the increase in intra-thoracic pressure caused by IAH or due to prolonged coma, consequence of the polycompartment syndrome leading to lowercerebral perfusion pressure. This insidious process must be noticed by the surgeon or intensivist and promptly reversed in order to mitigate the endocrine-metabolic response and microcirculatory damage.

The data found in the present study are consistent with previous data on the subject, demonstrating that cumulative fluid balance plays an important role in the pathogenesis of IAH in septic patients [40]. Another risk factor identified was mechanical ventilation, which is also consistent with previous data [1, 42]. Increased CVP, P_{max} and SOFA were also related to the presence of IAH.

None of the findings were modified by the time influence in this study; in other words, length of stay did not influence the correlations found. While fluid administration in the first hour of septic shock treatment is the only mechanism capable of maintaining tissue perfusion, its disordered administration can also become an anchor that does not allow the patient to recover. In this way, it is important that the attending physician be aware of the International Fluid Academy (IFA) propositions about the stage of treatment and fluid management of the patient: 1) resuscitation phase, 2) optimization phase, 3) stabilization phase, or 4) evacuation phase [43].

The present study has the limitation of sample size due to a short time analysis in a tenbed unit; it is also a single institution study. Multicentric studies are needed in this area, including patients without abdominal risk factors for developing abdominal compartment syndrome. However, the observed data allow us to infer that patients with septic shock in intensive care units are candidates for IAP monitoring regardless of other risk factors.

CONCLUSIONS

Elevated CVP, PEEP, SOFA, airway pressure and accumulated fluid balance are correlated with elevated IAP in septic shock patients. Acidosis correction appears to decrease the risk for IAH. Bearing in mind these correlations, a next step would be to set a trigger point for each of the variables, where the chance of developing intra-abdominal hypertension is higher, in order to establish a prevention protocol with active measures to be triggered when these trigger points are reached.

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