ARTICLE IN PRESS

Journal of Critical Care xxx (2015) xxx–xxx

Contents lists available at ScienceDirect

Journal of Critical Care

journal homepage: www.jccjournal.org

The prevalance of and factors associated with intra-abdominal hypertension on admission Day in critically Ill pediatric patients: A multicenter study

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ARTICLE INFO

Keywords:
Intra-abdominal pressure
Lactate
Hypothermia
Abdominal compartment syndrome
Organ dysfunction
Oligo-anuria

ABSTRACT

Purpose: To investigate admission prevalence of intraabdominal hypertension (IAH) and to determine clinical and laboratory characteristics on admission day associated with IAH in critically ill pediatric patients.
Materials and Methods: One hundred thirty newly admitted critically ill pediatric patients were included. Intra-abdominal pressure (IAP) was measured 4 times (every 6 hours) with the bladder pressure method. Data included the demographics, diagnostic category, pediatric logistic organ dysfunction score and pediatric risk of mortality score II, clinical concomitant factors, and conditions potentially associated with increased intra-abdominal pressure.
Results: Seventy patients (56.1%) had a normal IAP ($\leq$10 mmHg, mean IAP [mmHg] 7.18 $\pm$ 1.85), while 60 patients (43.9%) had IAP $>$10 mmHg (mean IAP [mmHg] 15.46 $\pm$ 5.21). Hypothermia frequency, lactate levels, number of patients with oligo-anuria, and mechanical ventilation requirement were higher among patients with IAH compared to patients without IAH (both, $P<.05$). Hypothermia (OR, 3.899; 95% CI, 1.305-11.655; $P<.03$) and lactate levels (OR, 1.283 for each mmol/L increase; 95% CI, 1.138-1.447; $P<.001$) were only significantly associated with IAH.
Conclusions: Intra-abdominal hypertension seems to affect nearly half of newly admitted critically ill pediatric patients. Lactate level and the presence of hypothermia seem to be the independent predictors of the presence of IAH.

1. Introduction

Intraabdmominal pressure (IAP) is the steady-state pressure concealed within the abdominal cavity [1]. Intraabdmominal hypertension (IAH) is defined as a sustained increase in intraabdominal pressure and, especially, if it develops over hours, can lead to the development of abdominal compartment syndrome (ACS), which clearly worsens patient outcome even further and carries up to 60% mortality in affected pediatric patients [2–4].

Given the growing awareness of IAH and ACS, The World Society of the Abdominal Compartment Syndrome (WSACS) has first developed definitions and recommendations consensus guidelines published at 2006 and 2007, respectively [5,6]. Until updated consensus definitions and clinical practice guidelines from the WSACS were published at 2013 [1], no consensus guidelines were available for pediatric patients, and guidelines developed for adult patients were also applied for pediatric patient population. Fortunately, pediatric consensus definitions and management statements for pediatric patients were included in the 2013 updated consensus guidelines from WSACS, and IAH was

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http://dx.doi.org/10.1016/j.jcrc.2015.01.021
0883-9441/© 2015 Published by Elsevier Inc.

defined as a sustained or repeated pathological elevation in IAP >10 mmHg [1]. In the 2013 updated consensus from WSACS, however, it has been stated that although the Pediatric Sub-Committee reviewed and made recommendations regarding the appropriateness of the updated consensus definitions and management statements for pediatric patients, further work in this area is needed. It has also been stated that as overt ACS becomes less common [7], further researches must be performed in order to delineate the role of IAH without ACS in clinical situations which may be associated with increased IAP [1].

According to WSACS [1], risk factors that predispose patients to IAH/ACS can be categorized into 5 major conditions including diminished abdominal wall compliance, increased intra-luminal contents, increased intra-abdominal contents, capillary leak/fluid resuscitation and others/miscellaneous. WSACS has also recommended to perform IAP measurement when at least one risk factor for IAH or ACS is present [1]. The prevalence of IAH is variable ranging from 18 to 81 per cent depending on the IAP threshold used to define it and on the patient population studied, differing in trauma, surgical, or medical patients [8]. To the best of our knowledge, there is no study reporting the prevalence of IAH in children.

Therefore, in the present multicenter prospective study, in a mixed medical-surgical population of newly admitted critically ill pediatric patients, we aimed:

1. To investigate the prevalence of IAH on admission day using the definitions and recommendations specified by the 2013 updated consensus guidelines from WSACS,
2. To determine clinical and laboratory characteristics on admission day which are associated with the presence of IAH.

2. Material and methods

2.1. Setting

The study was conducted as a 1-day snapshot study on the prevalence of IAH in 11 pediatric intensive care units (PICU) located in university or medical education and training hospitals in Turkey. The study protocol was approved by the local institutional ethics committee of each participating center and was performed in accordance with the Helsinki Declaration. Written parental consents were obtained from all participants.

2.2. Patients

There were a total of 130 eligible newly admitted critically ill pediatric patients included in the present study. Exclusion criteria consisted of PICU admission for monitoring only, PICU less than 24 hours, or having contraindication for intravesical pressure measurement, eg, pelvic fracture, hematuria, or neurogenic bladder.

2.3. Data collection

On admission, age, gender, weight, height, diagnostic category (medical/surgical/trauma), Pediatric Logistic Organ Dysfunction (PELOD) score, and Pediatric Risk of Mortality (PRISM) score II were recorded. Predisposing conditions for the development of IAH were recorded according the risk factors presented in Table 1[1].

Clinical concomitant factors and conditions potentially associated with increased IAP at intensive care admission were recorded for each patient. Clinical concomitant conditions were defined as follows: acidosis, arterial pH <7.2; hypothermia, core temperature <35 °C; coagulopathy, platelet count <55,000/mm^3, or activated partial thromboplastin time more than 2 times normal or prothrombin time <50% or an international standardized ratio >1.5; sepsis, defined according to the American-European consensus conference definitions; and liver dysfunction, de-compensated or compensated cirrhosis, or other liver failure with ascites, eg, paraneoplastic, cardiac failure, portal vein thrombosis, or ischemic hepatitis. As massive fluid resuscitation and polytransfusion definitions for children are not still clear, we calculated and recorded the total amount of fluid and blood product received.

Table 1
Risk factor for intraabdominal hypertension/abdominal compartment syndrome

1. Diminished abdominal wall compliance
Acute respiratory failure, especially with elevated intrathoracic pressure
Abdominal surgery with primary fascial closure
Major trauma/burns
Prone positioning
2. Increased intra-luminal contents
Gastroparesis
Ileus
Colonic pseudo-obstruction
3. Increased abdominal contents
Hemoperitoneum/pneumoperitoneum
Ascites/liver dysfunction
4. Capillary leak/fluid resuscitation
Acidosis
Hypotension
Hypothermia
Polytransfusion
Coagulopathy
Massive fluid resuscitation
Oliguria
Sepsis
Major trauma/burns
Damage control laparotomy

2.4. Measurement of intra-abdominal pressure

Intra-abdominal pressure was measured through a Foley bladder catheter as defined in the *Final 2013 adapted pediatric consensus definitions* section of the 2013 updated consensus from WSACS [1]. Briefly, in a complete supine position, 1 mL/kg of normal saline, with a minimal instillation volume of 3 mL and a maximum installation volume of 25 mL, was instilled in to the bladder through a Foley catheter. The end of the catheter was connected to transparent, open ended plastic tubing, and the level of the water column above the midaxillary line reflects IAP. Intra-abdominal pressure was measured 4 times at 6-hour intervals during 1 day.

2.5. Definitions

In accordance with 2013 updated consensus guidelines from WSACS, IAH in children was defined as a sustained or repeated pathological elevation in IAP >10 mmHg [1]. Thus, we preferred to use mean IAP (mean of 4 measurements), not the single elevated IAP value (IAP_{max}, the highest daily value), either to define IAH or to use for analysis.

2.6. Statistical analysis

Categorical variables were expressed as counts and percentages, while numerical variables were expressed as mean ± SD or median (minimum, maximum) where appropriate. Categorical variables were compared by means of χ^2 test. Kolmogorov-Smirnov test was used to assess the normality of the distribution of numerical variables. Normally distributed continuous variables were compared with the Student *t* test, while Mann–Whitney *U* test used for non-normally distributed variables. To assess the independent predictors of IAH, all the variables that differed significantly between patients with and without IAH were entered in a backward multiple logistic regression models. Because of the study design, a 1-day snapshot study, not a prospective or a retrospective study, we chose odds ratio (ORs) instead of relative risk for statistical analysis. Odds ratios are given with 95% confidence intervals (CIs). $P < .05$ was considered to be statistically significant.

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Table 2
Comparison of clinical and laboratory characteristics on admission day of PICU patients with and without intraabdominal hypertension

	Without IAH n = 70	With IAH n = 60	p value
Age (mo)	11 (2, 194)	12.5 (2, 204)	NS
Gender (male/female)	34/36	23/37	NS
Body mass index (kg/m^2)	17.9 ± 4.7	17.5 ± 5.2	NS
PRISM II score	19.4 ± 13.4	17.7 ± 9.5	NS
PELOD score	19.0 ± 13.7	17.9 ± 12.8	NS
Etiology of critical illness			
Medical	54 (77.14%)	45 (75%)	NS
Surgical	12 (17.14%)	9 (15%)	NS
Trauma	4 (5.7%)	6 (10%)	NS
Abdominal surgery	13 (18.57%)	12 (20%)	NS
Pneumoperitoneum	0 (0%)	2 (3.33%)	NS
Hemoperitoneum	3 (4.28%)	4 (6.66%)	NS
Abdominal infection	9 (12.85%)	4 (6.66%)	NS
Total fluid received (mL)	1043 (250, 6600)	1100 (229, 5100)	NS
Ileus	10 (14.28%)	7 (11.66%)	NS
Lactate (mmol/L)	3.83 ± 2.39	7.95 ± 6.11	<0.001
HCO_3 (mmHg)	21.7 ± 9.7	20.7 ± 7.7	NS
$PaCO_2$ (mmHg)	51.17 ± 21.88	56.15 ± 26.06	NS
Acidosis	31 (41.28%)	29 (48.33%)	NS
Hypothermia	7 (10%)	16 (26.66%)	=0.02
Coagulopathy	34 (48.57%)	38 (63.33%)	NS
Sepsis	40 (57.14%)	39 (65%)	NS
Liver dysfunction	21 (30%)	13 (21.66%)	NS
Mechanical ventilation	54 (77.14%)	57 (95%)	=0.005
PEEP (cmH_2O)	8.89 ± 2.27	7.42 ± 2.5	NS
Blood product received (mL)	235 (60, 2300)	322 (50, 2000)	NS
Pneumonia	36 (51.4%) 35 (58.3%)	NS	
*Urine output (mL/kg per hour)	2.361 ± 1.54	1.95 ± 1.43	NS

$PaCO_2$, partial arterial carbon dioxide pressure; HCO_3, arterial bicarbonate; PEEP, positive end-expiratory pressure.

3. Results

We enrolled 130 pediatric patients [59 female, median age 11.5 (2-204) months, mean body mass index 17.7 ± 4.9 kg/m^2] fulfilling inclusion/exclusion criteria. The causes for PICU admission were as follows: 73.07% (n = 95) medical, 19.23% (n = 25) surgical, 7.7(n = 10) traumas (P< 0.05). Mean PRISM II score and PELOD score of all patients were 18.6 ± 11 and 18.4 ± 13.2, respectively. Most of the study population required mechanical ventilation (85.4%). Mean positive end-expiratory pressure was 7.1 ± 2.3 for all patients.

Mean IAP was 11 ± 5.6 for all measurements. Seventy patients (56.1%) had a normal IAP (≤10 mmHg, mean IAP [mmHg] 7.18 ± 1.85), while 60 patients (43.9%) had IAP >10 mmHg (mean IAP [mmHg] 15.46 ± 5.21).

Clinical and laboratory characteristics of patients subdivided as with and without IAH are shown in Table 2. Hypothermia frequency, lactate levels, and mechanical ventilation requirement were higher among patients with IAH compared to patients without IAH (both, P< .05). We did not find any statistical differences in regard to other clinical and laboratory characteristics between patients with and without IAH (all P> .05). Although mean urine outputs of patients with and without IAH were not statistically different, the number of oligo-anuric patients were higher among patients with IAH compared to patients without IAH (55% [n = 33 out of 60 patients] vs. 38.57% [27 out of 70 patients], P< .005).

Logistic regression analysis revealed that the presence of hypothermia (OR 3.899, 95% CI 1.305-11.655, P< .03) and lactate levels (OR 1.283 for each mmol/L increase, 95% CI 1.138-1.447, P< .001) were only significantly associated with the presence of IAH.

Although it was beyond the scope of the study, information obtained through chart review revealed that duration of mechanical ventilation, PICU length of stay, hospital length of stay, and 30-day mortality of patients with IAH vs. patients without IAH were 10 (0, 83) vs 5 (0, 81) days (P> .05), 11 (3, 83) vs 9 (3, 91) days (P> .05), 17 (3, 111) vs 16 (8, 151) days (P> .05), and 46.7 % (n = 28) vs 25.7% (n = 18) (P= .001), respectively. During follow-up at the PICU, 16 patients with IAH vs 9 patients without IAH developed organ failure (P< .05). Of 16 patients with IAH, 5 patients had renal failure, 4 patients had respiratory failure, 1 patient had either liver or cardiovascular organ failure, and 5 had multiorgan dysfunction syndrome (MODS). Organ failure was considered to be associated with IAH in the absence of any other etiological cause in patients with IAH. Of 9 patients without IAH, 3 patients had renal failure, 4 patients had respiratory failure, and 2 patients had MODS. Renal replacement therapy (either peritoneal dialysis or hemodiafiltration) was applied to 3 patients with IAH and 1 patient without IAH who developed renal failure and 2 patients with IAH and 1 patient without IAH who developed MODS, while plasmapheresis was performed in 2 patients with IAH who developed MODS. All patients with and without IAH who developed respiratory failure required mechanical ventilation.

4. Discussion

The main findings of the present study are the high prevalence of IAH in newly admitted pediatric critically ill patients, the independent association of IAH with hypothermia, and increased lactate levels.

The IAP is generated by the relationship of abdominal wall with the intraabdominal content and can be measured directly through a needle or catheter placed in the peritoneal cavity or indirectly by gastric or the urinary bladder pressure [9,10]. Trans-bladder technique is a simple, reliable, and minimally invasive procedure that can be easily used in pediatric patients. In fact, in the 2013 updated consensus from WSACS, trans-bladder technique has been accepted as the standard IAP measurement technique for clinical studies [1]. Therefore, in the present study, we preferred the trans-bladder technique in measuring IAP in our study cohort. Although the bladder pressure can be unreliable in case of low intrinsic bladder compliance, bladder trauma or pelvic hematoma [9], patients with those problems were not included in our study.

In the 2013 updated consensus from WSACS, the Pediatric Guidelines Sub-Committee defined IAH in children as a sustained or repeated pathological elevation in IAP >10 mmHg [1]. As sustained or repeated elevation in IAP was recommended for the diagnosis of IAH at last consensus, and as IAP substantially fluctuates during the day like any other "body pressure", in the present study, we chose to use the mean of 4 measurements (IAPmean) rather than the maximal value of the 4 measurements (IAPmax) in diagnosing IAH. The occurrence of IAH in an adult critically ill patient population has been reported in the range of 18% to 81% depending on the threshold used to define it and the population studied, differing in trauma, surgical or medical [8,11]. To the best of our knowledge, there is no paper reporting the prevalence of IAH in pediatric critically ill patients and this is the first report on the prevalence of IAH in a medical-surgical population of newly admitted critically ill pediatric patients. Using the cutoff value accepted for pediatric patients at last consensus [1], we found a considerably high IAH prevalence (43.9%) on admission in our study cohort.

Depending on the severity and overall hemodynamic conditions, IAH has been reported to be associated with bowel ischemia, bacterial translocation, acute renal failure, respiratory failure and central nervous impairment [9,10,12–17]. Therefore, early identification of factors leading to or predicting IAH has a vital role in preventing the development of IAH or improving the clinical management and outcome of patients with IAH. Several etiological and predisposing factors that predict the presence of IAH in a surgical, medical, or mixed surgical-medical critically ill patient population have been reported. Ivatury et al found that lactate levels, mesh closure, and abdominal trauma were the best predictors for IAH in surgical patients [12]. High crystalloid volume and low systemic blood pressure were found as independent factors for IAH in surgical and trauma patients in the study of Balogh et al [18]. In a previous study by Malbrain et al, in a mixed ICU population, they found that the only variable significantly associated with IAH was body mass index, although the transfusion rate and the fluid resuscitation

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(probably associated with a positive net balance) were close to the limit of statistical significance [11].

As a matter of fact, in the 2013 updated consensus from WSACS [1], the risk factors and predisposing conditions for IAH/ACS has been categorized into 5 major conditions, and IAH measurement has been recommended to be done in every patient with at least one risk factor. In the present study, we found that although mechanical ventilation requirement was also higher among patients with IAH, only the presence of hypothermia and lactate levels significantly predicted the presence of IAH in our patient population.

In the presence of IAH, increased lactate production is the result of impaired oxygen delivery and decreased lactate clearance by the liver [19]. Impaired oxygen delivery is caused mainly by decreased cardiac output and, in part, decreased mean arterial pressure. Several mechanisms such as direct compression of the heart, decreased contractility due to displacement of the diaphragm and decreased venous return due to compression of the inferior vena cava have been suggested for the decrease in cardiac output in the presence of IAH [20]. Although mean arterial pressure may initially rise due to shunting of blood away from the abdominal cavity, it normalizes or decreases thereafter [21,22]. Besides decreased cardiac output and decreased mean arterial pressure, critical illness can increase oxygen demand and impair global oxygen delivery. Furthermore, critical illness can reduce the capability of cells to utilize oxygen despite an adequate oxygen supply. This may be caused by inhibition of mitochondrial oxidative phosphorylation by substances such as nitric oxide and proinflammatory cytokines [23]. The vascular liver is extremely susceptible to IAH. With increased IAP, blood flow decreases in both the hepatic artery and the portal vein. This change in blood flow leads to decreased lactate clearance by the liver [24]. Although IAH via compromising tissue perfusion due to mechanisms presented above may result in accumulation in lactate, accumulated lactate also may lead to metabolic acidosis, which may further decrease cardiac output by compromising cardiac function and may contribute to increase in IAP by causing capillary leak. It is well known that both acidosis and hypothermia, another independent predictor of IAH that we found in the present study, are among the factors which have been suggested to lead to IAH via inducing capillary leak [1].

In the present study, PRISM II and PELOD scores of patients with and without IAH were not statistically different. This might be due to several factors such as time period chosen for the study (early critical illness period), similar severe illness pattern of the study groups (as presented in Table 2), and single measurement of those organ dysfunction scores. In fact, daily measurements of those organ dysfunction scores have been suggested to provide more definitive prognostic results [25,26]. However, due to study design, we did not repeat the measurement of those organ dysfunction scores.

In the present study, information obtained through chart review revealed that duration of mechanical ventilation, PICU length of stay, hospital length of stay were also similar between patients with and without IAH. These similarities might be, in part, due to similar severe illness pattern of the study groups, as also shown by similar PRISM II and PELOD scores between groups, as well as application of preventive measures such as avoiding a positive cumulative fluid balance, usage of enhanced ratio of plasma/packed red blood cells instead of low or no attention to plasma/packed red blood cell ratios, enteral decompression with nasogastric or rectal tubes, neuromuscular blockade, optimal pain and anxiety relief, which might exert improving effect on outcomes of patients with IAH, as suggested in the 2013 updated consensus from WSACS [1]. However, despite all of those preventive measures stated above, patients with IAH developed significantly higher organ failure during follow-up and had significantly higher 30-day mortality compared with patients without IAH.

In fact, it is important to emphasize that the main purpose of the present study was to determine the prevalence of IAH and factors associated with the presence of IAH on admission day in critically ill pediatric patients. The association of IAH with organ dysfunction during follow-up was beyond the scope of this study. The lack of association of IAH with other risk factors and predisposing conditions presented in Table 1 might be, in part, due to the design of our study.

In conclusion, IAH seems a substantial clinical problem that may affect nearly half of newly admitted critically ill pediatric patients and seems to have a significant impact on morbidity and mortality of those patient populations. It seems that, at least for initial characterization, IAP should be measured routinely on admission. Although during ICU stay many other factors may influence and predict the development of IAH, on admission day, lactate level and the presence of hypothermia seem to be the independent predictors of the presence of IAH. Long-term prospective follow-up studies are needed to better understand the factors associated with the development and the clinical consequences of IAH in critically ill pediatric patients.

Conflict of interest

The authors declare that they have no conflict of interest.

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