Intraabdominal Hypertension in Patients with Septic Shock

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Intraabdominal hypertension (IAH) develops frequently in patients with septic shock. Even a moderate increase in intraabdominal pressure (IAP) in this setting could be associated with high lactate levels. The authors conducted a prospective, observational, nonrandomized control trial in the surgical intensive care unit of an academic tertiary center. Twenty-seven patients with septic shock (septic shock group) and 19 patients undergoing abdominal surgery with more than two risk factors for IAH (postoperative control group) were admitted consecutively to the intensive care unit. IAP was measured every 6 hours during the first 48 hours. IAH was diagnosed with two consecutive measurements greater than 20 mm Hg. The main outcome measures were prevalence of IAH in septic shock and control groups; and comparative lactate levels, norepinephrine requirements and organ dysfunctions in patients with and without IAH in both groups. Fifty-one per cent of patients with septic shock and 31 per cent of control patients developed IAH. Patients with septic shock with and without IAH were comparable in peak norepinephrine dose, sequential organ failure assessment score, and mortality. However, peak lactate levels were significantly higher in patients with septic shock and IAH compared with those without IAH (3.5 mmol/L versus 1.9 mmol/L, P < 0.04). There was a significant positive temporal correlation between IAP and lactate levels in patients with septic shock with IAH. Peak levels of both occurred early and decreased progressively over time. Control patients with and without IAH exhibited comparable peak lactate levels. Intraabdominal hypertension is very common in septic shock and appears to be related to high lactate levels, which diminish as IAP decreases. Future studies should address the usefulness of IAP monitoring in patients with septic shock.

The deleterious effect of a sustained increase in intraabdominal pressure (IAP) was first described by Wendt in 1876. Recent research has consistently demonstrated that persistent elevation of IAP impairs cardiovascular, respiratory, and renal system function. In addition, IAP may contribute to splanchnic hypoperfusion and to development of multiple organ failure in critically ill patients.

The specific threshold to define intraabdominal hypertension (IAH) has not been established, explaining the wide range reported (from 12 to 25 mm Hg). The incidence of IAH is variable, depending on the threshold selected, medical or surgical condition and the technique used to measure IAP.

The most severe form of IAH is the abdominal compartment syndrome (ACS). ACS is defined as a persistently elevated IAP, generally above 20 mm Hg, associated with progressive organ dysfunction. A relationship between IAH and ACS with morbidity and mortality exists, particularly in trauma and surgical patients. With the risk of organ dysfunction greater beyond 20 mm Hg, our intensive care unit’s (ICU’s) monitoring and management protocol considers that level of IAP as clinically relevant and implements several interventions to control IAH.

Previous studies of IAH have focused on trauma, surgical, burned, and obese patients. Surprisingly, limited information exists about the prevalence and consequences of IAH in critically ill patients with septic shock (SS). Splanchnic hypoperfusion is well established during SS; therefore, it is reasonable to assume that even moderate increases in IAP in these patients may trigger or aggravate anaerobic metabolism and lactic acidosis at the splanchnic level. Hence, it is important to establish the incidence of IAH in SS and determine its relation to lactic acidosis or organ dysfunction.
We hypothesized that patients with SS commonly develop IAH and that elevations of IAP can be associated with higher lactate levels.

The aim of this study was to determine: 1) the prevalence of IAH and 2) the effect of IAH on lactate levels, organ dysfunction (sequential organ failure assessment [SOFA] score), and vasopressor requirements in patients with SS during their first 48 hours of ICU stay as compared with postoperative control patients with a high risk of IAH.

Materials and Methods

A prospective observational study was conducted from December 2003 to May 2004 at the Surgical Intensive Care Unit. The study was approved by the Ethical Committee of the Pontificia Universidad Católica de Chile and all patients or their relatives signed an informed consent.

Patients

Twenty-seven consecutive patients with SS, according to the ACCP/SCCM Consensus Conference, admitted to the ICU during the study period were included. Sixteen patients had a medical condition and 11 were surgical (post-laparotomy).

Nineteen postoperative patients from major non-elective abdominal surgery with more than two risk factors for IAH and planned ICU stay longer than 48 hours, were included as a control group.

Demographic and clinical data, including age, gender, date of enrollment, medical or surgical condition, APACHE II, and IAH risk factors, were registered on admission. During follow up, SOFA, peak norepinephrine infusion rate, lactate levels, and fluid balance were registered on a daily basis. Patients were followed until death, hospital discharge, or for a maximum of 28 days.

Hemodynamic Management

All patients with SS were treated with a standard hemodynamic management algorithm based on norepinephrine as a vasopressor with implementation of sequential interventions pursuing a mean arterial pressure of greater than 70 mm Hg and normalization of lactate and venous oxygen saturation values.

Intraabdominal Pressure Monitoring and Management

Intraabdominal pressure was measured every 6 hours during the first 48 hours of ICU stay using a Foley bladder catheter according to a previously described technique with the patient in complete supine position and adequately sedated if on mechanical ventilation. Our IAP monitoring protocol recommends IAP measurement every 6 hours during the initial 48 hours of the patient’s stay and thereafter only if the patient remains in shock. Any IAP measurement greater than 20 mm Hg is repeated after 30 minutes and IAH is diagnosed if the second measurement remains greater than 20 mm Hg. IAH is managed with additional sedation, nasogastric tube continuous aspiration, rectal tube insertion, and surgical drain aspiration. If despite these measures, ACS develops, surgical decompression is considered.

Statistical Analysis

All variables were compared with the Mann-Whitney test for nonnormally distributed variables and with the χ² test. Correlation between lactate levels and IAP over time was calculated using a generalized linear model. SAS (version 8.2) and SPSS (Windows version 10.0; Chicago, IL) statistical software were used. Results are expressed as mean ± standard deviation and a P < 0.05 was considered significant.

Results

Baseline characteristics are shown in Table I. In the SS group, the most frequent sources of infection were pulmonary (42%) and intraabdominal (37%). Surgical sepsis was present in 11 patients and medical sepsis in the remaining 16.

In the control group, the principal diagnoses were bowel obstruction (seven patients), diverticulitis (five), ischemic bowel disease (four), and liver transplantation (three).

Use of mechanical ventilation, a pulmonary artery catheter, and dialytic procedures are summarized in Table I.

Patients with SS exhibited a mean of 2.5 ± 1.9 risk factors for IAH compared with a mean of 3.3 ± 1.9 in control patients (P = not significant).

Prevalence of Intraabdominal Hypertension

For all patients, intraabdominal pressure at admission presented a normal distribution (Fig. 1). Mean IAP for all measurements was 15.6 ± 4.9 mm Hg.

All septic shock patients recovered from shock or died within the first 48 hours. Therefore, IAP measurements every 6 hours were stopped at 48 hours.

IAH was present in 14 of the 27 patients with SS (51%) compared with six of 19 patients (31%) in the control group (P = 0.17). Patients with SS exhibited
TABLE 1. Demographic Characteristics of Patients With Septic Shock Compared With Postoperative Control Subjects

<table>
<thead>
<tr>
<th></th>
<th>Septic Shock</th>
<th>Postoperative Controls</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(27)</td>
<td>(19)</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>66 ± 17</td>
<td>49 ± 18</td>
<td>0.004*</td>
</tr>
<tr>
<td>Medical/surgical patients</td>
<td>16:11</td>
<td>19</td>
<td>0.17</td>
</tr>
<tr>
<td>IAH (%) (mm Hg)</td>
<td>14 (51)</td>
<td>6 (31)</td>
<td>0.007*</td>
</tr>
<tr>
<td>APACHE II at admission</td>
<td>22 ± 4.8</td>
<td>14 ± 5.1</td>
<td>0.001*</td>
</tr>
<tr>
<td>SOFA score at 48 hours</td>
<td>9.6 ± 2.3</td>
<td>6.8 ± 2.8</td>
<td>0.09</td>
</tr>
<tr>
<td>Lactate maximum 48 hours (mmol/L)</td>
<td>2.5 ± 1.9</td>
<td>1.5 ± 1.2</td>
<td>NS</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>20 (77%)</td>
<td>14 (74%)</td>
<td>NS</td>
</tr>
<tr>
<td>Pulmonary artery catheter</td>
<td>18 (66%)</td>
<td>12 (63%)</td>
<td>NS</td>
</tr>
<tr>
<td>Dialytic procedure</td>
<td>8 (29%)</td>
<td>3 (16%)</td>
<td>NS</td>
</tr>
<tr>
<td>Mortality rate</td>
<td>29.6%</td>
<td>5.26%</td>
<td>0.04*</td>
</tr>
</tbody>
</table>

* P < 0.05 significant.
IAH, intraabdominal hypertension (see text for definition); SOFA, sequential organ failure assessment score; NS, not significant.
Mann-Whitney test and χ² test for P values.

higher APACHE II and SOFA scores and also higher lactate levels and mortality (Table 1).

In patients with SS who developed IAH (14), protocolized management was able to normalize IAP within the next 6 hours in 10 patients, whereas the remaining four had similar levels that normalized on the subsequent measurement (12 hours). No patients developed ACS; hence, surgical management was not required.

Six of eight deaths in septic shock patients occurred beyond the 48-hour study period; therefore, we could not observe IAP values between the study period and death. Nevertheless, all patients who finally died maintained IAP greater than 15 mm Hg throughout the study period.

Intraabdominal Hypertension and Lactate Levels

Patients with SS with and without IAH were comparable in peak norepinephrine infusion dose, peak SOFA score, first 48-hour fluid balance, and mortality rate (Table 2). However, peak lactate levels were significantly higher in patients with SS with IAH compared with those without IAH (3.5 mmol/L versus 1.9 mmol/L, P < 0.04) (Table 2 and Fig. 2).

No significant difference was found in lactate levels between patients with SS without IAH and the control group (1.9 ± 1.0 mmol/L versus 1.5 ± 1.2 mmol/L). There was also no difference in lactate levels within the control postoperative group comparing those patients with and without IAH (Fig. 2).

There was a significant positive temporal correlation between IAP and lactate levels in patients with SS with IAH. Peak levels of both occurred early and thereafter decreased progressively (Fig. 3). Lactate levels were comparable between medical and surgical patients in this same subgroup (3.63 ± 2.1 mmol/L versus 3.27 ± 2.2 mmol/L, respectively; NS).

There were no complications associated with IAP monitoring. No patients developed urinary tract infection.

Discussion

In this study, more than half (51%) of patients with SS presented with IAH early in their ICU stay, irrespective of their medical or surgical condition. The presence of IAH in patients with SS was associated with higher levels of lactate, which kept a close temporal correlation with IAP.

This is the first prospective study focusing on IAH prevalence in SS. Few prospective studies have assessed IAH in critically ill patients but none specifically in patients with SS.

Establishing prevalence of IAH in SS is important, because these patients have regional distributive and microcirculatory abnormalities, which confers them a high risk of splanchnic hypoperfusion. IAH could
Table 2. Lactate Levels, SOFA Score, Fluid Balance, Vasopressor Drugs, and Mortality in Patients With Septic Shock With and Without IAH

<table>
<thead>
<tr>
<th></th>
<th>Septic Shock With IAH</th>
<th>Septic Shock Without IAH</th>
<th>( P )</th>
</tr>
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<tbody>
<tr>
<td>Number</td>
<td>14</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Lactate maximum 48 hours (mmol/L)</td>
<td>3.5 ± 2.1</td>
<td>1.9 ± 1.0</td>
<td>0.04 *</td>
</tr>
<tr>
<td>SOFA maximum 48 hours</td>
<td>9.69 ± 2.8</td>
<td>9.54 ± 1.7</td>
<td>NS</td>
</tr>
<tr>
<td>Fluid balance 48 hours (mL)</td>
<td>4009 ± 3421</td>
<td>4646 ± 3224</td>
<td>NS</td>
</tr>
<tr>
<td>Vasopressor drugs (µg/K/min)</td>
<td>0.25 ± 0.258</td>
<td>0.21 ± 0.156</td>
<td>NS</td>
</tr>
<tr>
<td>Mortality (%)</td>
<td>5 (35)</td>
<td>4 (30)</td>
<td>NS</td>
</tr>
</tbody>
</table>

\* \( P < 0.05 \) significant.
SOFA, sequential organ failure assessment score; IAP, intraabdominal pressure; NS, not significant.

Mann-Whitney test and \( \chi^2 \) test for \( P \) values.

![Fig. 2. Lactate levels in patients with (A) septic shock with intraabdominal hypertension (IAH) compared with those without IAH; (B) postoperative control patients with IAH compared with those without IAH.](image)

augment this risk and compromise tissue perfusion, therefore aggravating the patients' condition.

This is the highest prevalence of IAH reported for any group of critically ill patients. Previous reports, using a threshold value of 12 mm Hg, have ranged from 12 per cent in sepsis without shock\(^7\) to 21% in patients with abdominal surgery.\(^{16}\) Although higher than expected, this 51 per cent prevalence of IAH in SS should not be surprising. Several predisposing factors for IAH such as ileus, splanchnic hypoperfusion, bowel distention, intraabdominal third space, and massive fluid resuscitation are usually present in SS and could explain the higher prevalence. It is noteworthy that although the same number of risk factors for IAH was found in the control and SS groups of this study, patients in the latter group had a nonsignificant trend toward higher prevalence of IAH.

IAH is equally prevalent in medical and surgical patients with SS. IAP is monitored with increasing frequency in trauma and surgical patients but often overlooked in medical patients. Our data suggest that IAP monitoring should be considered with the same strength in medical critically ill patients.

Patients with SS may be especially vulnerable to the deleterious effects of IAH on the splanchnic perfusion. Patients with SS with IAH had higher lactate levels compared with those without IAH and control group patients with IAH. Within patients with SS, this difference was significant and could not be explained by severity of the disease because both groups had similar peak doses of norepinephrine, SOFA scores, and fluid balances. Neither could it be explained solely by the level of IAH because patients in the control group with similar IAP but without shock had lower levels of lactate. Severe distributive and microcirculatory flow abnormalities causing splanchnic hypoperfusion are well established in SS.\(^{19, 25, 26}\) In other groups of critically ill patients, splanchnic regional hypoperfusion independently associated with IAH has been documented through different flow and metabolic monitoring techniques.\(^{27-32}\)

IAH in patients with SS may further compromise an already jeopardized splanchnic perfusion and could synergistically trigger episodes of regional dysxia. Temporal correlation observed between IAP and lactate levels in our study is in agreement with studies in which surgical abdominal decompression in patients with ACS results in a decrease of lactate levels and improvement of cardiac output, tidal volume, and urine output.\(^{33}\) Therefore, it is possible that SS and IAH have a negative synergistic effect on mesenteric perfusion.

We cannot explain why patients with SS and IAH did not require more vasopressors or had more organ dysfunctions despite presenting higher lactate levels. An aggressive protocol of resuscitation with fluids and vasopressors aimed at restoring global and splanchnic perfusion combined with protocolized maneuvers to
lower intraabdominal pressure may have contributed to shorten the risk period, preventing progressive hypoxia in these patients. In fact, none of the patients in this study required surgical decompression.

IAP monitoring should be considered in patients with SS. Diagnosing IAH can lead to early and aggressive management attempting to prevent or minimize its deleterious effects. In fact, progressive pathologic mechanisms of IAH seem to be level-dependent, starting even with an IAP of 10 mm Hg. Splanchnic flow is markedly reduced and cardiovascular and respiratory adverse effects appear with IAP above 20 mm Hg. At least, such level of IAH should be diagnosed and treated with urgency, especially in patients who have an already jeopardized microcirculatory flow.

The prognostic significance of our results is uncertain, because we did not evaluate the effect on mortality. Nevertheless, IAH has been associated with increased mortality in surgical and trauma patients. Increased IAP within the first 3 days is a significant predictor of mortality in patients with thoracic trauma and patients undergoing liver transplant. Furthermore, IAH has been associated with bacterial translocation and augmented oxidative stress, which could be related to late septic complications and multiorgan failure.

Important limitations of our study are the small number of patients included, a heterogeneous population, and the lack of a more specific splanchnic perfusion monitoring method such as gastric tonometry.

More studies with the proper power are necessary to understand the effects of intraabdominal hypertension in patients with SS, to determine the need for specific monitoring or treatment, and to identify patients at high risk of splanchnic hypoperfusion.

In summary, IAH is present in half of patients with SS and seems to be equally prevalent in medical or surgical patients. Patients with SS may be more vulnerable to splanchnic hypoperfusion in the presence of IAH and, therefore, IAP should be monitored in patients with SS regardless of medical or surgical condition.

REFERENCES

17. Ivy ME, Atweh NA, Palmer J, et al. Intra-abdominal hy-