Predictive Factors Associated With the Development of Abdominal Compartment Syndrome in the Surgical Intensive Care Unit

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Hypothesis: Intraoperative and postoperative variables contribute to the development of abdominal compartment syndrome (ACS) in general surgical patients.

Design: Case-control cohort study of 44 patients admitted to the surgical intensive care unit from March 1, 1995, to January 1, 2001. Groups were matched with respect to age, sex, diagnosis, and procedure. Prospectively collected data included demographics, ventilatory parameters, fluid requirements, hemodynamic and oxygen-derived variables, length of stay, and mortality rates. Statistical analysis was done with the Fisher exact test and/or \( \chi^2 \) analysis. Continuous variables were analyzed with multivariate and univariate analysis. Data are presented as mean±SD. Statistical significance is defined as \( P<.05 \).

Setting: Long Island Jewish Medical Center (New Hyde Park, NY) is a large tertiary teaching hospital.

Patients: Twenty-two patients admitted to the surgical intensive care unit who developed ACS, and 22 case-control patients without ACS.

Main Outcome Measures: Identification of variables that predict the development of ACS.

**Results:** Twenty-two patients with episodes of ACS (group 1) were examined and contrasted with 22 matched patients without ACS (group 2). Using univariate analysis, the groups differed with respect to 24-hour fluid administration and balance, number of emergency procedures, peak airway pressure, central venous pressure, pulmonary artery occlusion pressure, lengths of stay in the hospital and intensive care unit, and mortality rates. With multivariate analysis, only 24-hour fluid balance and peak airway pressure (group 1 vs group 2: mean±SD, 15.9±10.3 L vs 7.0±3.5 L, and 57.9±11.9 mm Hg vs 32.2±7.1 mm Hg, respectively; \( P<.05 \)) remained significantly different. The groups did not differ with regard to age, cardiac index, operative blood loss, duration of surgery, intraoperative fluid input, or balance. A predictive equation for ACS development was created: \( P = \frac{1}{1 + e^{-z}} \), where \( z = -18.6763 + 0.1671 \text{ (peak airway pressure)} + 0.0009 \text{ (fluid balance)} \).

Conclusion: The results of this study indicate that 24-hour fluid balance and peak airway pressure are 2 independent variables predictive of the development of ACS in nontrauma surgical patients.

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MATERIALS AND METHODS

The medical records of all patients with ACS in the SICU of Long Island Jewish Medical Center (New Hyde Park, NY) were examined retrospectively and contrasted with a case-matched cohort. The SICU is a closed unit managed by surgical residents who are supervised by surgical intensivists. The diagnosis of ACS was made if patients had a bladder pressure reading greater than 25 mm Hg, with oliguria and increased peak airway pressure (PAP). Following the diagnosis of ACS, patients underwent a decompressive laparotomy. The control group and ACS group were matched according to diagnoses and procedures. Patients in both the ACS and control groups were identified from a prospective database (ICU Assistant; Levy and Associates, Houston, Tex.). Information related to the patient’s demographics, hemodynamics, respiratory function, and fluid requirements was recorded. In addition, predictive and outcome data were obtained for each patient. The predictive variables examined included scores from the Acute Physiology and Chronic Health Evaluation II (APACHE II) and APACHE III, the Simplified Acute Physiology Score II (SAPS II), the multiple organ dysfunction syndrome (MODS) score, and predictive mortality rates using the SAPS II and APACHE II. Contrasted intraoperative variables included emergent vs elective surgery, estimated blood loss, duration of surgery, net fluid administered, and net fluid balance. Postoperatively, we examined net fluid administration at 24 hours, fluid balance at 24 hours, the ratio of arterial oxygen to inspired oxygen (PaO2/FIO2), PAP, central venous pressure, pulmonary artery occlusion pressure, cardiac index, base excess at 24 hours, and urine output at 24 hours. Finally, outcome data were examined such as length of stay in the hospital and SICU as well as mortality rates.

Statistical analysis was performed using a statistical package (Statistical Product and Service Solutions for Windows, version 6.1; SPSS Inc, Chicago, III). The univariate analysis was conducted as follows: continuous variables were compared using the t test, whereas categorical variables were evaluated with the Fisher exact test or χ2 test. Potential risk factors for ACS were included using stepwise logistic regression analysis. A best-predictive model was constructed with stepwise logistic regression and best regression coefficient values. From this model, an equation predictive of ACS development was created. Data are presented as mean ± SD. Statistical significance was defined as P<.05. The probability of mortality in an individual patient was derived from the following formula:

\[ P = \frac{1}{(1 + e^{-z})} \]

where e is the base of the natural logarithm, and z is the sum of the coefficients of the significant variables and a derived constant. Because the study was a retrospective medical record and database review, institutional review board approval was not required.

Table 1. Ages and Diagnoses of Patients With Abdominal Compartment Syndrome and Case Controls*

<table>
<thead>
<tr>
<th>Age, mean ± SD, y</th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>69.9 ± 13.7</td>
<td>70.3 ± 12.9</td>
<td></td>
</tr>
<tr>
<td>Abdominal aortic aneurysm</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Postoperative laparotomy</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Nephrectomy</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

*Data are presented as number of patients unless otherwise indicated.

Table 2. Predictive Variables in the Surgical Intensive Care Unit*

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>APACHE II score</td>
<td>20.3 ± 6.5</td>
<td>11.0 ± 3.2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>APACHE II score, predictive mortality</td>
<td>38.1 ± 19.8</td>
<td>16.6 ± 8.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SAPS II score</td>
<td>45.1 ± 8.1</td>
<td>26.5 ± 6.2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SAPS score, predictive mortality</td>
<td>35.9 ± 15.7</td>
<td>8.6 ± 4.6</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>APACHE III score</td>
<td>77.5 ± 23.4</td>
<td>42.1 ± 13.2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MODS score</td>
<td>6.7 ± 3.3</td>
<td>2.9 ± 2.1</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

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Table 3. Intraoperative Data*

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emergent patients, No. (%)</td>
<td>16/22 (72)</td>
<td>3/22 (14)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Estimated blood loss, L</td>
<td>2.01 ± 3.39</td>
<td>1.51 ± 1.75</td>
<td>&gt;.5</td>
</tr>
<tr>
<td>Duration of surgery, h</td>
<td>3.21 ± 2.01</td>
<td>2.72 ± 1.31</td>
<td>&gt;.3</td>
</tr>
<tr>
<td>Net fluid administered, L</td>
<td>7.07 ± 5.14</td>
<td>6.30 ± 3.62</td>
<td>&gt;.5</td>
</tr>
<tr>
<td>Net fluid balance, L</td>
<td>4.57 ± 4.9</td>
<td>4.28 ± 2.29</td>
<td>&gt;.6</td>
</tr>
</tbody>
</table>

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A total of 44 patients (22 with ACS [group 1]; 22 controls [group 2]) were examined in this retrospective case-matched medical record review. As presented in Table 1, the groups were closely matched with respect to diagnosis and age. Patients in group 1 had higher MODS, APACHE II, APACHE III, and SAPS II scores and, consequently, higher predicted mortality rates compared with patients in group 2 (Table 2). Additionally, more patients in group 1 underwent emergent operative procedures compared with group 2 (16/22 vs 3/22, group 1 vs group 2, respectively, P<.05). The groups did not differ with respect to estimated blood loss, duration of surgery, net fluid administered, or net fluid balance (Table 3). However, differences did emerge in the first 24 hours of postoperative care. Group 1 patients had a significantly higher fluid intake at 24 hours, fluid balance at 24 hours, PAP, central venous pressure, and pul-
Abdominal compartment syndrome has been especially well described in the trauma literature and is now increasingly recognized in patients with burns and those undergoing general surgery. Abdominal compartment syndrome has been shown in multiple studies to exert deleterious effects on all physiologic systems. Despite a substantial body of literature, very little data are available on prognostic factors leading to the development of ACS. Barnes et al induced ACS in the canine model using a retroperitoneal injection of Tyrode solution, a sclerosing agent. As the compliance of the abdominal wall decreased, the intra-abdominal pressure rose, resulting in ACS when the intra-abdominal pressure exceeded 40 mm Hg. Maxwell et al described an increased risk of ACS development when the fluid administration exceeded 10 L of crystalloid or 15 units of packed red blood cells. In a porcine model, Simon et al associated the effects of hemorrhagic shock and fluid resuscitation with the earlier onset of the deleterious ACS sequelae. They reported that pigs undergoing initial hemorrhage and resuscitation developed an exaggerated response to the effects of increasing intra-abdominal hypertension. Oelschlager et al implicated heavy fluid administration in the development of ACS in patients with ruptured abdominal aortic aneurysms, and advocated delayed closure in those with ruptured aneurysms who required massive fluid resuscitation.

In our study, we sought to identify factors predictive of ACS in a case-matched cohort. We identified increased 24-hour net fluid balance and increased PAP as the factors predictive of ACS formation. The other factors were found to be nonsignificant with multivariate analysis. From our analysis we were able to develop an equation predicting ACS development using the formula

\[ P = \frac{1}{1 + e^{-z}}, \]

where \( P \) represents the probability of ACS formation. The exponent \( z \) indicates \(-18.6763 + 0.1671 \times \text{PAP} + 0.0009 \times \text{fluid balance at 24 hours} \). Therefore, a patient with a PAP of 45 mm Hg and a 12-L net fluid balance at 24 hours will have a 41% chance of ACS development.

### Comment

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\[ P = \frac{1}{1 + e^{-z}}, \]

Statistical derivation of weighted coefficients of each variable allowed for the development of the equation. According to our predictive equation, a patient with a PAP reading of 45 mm Hg and a 12-L positive net fluid balance will have a 41% chance of developing ACS. Similarly, a patient with a PAP level of 57 mm Hg following neuromuscular blockade and a 12-L net fluid balance has a 100% chance of developing ACS.

Interestingly, none of the intraoperative variables examined were predictive of ACS development. Using univariate analysis, the number of patients requiring emergent surgery was found to be statistically different between groups. However, emergent surgery was not significant with stepwise logistic regression analysis. Twenty-four-hour postoperative fluid balance was significant, whereas intraoperative fluid administration and balance and estimated blood loss were not. This may indicate that fac-
tors leading to ACS development go beyond intraoperative events and may reflect ongoing physiologic derangements. Although we observed a difference in base excess between the ACS group and the control group in univariate analysis, it was not an independent variable in multivariate analysis. Ivatury et al. reported a correlation between intramucosal gastric pH (pHi) and ACS. Whether this decrease in pH is a manifestation or a result of ACS is unclear. Increased intra-abdominal pressure has been demonstrated to cause decreased splanchnic flow and mucosal acidemia. In the future, it may be of interest to examine the levels of various cytokines in patients who develop ACS to ascertain which, if any, systemic inflammatory mediators are elevated.

We have identified increased PAP as an independent variable associated with the development of ACS. Richardson and Trinkle noted an increase in airway pressure with increasing intra-abdominal pressure. Barnes et al. demonstrated a decreasing oxygenation and pH level and increasing intrathoracic pressure with increasing intra-abdominal pressure. The mechanism by which intra-abdominal hypertension impairs pulmonary function is postulated to be primarily mechanical. Diaphragm elevation results in decreased thoracic volume, decreased compliance, and increased intrapleural pressure. Adequate ventilation can be achieved only with increased airway pressure; hence, the elevated PAP that accompanies ACS. The upward displacement of the diaphragm also results in increased ventilation-perfusion mismatch, hypoxia, hypercapnia, and acidosis. Our opinion is that the increase in PAP follows the onset of ACS.

If PAP is considered a consequence and not a cause of ACS, only 24-hour fluid balance remains as a causative variable. Using the equation

\[ P = \frac{1}{(1 + e^{-z})} \]

a new predictive equation can be developed with 24-hour net fluid balance as the sole independent predictive variable:

\[ z = -6.7291 + 0.005 \times \text{(net fluid balance)} \]

Therefore, an 8-L 24-hour net fluid balance would be associated with a 0.7% chance of ACS development, whereas a 15-L balance would be associated with a 70% incidence of ACS development. An 18-L fluid balance at 24 hours would be associated with a 99% chance of ACS development. Although PAP may not be a causative factor in ACS development, it can clearly be used as a predictive factor to assess the probability of ACS development in a critically ill patient. We feel that our predictive formula is most effective using both PAP and 24-hour net fluid balance.

In our study, no difference in urine output was noted between the ACS group and the control group. In all descriptions of ACS, the onset of oliguria closely correlates to ACS development. Meldrum et al. demonstrated the effect of increasing abdominal pressure on urine output. They found that patients with an intra-abdominal pressure reading less than 25 mm Hg were able to maintain a urine output greater than 0.5 mL/(kg·min). Sixty-five percent of patients whose intra-abdominal pressure level was between 26 and 35 mm Hg had a urine output less than 0.5 mL/(kg·min), whereas 100% of patients with an abdominal pressure reading greater than 35 mm Hg were oliguric. Our observations are not inconsistent with the findings of Meldrum and colleagues.

Our predictive equation should not be employed in lieu of clinical judgment. Patients with intra-abdominal hypertension who are suspected of having the clinical criteria for ACS should expeditiously undergo decompression before the development of the deleterious sequelae. We do feel, however, that our study has identified 2 variables associated with the development of ACS.

**CONCLUSIONS**

Our study identifies 24-hour fluid balance and elevated PAP as independent predictive variables for the development of ACS. Using regression analysis, weighted coefficients were obtained and a predictive equation was developed. However, the lack of significance of operative variables in the development of ACS infers ongoing physiologic derangements that persist beyond the time of operation. Our predictive equation should not supplant clinical judgment in the diagnosis and treatment of ACS.

**REFERENCES**