Abdominal Compartment Syndrome as an Independent Mortality Predictor Factor During Acute Pancreatitis

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Abstract

Aim: The aim was to study the role of abdominal compartment syndrome and intra-abdominal hypertension (IAH) as independent predictor factors on outcome and mortality in patients during early phase of acute pancreatitis (AP).

Material and Methods: According the IAP 102 patients with medically treated AP were divided: in the first group (N = 32 patients) the IAP was over 12 mmHg, in second group (N = 27 patients) the IAP was over 20 mmHg (ACS), and the third group (N = 43 patients) with normal pressure. There were recorded APACHE 2 score in admission, the incidence of multiorgan dysfunction syndrome and mortality.

Results: No statistical significance is observed between three groups regarding age, gender, APACHE II score, Ranson score, CTSI. Kruskal-Wallis test resulted positive for all variables suggesting a statistically significant difference between groups. The pairwise test for comparison of subgroups according to Conover, yielded a statistically significant difference of ACS (P<0.05). ACS group resulted with more early deaths (13 patients) and total deaths (15 patients).

Conclusion: The abdominal compartment syndrome and increased intrabdominal pressure occurred during the early fase of AP may be predictors of increased MODS and mortality.

Introduction

According to World Society of the Abdominal Compartment Syndrome: Abdominal compartment syndrome (ACS) is the clinical syndrome resulting from a persistent increase in IAP [1, 2], which has gained growing attention when resulting after onset of acute pancreatitis (AP) [3, 4]. Hemodynamic, respiratory, renal, intestinal, and neurological dysfunction are consequences of the abdominal compartment syndrome, resulting in Multiorgan Dysfunction Syndrome (MODS) which if unrelieved, eventually results in death. There is a relationship between intra-abdominal hypertension and mortality [5-8]. The goal of this paper is to study the role of abdominal compartment syndrome (ACS) and intra-abdominal hypertension (IAH) as independent predictor factors on outcome and mortality in patients during the early phase of acute pancreatitis (AP).

Materials and Methods

In this retrospective study, 102 patients with AP admitted to the surgical intensive care unit (SICU),
The inclusion criteria for AP were defined as: (1) the time interval between onset of typical abdominal symptoms and study inclusion less than 48 h; (2) the presence of systemic inflammatory response syndrome (SIRS) manifested by two or more of the following conditions: temperature > 38°C or < 36°C; heart rate (HR) > 90 beats/min; respiratory rate > 20 breaths/min or PaCO₂ < 32 mmHg; WBC count > 12,000/mm³ or < 4000/mm³, or > 10% immature (band) forms; (3) CT and ultrasound findings; and (4) elevated serum amylase or lipase levels, or a APACHE II score > 8, or a C-reactive protein (CRP) > 250 mg/L.

The patients in which an urgent surgical decompression was performed were excluded by the study.

The enrolled patients in the study, were divided into three groups, according to intra-abdominal pressure (IAP), which was determined by indirect measurement using the transvesical route via Foley bladder catheter during the 2 first weeks after admission. In the first group (n = 32 patients) the IAP was over 12 mmHg (intraabdominal hypertension; IAH), in the second group (n = 27 patients) the IAP was over 20 mmHg considered as Abdominal Compartment Syndrome group (ACS), and the third group (n = 43 patients) resulted with normal intrabdominal pressure (NIAP). Intermittent measurement of IAP, recording of the clinical data and calculation of MODS scores by Marshall were conducted in all patients: Urinary bladder pressure (UBP) was routinely measured in all AP patients in our study, since intravesical pressure (IVP) measurement had been described as a standard and validated technique [9]. IVP was measured using a pressure transducer, and a series of readings were obtained every 4 h apart and their average calculated as one standardized measurement in mmHg (1 mmHg = 1.36 cmH₂O). There were recorded only end-expiratory values, in order to avoid interference of the diaphragm excursion to the intra-abdominal space.

There were recorded Acute Physiology and Chronic Health Evaluation (APACHE) II score, Ranson scoring system during the first 24 or 48 h and Marshall subscore of 2 or more in any of the 6 organs (respiratory, cardiovascular, coagulation, central nervous system, hepatic, and renal) to indicate organ dysfunction associated with IAH. MODS was defined as two or more organs having persistently 2 or more Marshall subscores for more than 24 h despite aggressive resuscitation and organ support. The incidences of MODS, and the in-hospital mortality were also recorded. Early death was considered the death occurring within the first two weeks, after which it was considered late death.

In order to evaluate the severity of pancreatitis, we used contrast enhanced computed tomography (CT) and the contrast-enhanced CT scan severity index (CT-SI) developed by Balthazar.

Continuous data are presented as mean ± SD. The comparisons of age, gender, APACHE II score and CT-SI on admission, Ranson score within 48 h after admission, incidences of MODS, and the in-hospital mortality between the two groups were analyzed using the independent sample t test or ÷² test, Anova and Kruskall-Wall test considering a value P< 0.05 as statistically significant.

Post-hoc analysis was realised related to the pairwise test for comparison of subgroups according to a value of P < 0.05 which was defined as statistically significant.

Results

One hundred and two patients (54 men and 48 women) were included in the study; the mean age was 56.53 ± 11.2, 54.47 ± 9.8, and 54.34 ± 7.7 years, respectively in IAH, ACS, and NIAP groups. IAH developed in 32 (31.37 %), ACS occurred in 27 patients (26.47 %), and NIAP in 43 patients (42.15 %) out of 102 patients with AP. No statistical significance is observed between three groups regarding age, gender, APACHE II score, Ranson score, and CT-SI.

The demographic data are summarized in Table 1.

<table>
<thead>
<tr>
<th>IAH</th>
<th>ACS</th>
<th>NIAP</th>
<th>Test</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>56.53 ± 11.2</td>
<td>54.47 ± 9.8</td>
<td>54.34 ± 7.7</td>
<td>Anova (F)</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>17/15</td>
<td>14/13</td>
<td>33/30</td>
<td>x²</td>
</tr>
<tr>
<td>APACHE II</td>
<td>17.35 ± 3.90</td>
<td>16.38 ± 4.25</td>
<td>15.70 ± 4.11</td>
<td>Kruskal-Wallis (H)</td>
</tr>
<tr>
<td>Score</td>
<td>Ranson score</td>
<td>3.72 ± 0.91</td>
<td>4.11 ± 0.94</td>
<td>3.47 ± 0.89</td>
</tr>
<tr>
<td></td>
<td>CT-SI</td>
<td>5.35 ± 2.12</td>
<td>5.47 ± 1.95</td>
<td>5.12 ± 3.07</td>
</tr>
</tbody>
</table>

APACHE II score: Acute physiology and chronic health evaluation II score; CT-SI: CT scan severity index; IAH: Intraabdominal hypertension; ACS: Abdominal Compartment Syndrome; NIAP: Normal Intraabdominal Pressure.

In our series, gallstones remained the most important etiologic cause, found in 61 patients (59.80 %). Alcohol and hyperlipidemia were second and third respectively with 24 (23.52 %), and 13 patients (12.74
The other etiologic factors (trauma, ERCP) caused only 4 (3.9%) out of 102 AP.

The MODS and Sepsis rate are prominent in ACS group (respectively 9 and 4 patients, $P < 0.05$), in comparison with other two groups. ACS group resulted with more early deaths (5 patients, $P < 0.01$) and total deaths (7 patients, $P < 0.01$), as presented in Table 2.

Table 2: Complications and mortality.

<table>
<thead>
<tr>
<th>Complications</th>
<th>IAH (n)</th>
<th>ACS (n)</th>
<th>NIAP (n)</th>
<th>Test</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MODS</td>
<td>4 pts</td>
<td>9 pts</td>
<td>2 pts</td>
<td>Kruskal-Wallis (H)</td>
<td>0.02</td>
</tr>
<tr>
<td>Sepsis</td>
<td>3 pts</td>
<td>4 pts</td>
<td>1 pts</td>
<td></td>
<td>0.03</td>
</tr>
<tr>
<td>Early deaths</td>
<td>3 pts</td>
<td>13 pts</td>
<td>0 pts</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>Late deaths</td>
<td>4 pts</td>
<td>2 pts</td>
<td>2 pts</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>Total deaths</td>
<td>7 pts</td>
<td>15 pts</td>
<td>2 pts</td>
<td>&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

IAH: Intraabdominal hypertension; ACS: Abdominal Compartment Syndrome; NIAP: Normal Intraabdominal Pressure.

Kruskal-Wallis test resulted positive for all variables suggesting a statistically significant difference between groups and the performance of post-hoc analysis. The pairwise test for comparison of subgroups according to Conover, yielded a statistically significant difference of ACS compared to IAH and NIAH. $P < 0.05$

Discussion

Any abnormality that elevates the pressure within the abdominal cavity can induce intra-abdominal hypertension. In some situations, such as acute pancreatitis or ruptured abdominal aortic aneurysm, retroperitoneal processes are potential causes [10]. Severe acute pancreatitis is responsible for a considerable part of the cases of ACS in ICU. Among 18 patients treated in a surgical ICU in New York with documented ACS, the underlying condition was severe acute pancreatitis in three patients (17%) [11]. In a study reporting the ACS in medical patients, severe acute pancreatitis was the main cause (5/13 patients, 38%) [12]. There were the same findings in our series; ACS was faced in 26.47% out of the 102 patients admitted with acute pancreatitis in ICU.

Patients suffering with AP are at risk for IAH/ACS because of the large volume shifts of intra-abdominal and peripancreatic inflammatory fluid collection, capillary leakage caused by increased permeability, bowel and splanchnic edema, resuscitation fluid, and other factors. Massive fluid resuscitation in the early course of the disease combined with the severe inflammatory process in the retroperitoneum could contribute to visceral oedema leading to IAP [13]. Increased IAP resulted in a decrease of mucosal blood flow to 63% of baseline despite maintaining normal mean arterial blood pressure [14]. In addition, elevated IAP could significantly reduce bowel tissue oxygenation due to bowel ischemia [15]. The gut clearly plays a major role in the development of MODS. Recently, a subset of patients with severe acute pancreatitis who develop early MODS has been described [16, 17]. In our series MODS was found in 9 patients (33.33%), 2 patients (4.65%), and 4 patients (12.50%) respectively in ACS patients, NIAP group, and IAH patients.

In clinical practice, direct measurement of intra-abdominal pressure is impractical because it would require placement of an invasive line into the peritoneal space solely for the purpose of measuring the pressure. Fortunately, a reliable, indirect method of assessing intra-abdominal pressure exists. The urinary bladder is an intra-abdominal structure with a compliant wall. When the bladder is partially filled, the pressure in it accurately reflects intra-abdominal pressure. Fusco et al. [18], compared direct laparoscopic insufflation pressure with IVP measured with different bladder volumes, founding that a bladder volume of 50 mL revealed the least bias in measuring elevated IAP. The bladder gold standard measurement techniques reported are not uniform [5]. An automated IAP measurement technique was described minimizing the pitfalls. The IAP catheter is introduced like a nasogastric tube, equipped with an air pouch at the tip. Automated IAP measurement had good correlation with the standard IVP method [19].

This study confirms that IAH and ACS are frequent findings during AP because these conditions were observed in 31.37% and 26.47% of the studied patients, respectively. Several studies reported an incidence of IAH and ACS (defined as an IAP > 15 mmHg) in as many as 78% of patients with early severe acute pancreatitis; 90% of the fatalities in this group had developed ACS [5, 6, 17, 20]. In a study of 45 patients with acute pancreatitis, maximal IAP correlated well with the severity of pancreatitis as measured by the APACHE II score on admission and at 72 h, Ranson score and Balthazar score on CT [23]. The development of a maximum IAP > 14 mmHg could also adequately predict mortality [20].

The medical treatment is a first line strategy [21-25], but surgical intervention (decompression or a novel approach: vacuum-assisted closure (VAC) therapy, recently applied by Zanus et al. [26] is commonly considered as the only treatment for aggravated ACS.
The nasogastric decompression, prokinetic agents, bowel care, sedation, analgesia and pharmacologic paralysis were administered in order to decrease the elevated IAP. However, there is no clear consensus on the critical level of IAP at which decompression is necessary. Decompression must be strongly considered if the IAP continues to rise or if clinical deterioration occurs.

In our series, the frequency of early mortality rates was 48.1% in ACS group, whereas in IAH patients was 9.3%. Our findings correlated with the results of several studies [8, 16, 24-35] that described the early and total deaths during acute pancreatitis.

As a conclusion ACS is one of the most important causes of significant morbidity and mortality in AP, therefore occurrence of ACS should be diagnosed early and perhaps the best way to do it is to monitor IAP via bladder catheter in this subgroup of patients.

ACS and IAP could be sensitive indicators of poor prognosis in patients with AP, becoming a target for intervention.

References