INTRODUCTION

Acute pancreatitis is a disease with a large spectrum of clinical and morphological manifestations, with various forms from mild edematous pancreatitis to severe necrotizing hemorrhagic forms with significant mortality and morbidity. (1) Acute severe pancreatitis is one of the main causes of intra-abdominal hypertension which can lead to multiple organ dysfunction. (2-3)

The mortality in patients with severe acute pancreatitis remains high. In patients with severe acute pancreatitis, increased intra-abdominal pressure was associated with early development of organ failure, increased mortality and hospitalization days in the Intensive Care Unit. (4) Several biochemical markers, medical imaging procedures and multiple clinical and biochemical scores were used to assess the severity and prognosis of acute pancreatitis.

Normally, the intra-abdominal pressure is < 5-7 mmHg. (6)

Intra-abdominal pressure (PIA) is defined as a sustained increase of intra-abdominal pressure above 12 mmHg (PIA > 12 mmHg), while intra-abdominal compartment syndrome (ACS) is the combination of PIA > 20 mmHg and the onset of organ dysfunction. (5,6,7,8,9)

Intra-abdominal hypertension can be classified into:

- Grade 1: PIA 12-15 mmHg
- Grade 2: PIA 16-20 mmHg
- Grade 3: PIA > 25 mmHg (7)

Abdominal pain present at admission to the Intensive Care Unit can influence intra-abdominal pressure measurement.

Therefore, intra-abdominal pressure measurement should be made after pain control. (2)

Balthazar score was used to classify the severity of acute pancreatitis according to the aspect ratio of CT (computed tomography). (10)

Grade A – Normal pancreas – 0 points
Grade B – Focal or diffuse enlargement of pancreas – 1 point
Grade C – Peripancreatic inflammation with intrinsec pancreatic abnormalities – 2 points
Grade D – Intra-pancreatic or extra-pancreatic fluid collections – 3 points
Grade E – Two or more large collections of gas in the pancreas or retroperitoneum – 4 points.

Necrosis score (10)

No necrosis – 0 points
< 30% – 2 points
30 – 50% – 4 points
> 50% – 6 points

CT severity index = Balthazar Grading + Necrosis Score (10)

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METHODS

The study was approved by the Ethics Committee of the Emergency County Hospital of Sibiu. It is a prospective and randomized study.

From October 2011 to May 2014, 64 patients admitted to the Clinical Department of Anesthesia and Intensive Care of Emergency County Hospital of Sibiu, with the diagnosis of acute pancreatitis, was included in this study. The diagnosis of acute pancreatitis was made due to the clinical and laboratory criteria. Patients were assessed by APACHE II and Ranson scoring, as prognosis indicators, by serial CT images and intra-abdominal pressure measurement. The severity of acute pancreatitis was defined on the basis of scoring systems APACHE II and Ranson. Severe acute pancreatitis was defined by APACHE II scoring greater than or equal to 8 and Ranson scoring above 3 (11, 12, 13).

Table no. 1. Ranson criteria

<table>
<thead>
<tr>
<th>Criteria</th>
<th>At admission</th>
<th>Within 48 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 55 years</td>
<td>Hemocrit fall &gt; 10%</td>
<td></td>
</tr>
<tr>
<td>Blood glucose &gt; 200 mg/dL</td>
<td>Serum Calcium &lt; 8 mg/dL</td>
<td></td>
</tr>
<tr>
<td>WBC &gt; 16.000/mm$^3$</td>
<td>Blood urea nitrogen increase 5 or more mEq/L after fluid hydration</td>
<td></td>
</tr>
<tr>
<td>LDH &gt; 350 U/L</td>
<td>Base deficit &gt; 4 mEq/L</td>
<td></td>
</tr>
<tr>
<td>AST &gt; 250 U/L</td>
<td>PaO2 &lt; 60 mmHg</td>
<td></td>
</tr>
<tr>
<td>Hematocrit fall &gt; 10%</td>
<td>Fluid sequestration &gt; 6 L</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Ranson JHC, Rifkind KM, Roses DF, et al. Surg Gynecol Obstet 1974; 139:69

Table no. 1a. For non-gallstone pancreatitis:

<table>
<thead>
<tr>
<th>Criteria</th>
<th>At admission</th>
<th>Within 48 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 70 years</td>
<td>Hemocrit fall &gt; 10%</td>
<td></td>
</tr>
<tr>
<td>Blood glucose &gt; 220 mg/dL</td>
<td>Serum calcium &lt; 8 mg/dL</td>
<td></td>
</tr>
<tr>
<td>WBC &gt; 18.000/mm$^3$</td>
<td>Blood urea nitrogen increase 5 or more mg/dL after fluid hydration</td>
<td></td>
</tr>
<tr>
<td>LDH &gt; 400 U/L</td>
<td>Base deficit &gt; 5 mEq/L</td>
<td></td>
</tr>
<tr>
<td>AST &gt; 250 U/L</td>
<td>PaO2 &lt; 60 mmHg</td>
<td></td>
</tr>
<tr>
<td>Hematocrit fall &gt; 10%</td>
<td>Fluid sequestration &gt; 4 L</td>
<td></td>
</tr>
</tbody>
</table>

Interpretation:
- If the score is 3 or >3, severe pancreatitis is likely.
- If the score < 3, severe pancreatitis is unlikely (12, 13).

The APACHE II scoring system (The acute physiology score and chronic health evaluation) takes into account 12 variables:

1. Body temperature
2. Mean arterial pressure (mmHg)
3. Heart rate
4. Respiratory rate
5. Oxygenation: PaO2 (mmHg)
6. Blood pH
7. Serum Na$^+$ (mmHg)
8. Serum K$^+$ (mmHg)
9. Serum creatinine (mg/100ml)
10. Haematocrit
11. Leucocyte count
12. Glasgow coma score

Interpretation: if the score is greater or equal to 8, severe pancreatitis is likely (11).

Intra-abdominal pressure was measured each 12 hours and the maximum value was used to analysis and correlation with imaging prognosis factors (CT image). Intra-abdominal pressure (IAP) was measured by the technique described in Kron et al. To determine the intra-abdominal pressure a catheter inserted into the bladder connected to a pressure transducer has been used. We instilled 50 ml saline into the bladder and symphysis pubis was considered the 0 level. Maximum IAP was considered the highest pressure obtained in all measurements.

CT was performed at admission and repeated in the course of hospitalization, for about a week or more. Presence of pancreatic necrosis and evaluation of necrosis’s degree was realized with CT contrast substance. Pancreatic necrosis was defined as absence of substance of a portion of the pancreas or of all the gland (figures no. 1a,b).

Figure no.1. CT with contrast substance

Pancreatic necrosis in a 50 year old man with the diagnosis severe acute pancreatitis, the 10th day after admission, with a favorable clinical evolution.

Figure no. 1a. CT with contrast substance

Statistical analysis was performed with SPSS version 11.5. Statistical processing was performed with Student t test or Mann – Whitney U test. Data were expressed as mean +/- SD (standard deviation). It was statistically considered a p value < 0.05.

RESULTS

A total of 64 patients (40 men and 24 women) with acute pancreatitis have been included in our study, aged 24 to 66 years. The time from onset to admission in Intensive Care Unit was 35 +/- 30 hours. Of the 64 patients, 40 (62.8%) had intra-abdominal hypertension (PIA > or equal to 12 mmHg) and 24 (37.2%) had PIA < 12 mmHg. Etiological factors have been described in table no. 2.

We observed a correlation between intra-abdominal pressure, APACHE II scoring, CT severity index and presence
of complications (table no. 3, figure no. 2, figure no. 3). A relationship was not found with Ranson criteria > 3 (table no. 3).

Table no. 2. Demographic, clinical and laboratory variables of the study group

<table>
<thead>
<tr>
<th>Demographic variables</th>
<th>Presence of variables</th>
<th>Absence of variables</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age 66 (24)</td>
<td>40 men/24 women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>72% lithiasic</td>
<td>25% alcohol</td>
<td>3% dyslipidemic</td>
</tr>
<tr>
<td>Etiology</td>
<td>APACHE II score at admission</td>
<td>7.6 (6.8)</td>
<td>6.4 (4.2)</td>
</tr>
<tr>
<td>APACHE II score to 72 hours</td>
<td>1.6 (1.2)</td>
<td>0.6 (1.2)</td>
<td></td>
</tr>
<tr>
<td>Ranson scoring at admission</td>
<td>2.6 (2.3)</td>
<td>3.8 (3.3)</td>
<td></td>
</tr>
<tr>
<td>Ranson scoring after 48 hours</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laboratory variables</td>
<td>Balthazar scoring at first CT examination</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Balthazar scoring to repeated CT</td>
<td></td>
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</tbody>
</table>

Table no. 3. Relationship between the maximum intra-abdominal pressure and presence or absence of clinical and laboratory variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>Presence of variables</th>
<th>Absence of variables</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>APACHE &gt; or equal to 8</td>
<td>15,05</td>
<td>40</td>
<td>9,75</td>
</tr>
<tr>
<td>Ranson &gt; 3</td>
<td>12,43</td>
<td>33</td>
<td>10,06</td>
</tr>
<tr>
<td>CT severity index &gt; 4</td>
<td>16,09</td>
<td>42</td>
<td>9,63</td>
</tr>
<tr>
<td>SIRS &gt; 4</td>
<td>15</td>
<td>44</td>
<td>9,6</td>
</tr>
<tr>
<td>MSOF</td>
<td>17,05</td>
<td>36</td>
<td>10,21</td>
</tr>
<tr>
<td>Necessity of surgical intervention</td>
<td>17,05</td>
<td>36</td>
<td>10,35</td>
</tr>
<tr>
<td>Death</td>
<td>18,44</td>
<td>9</td>
<td>13,76</td>
</tr>
</tbody>
</table>

Table no. 4. Relationship between the maximum intra-abdominal pressure and CT severity index in clinical forms of acute pancreatitis

<table>
<thead>
<tr>
<th>Clinical forms of acute pancreatitis</th>
<th>APACHE II Score</th>
<th>CT severity index</th>
<th>Maximum IAP</th>
<th>Presence of SIRS</th>
<th>Presence of MSOF</th>
<th>N. cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>AP, acute pancreatitis; IAH, intra-abdominal hypertension; ACS, abdominal compartment syndrome</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure no. 2. Correlation between the maximum intra-abdominal pressure and CT severity index

Figure no. 3. Correlation between APACHE II scoring, CT severity index and maximum intra-abdominal pressure

DISCUSSIONS

The maximum intra-abdominal pressure is a cheap prognostic marker, easily obtained, which can be used in the assessment of the development of acute pancreatitis. Acute pancreatitis is one of the main causes of intra-abdominal hypertension of retroperitoneal origin. (14,15) Mechanisms include increased capillary permeability caused by sepsis, hypoalbuminemia, which enhance the space III, with retroperitoneal and visceral edema. (3) Acute pancreatitis can present from mild forms with spontaneous resolution to severe forms evolving to death within a few days. (3) It is very important to determine the severity of the disease in the early stage and to institute appropriate therapeutic measures. Intra-abdominal hypertension and abdominal compartment syndrome affects blood flow to all organs and plays a significant role in the patient’s prognosis. Early recognition of increased intra-abdominal pressure is primordial in management of acute pancreatitis and can reduce morbidity and mortality associated with the disease. (7) Intra-abdominal pressure contributes to organ failure in patients with severe acute pancreatitis. Massive edema associated with aggressive fluid resuscitation, paralytic ileus and pancreatic ascites can lead to abdominal compartment syndrome and may cause multi-organ and systems failure in the early stages of the disease. (3,8) Pancreatic infection and the presence of organ dysfunction are severe complications and remains important risk factors for mortality in acute pancreatitis. (16) Development of abdominal compartment syndrome in patients with acute pancreatitis is associated with increased mortality. Percutaneous drainage of pancreatic ascites and surgical decompression are preferential methods for the treatment of abdominal compartment syndrome. (8) However,
there are studies showing that, although a significant effect is obtained after decompression in decreasing of abdominal pressure, mortality remains high in patients with pancreatitis and abdominal compartment syndrome. Poor outcomes may be associated with delayed intervention. Intra-abdominal pressure was described by Etienne-Jules Marez in 1863 already and abdominal compartment syndrome was described in 1984 by Kron et al and much more information have been made ever since in the medical literature. It remains of great importance to find new approaches in the abdominal compartment syndrome of acute pancreatitis

CONCLUSIONS

A statistical relationship was observed between the maximum IAP and CT severity index in acute pancreatitis. Maximum IAP was significantly related to the CT severity index. Also, the maximum IAP was significantly higher in patients who required surgery intervention, who presented complications, SIRS, MSOF, intra-abdominal collections, infected necrosis and those who died. Intra-abdominal pressure was associated with significantly higher mortality rate. Intra-abdominal pressure measurement may have an important role as a predictive marker for severe acute pancreatitis.

Acknowledgement:
This paper was supported by the project “Sustainable performance in doctoral and post-doctoral research PERFORM - Contract no. POSDRU/159/1.5/S/138963”, project co-funded from European Social Fund through Sectoral Operational Program Human Resources 2007-2013.

REFERENCES

13. Ranson criteria – Wikipedia, the free encyclopedia.