Negative Pressure Therapy in Abdominal Compartment Syndrome

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The abdominal vacuum-assisted closure (VAC) system has been introduced, providing a new possibility to treat an open abdomen. Abdominal compartment syndrome has a great relevance in surgical practice and patient care in critical condition due to the effects of increased pressure in the enclosed space of the abdomen can lead to multiple organ failure. A prospective study was conducted on a sample of 15 patients with severe acute pancreatitis (SAP) was retrospectively analyzed, following the incidence of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), the effectiveness of the therapeutic methods applied in reducing the intra-abdominal pressure (PIA), the evolution of severity scores, length of stay in intensive care unit between January 2014 - March 2017, following negative pressure therapy. There were used vacuum assisted closure devices (VAC™ -Hartman) in order to apply negative pressure to the open abdomen, while complying with specified settings in accordance with patients' outcome. Surgery for abdominal decompression in PAS with SCA is an emergency and was imposed on 14 of the 15 patients. In the studied group, the first decompression procedure was performed on days 2 to 5 from intake, as PIA increased in evolution despite medical methods. Only 1 patient hospitalized with SAP PIA decreased by medical methods and after haemofiltration. Acute severe pancreatitis remains a serious pathology in spite of a maximum medical and surgical therapy. Continuous venous haemofiltration has contributed to lowering intraabdominal pressure. Surgery with decompression vacuum systems with negative pressure lead to a significant decrease in PIA.

Keywords: Negative pressure therapy, compartment syndrome, acute pancreatitis

Abdominal compartment syndrome (ACS) in patients with severe acute pancreatitis (SAP) is a marker of severe disease. It occurs as combination of inflammation of retroperitoneum, visceral edema, ascites, acute peripancreatic fluid collections, paralytic ileus, and aggressive fluid resuscitation.

IAH has been defined as an intra-abdominal pressure (IAP) of 12mm Hg or higher [1]; this is the threshold at which organ dysfunction may set in, although it is often undetectable unless specifically sought for.

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been found to be significant contributors to organ dysfunction in a variety of critically ill patients, and several strategies have been developed to prevent and treat ACS [2].

In case of ACS, the IAP is 20 mmHg or higher with clinically evident new organ dysfunction; acute kidney injury, cardiovascular instability and respiratory insufficiency are the most often encountered organ dysfunctions in ACS. IAH and ACS are typically an early phenomenon in SAP and in most reports IAH develops in the first 3-5 days after hospital admission [3].

Pancreatitis is a dynamic disease with a rapid and unpredictable development where complications change the course of the disease. Severe acute pancreatitis still presents a high mortality rate due to its numerous local and / or general complications, regardless of the etiology of the disease, despite progress in diagnosis, therapy and surgical treatment. Clinical examination is notoriously unreliable in diagnosing IAH and ACS, but IAP measurement should be now in the armamentarium of all contemporary ICUs. The bladder is used as a window to the abdomen, and several methods for reproducible IAP measurement are now available. Several reviews describing the techniques for IAP measurement have been published [4].

The incidence of intra-abdominal hypertension (HIA) in severe acute pancreatitis (PAS) is reported between 60-80% being prematurely installed due to inflammation, fluid accumulation, ileus but also aggressive volumetric resuscitation.

HIA is reported from admission at patients with PAS but worsens within the first 3-5 days of intake, frequently leading to abdominal compartment syndrome (SCA).

Mortality in PAS associated with SCA is 49% compared to PAS cases without SCA where it was 11% [5].

The HIA reduction protocol targets: Nasogastric decompression and prokinetic use, adequate fluid management, hemodynamic guidance with invasive monitoring, PICCO, contour pulse analysis, early initiation of haemofiltration using the cytokine absorption filters, percutaneous drainage of peritoneal collections, surgical abdominal decompression surgery with temporary open abdomen with continuous negative pressure system (According to the recommendations of the International Society of Abdominal Complication Syndrome).
When non-operative measures fail to decrease IAP and to improve respiratory, renal and cardiovascular function, one should consider surgical decompression (decompressive laparotomy) (6).

Experimental part

Material and method

A prospective study was conducted on a sample of 15 patients with severe acute pancreatitis (SAP) was retrospectively analyzed, following the incidence of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), the effectiveness of the therapeutic methods applied in reducing the intra-abdominal pressure (PIA), the evolution of severity scores and length of stay in intensive care unit between January 2014 - March 2017, following negative pressure therapy.

There were used vacuum assisted closure devices (VAC™-Hartman) in order to apply negative pressure to the open abdomen, while complying with specified settings (negative pressure, time of use of a kit) in accordance with patients’ outcome.

Determination of PIA with a device by attachment to a 50 mL closed circuit urinal probe and a pressure transducer included. The PIA value is displayed on the monitor in mmHg.

NPWT dressing has been designed according to the wounds, which are then sealed with a semi-permeable film from the kit.

Pressure level was set individually for each patient based on their needs, using continuous suction initially, and intermittent suction subsequently.

Pressure settings were dependent on the local conditions of the wound and pathological conditions of the patient were considered.

Dressing change was performed every 48-72 h by doctor.

Results and discussions

In relation to the admission of the patients in the hospital, their number was variable.

Demographic data revealed an average age of 59.28 years with a women to men ratio of 6:9.

Causes of severe acute pancreatitis were in 6 cases vesicular lithiasis, in 5 cases toxic, in 3 cases after surgical procedures and in one case idiopathic.

APACHE II on admission was 19 (average). Average PIA value at admission was 22.28 mmHg. Upon admission, all patients had PIA > 12 mmHg and 53% (8 patients) had PIA > 20 mmHg. PIA growth trend from PIA (initially) of 22.28 mmHg to day 3 PIA 24.37, p = 0.8 (fig. 1).

Protein C reactive increases from 20.55 units in day 1 to 28.44 units in day 3. Positive hydrating balance was more than 2000 ml in day 1 and 2: day 1 = 3375.90 mL, day 2 = 3159.54 mL, day 3 = 1718.20 mL (fig. 2).

Hemofiltration was performed in 10 patients (67%). It was initiated even though patients did not have kidney failure, controlling the water balance and reducing the inflammatory response. After 48 hemofiltrations continued venous venous PIA decreased in all patients.

14 out of 15 patients (93%) for decompression surgery, followed by a decrease in PIA in the first 24 h after surgical intervention (table 1).

PIA decreased to only 10 out of 14 patients operated with temporary open abdomen and continued negative pressure.

Surgery for abdominal decompression in PAS with SCA is an emergency and was imposed on 14 of the 15 patients. In the studied group, the first decompression procedure was performed on days 2 to 5 from intake, as PIA increased in evolution despite medical methods. Only 1 patient hospitalized with PAS PIA decreased by medical methods and after haemofiltration (fig. 3,4).

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Table 1

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<tr>
<th>PIApreop</th>
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<td>28.46</td>
<td>25.37</td>
<td>0.03</td>
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<td>5.72</td>
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The number of abdominal interventions was between 1 and 9 interventions (needing to change the vacuum to 48-72 h) (fig.5-8).

Complications associated with these devices were related to sealing difficulties in 2 cases.

PAS complications possibly aggravated by negative aspiration were hemoptomeum in 6 cases and digestive fistulas in 4 cases.

In 3 cases that had an evolution duration of >4 weeks, the abdomen was closed at 10 days, 14 days and respectively at 21 days.

Survival at 28 days was 6 out of 14 patients which underwent surgical interventions (57%), and at 90 days 4 out of 14 patients (28.5%).

Evolution of PAS and SCA cases (table 2).

Mortality due to acute pancreatitis complications reaches two peaks: half of the premature deaths in the first week of illness due to the massive inflammatory response leading to multiple organ failure, increased abdominal pressure and compartment syndrome. In the late stages of the disease the major causes of death are septic complications due to pancreatic necrosis and haemorrhage.

In the context of acute pancreatitis, the effects of IAH may have an important impact, not only on organ functions, but also on pancreatic perfusion. Animal studies have found that pancreatic perfusion is decreased in IAH [7], which may further increase the risk of pancreatic necrosis. Also bacterial translocation, the presumed pathway for pancreatic infection in severe acute pancreatitis (SAP), is frequent in IAH. There is a dose dependent relationship with the extent of bacterial infection. The macrophages, T cells and cytokines (Interleukin-1-beta) cooperate in synergy to destroy B cells. Insulin dependent diabetes mellitus is the result of this action [8]. The expression of proinflammatory cytokines and other mediators, including adhesion molecules, suggests that the inflammatory process may contribute to the vascular disease in diabetes mellitus [9]. Numerous experimental studies prove the capacity of the vegetal polyphenols to diminish the lipid peroxidation and to reduce the LDL oxidation [10].

Acute pancreatitis is a typical SIRS caused by local inflammation in the pancreas. Prolongation and exacerbation of SIRS may lead to activation of neutrophils, the coagulation system, and various humoral mediator cascades, and various mediators produced from these cascades may cause vascular endothelial injury, cellular injury in vital organs, and disturbance of organ perfusion. As a result, organ dysfunction may develop, resulting in SAP [11].

Excessive production of inflammatory cytokines from immunocompetent cells triggered by insult plays pivotal role in the pathophysiology of SIRS. As SIRS plays an important role in the pathophysiology of SAP and the principle feature of SIRS is hypercytokinemia, it is safely assumed that a countermeasure against hypercytokinemia is effective in the treatment of SAP.

| Table 2 |
|-------------------|-----------------|----------------|---------------------|
| Admission         | Maximum value   | Last day in ICU |
|                   | p value         |                |
| APACHE II         | 19+-6.9         | 24.35+-7.9     | 0.01                |
| PIA                | 22.28+-6.68     | 36.5+-1.5      | 18.33+-7.5          |
| Length of stay in ICU | 12.22 days (between 3 days and 40 days) |                   |
| Laparostoma vacuum type | 93% (14 din 15) |                   |
| Hemofiltration    | 67%(10/15)      |                   |
| Deaths Causes: Peritoneal haemorrhage, digestive fistula, sepsis | 60% to 28 days (9 out of 15) | 73% at 90 days (11 out of 15 patients) |
It is reported that continuous hemodiafiltration using a polymethylmethacrylate membrane hemofilter (PMMA-CHDF) can effectively remove cytokines from the blood stream and that thereby it is useful for preventing or treating multiple organ dysfunction syndrome (MODS) [12].

Timing of surgical decompression is a topic of interest nowadays. It should not be surprising that in cases of prolonged exposure to high IAP, organs function is irreversibly damaged, but the exact time frame within which decompresive laparotomy can be successful is difficult to determine. Depending on the underlying pathology, chronic wounds and swelling are often concomitant, and accumulated excess fluids being accepted as a contraventional healing factor by the compression effect exerting locally on cells and tissues. Applying negative pressure in these situations reduces extracellular fluid accumulation resulting in a better blood perfusion [13].

Microbiological surveillance of the wound after initiation of negative pressure therapy was shown to significantly improve the evolution, with an important reduction in the contamination of the wound. From a clinical point of view, necrosis was produced in a limited number of cases, obvious remission of SIRS and hipercatabolic syndrome [14].

The resulting open abdomen should be managed appropriately. Whereas this once used to be the surgeons nightmare, negative pressure therapy has become the standard of care for the open abdomen, with the lowest complications and the highest primary fascial closure rates [15].

Also in patients with severe acute pancreatitis (SAP) this method has been used successfully [16].

Using a mesh-based technique has been found the most successful method in achieving early abdominal closure and a significant decrease in PIA [17]. Vascular complications arise through numerous mechanisms: enzymatic erosion with breakage of the vessel wall, formation and rupture of an aneurysm or venous thrombosis. Depending on vessel location and pathogenesis, bleeding may occur in the gastrointestinal tract, in the peritoneal or retroperitoneal cavity.

Conclusions

Acute pancreatitis is an entity that raises numerous diagnosis and treatment problems with unpredictable evolution, being a real challenge.

Establishing a rapid diagnosis and placing it in a risk category through severity is of at most importance. IAP monitoring is a first and essential step in the diagnosis and treatment of IAH. If medical therapy fails, decompresive laparotomy may be an appropriate option to reduce IAP and restore organ function.

Acute severe pancreatitis remains a serious pathology in spite of a maximum medical and surgical therapy.

Continuous venous haemofiltration has contributed to lowering intra abdominal pressure.

Surgery with decompresive vacuum systems with negative pressure continues to lead to a significant decrease in PIA.

Treatment of laparostomy with VAC for abdominal compartment syndrome results in a high rate of successful abdominal closure and we recommend vacuum therapy in such cases, the technique proving its usefulness in the management of PIA.

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