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Intra-abdominal Hypertension and Abdominal Compartment Syndrome in Acute Pancreatitis

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1. Introduction

Intra-abdominal Hypertension (IAH) is an entity that was described in the 19th century but which importance has been recognized in the last two decades. IAH is caused by persistent elevation in intra-abdominal pressure that is associated with multiple physiological derangements in almost all systems. In the setting of intensive care, this condition is considered as an organ failure that negatively impacts the prognosis and requires specific treatment. The most severe form of IAH is called Abdominal Compartment Syndrome (ACS) and is a high mortality entity [1].

Severe acute pancreatitis (SAP) is almost always accompanied by certain degree of IAH with an incidence of approximately 70%. Underlying conditions such as ileus, retroperitoneal edema, presence of fluid collections and fluid overload explain this phenomenon. IAH increases morbidity and mortality in acute pancreatitis (AP) and has become an issue of concern [2].

The aim of this chapter is to review basic pathophysiology and clinical concepts about prevention, diagnosis and medical-surgical management of IAH in the setting of AP.

First we will review some basic concepts about definitions and pathophysiology of IAH and then we will present a detailed analysis of IAH in the setting of AP.

2. Definitions

The abdomen is a closed compartment. The interaction between solid organs, hollow viscera, gas, fluids and the cavity generates a pressure known as intra-abdominal pressure (IAP).

Normal levels of IAP range from subatmospheric to 5-7 mm de Hg. Sustained elevation of IAP is associated with multiple physiologic alterations (Intra-abdominal Hypertension) and may be life threatening when exceeds certain levels (Abdominal Compartment Syndrome).

According to the consensus document by the World Society of the Abdominal Compartment Syndrome (WSACS), updated on 2013, **Intra-abdominal Hypertension** is defined by a persistent or repeated elevation of IAP over 12 mmHg [3].

IAP can be graded as follows:

Grade I:IAP 12-15 mmHg

Grade II:IAP 16-20 mmHg

Grade III:IAP 21-25 mmHg

Grade IV:IAP > 25 mmHg

A sustained increase of IAP over 20 mmHg associated with a new organ failure is recognized as the **Abdominal Compartment Syndrome** (ACS).

The **Abdominal Perfusion Pressure** (APP) is considered a resuscitation endpoint.

$APP = \text{Mean Arterial Pressure (PAM)} - IAP$

3. Measuring IAP

IAP can be measured in any abdominal organ but the standardized technique is via the bladder. By means of a Foley urinary catheter, a volume of no more than 25 mL of sterile saline is instilled into the empty bladder. After 60 seconds (to avoid detrusor contractil activity), at end-expiration, IAP is registered in a monitor. The patient must be in the complete supine position, and the transducer must be zeroed at the level of the midaxillary line (figure 1). IAP should be expressed in mm Hg [4]. $1 \text{ mmHg} = 1,36 \text{ cmH}_2\text{O}$

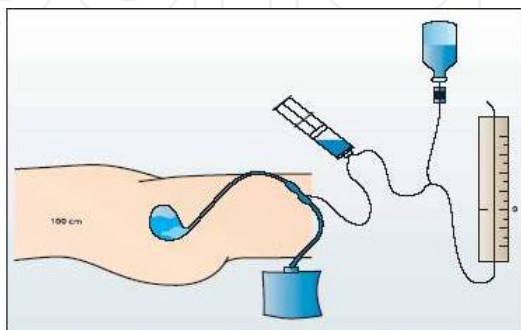


Figure 1. Intra-abdominal pressure recording via the bladder technique

4. Pathophysiology of IAH

4.1. Cardiovascular effects

IAH, by compression of porto-caval bed, diminishes venous return and cardiac pre-load. On the other hand, IAH increases systemic vascular resistance and left ventricular afterload. Both conditions lead to a decrease in cardiac output which is accompanied by an elevation of central venous pressure that reflects, in an imperfect fashion, the real status of intravascular volume in this scenario [5]. Stasis in deep venous circulation puts the patient in an increased risk for thromboembolism.

4.2. Respiratory effects

Cephalic displacement of diaphragm results in pulmonary basal atelectases with alveolar collapse. Patients develop respiratory insufficiency due to ventilation/perfusion mismatch and sometimes require positive pressure mechanical ventilation with need of positive end expiration pressure (PEEP) at levels that relate to the amount of IAH. Maximal, mean and plateau pressures will increase. Hypoxemia and hypercarbia are late signs [6].

4.3. Renal effects

Kidney is a very sensitive organ to elevations in IAP. IAH alters renal perfusion and decreases venous outflow. These phenomena, associated with lower cardiac output lead to activation of renin, angiotensin and aldosterone, resulting in oliguria and can progress to anuria. Urinary sodium is low, reflecting a "pseudo pre-renal" condition.

4.4. Gastrointestinal effects

Splachnic territory is the most susceptible to augmented IAP. Over 10 mmHg, it is possible to demonstrate a decrease in mesenteric flow leading to intestinal hypoperfusion and increasing the risk of bacterial translocation. It is likely that the decrease in abdominal perfusion pressure, by compromising perfusion of the pancreas, further contributes to pancreatic damage, necrosis, and local infection. In grade IV IAH necrosis of the gut can occur.

Otto et al, in a porcine model, showed that experimentally induced intra-abdominal hypertension provoked histological pancreatic findings similar to those of acute pancreatitis [7]. In another animal model of acute pancreatitis with and without IAH, IAH worsened evolution of severe experimental pancreatitis. No difference was found as concerns the pancreatic damage which was extremely severe in every experimental group [8]. Obviously, there are no data on the effect of IAH on the pancreas in human pancreatitis. However, these experimental data seem to be sufficient to affirm, that IAH is not only a consequence of SAP, but may contribute in the progressive pancreatic injury. It means, the more severe is AP, it is more likely that IAH develops, which in turn increases destruction of the already damaged pancreatic tissue.

Portal and hepatic artery blood flow are also altered and can explain ischemic hepatic insufficiency and lactic acidosis [9].

4.5. Central nervous systems effects

Transmission of IAP to the thorax can difficult brain venous return increasing intra-cranial pressure and eventually compromising cerebral perfusion pressure or leading to cerebral edema.

5. Risk factors for IAH in acute pancreatitis

The WSACS suggest to measure IAP in every patient who presents any risk factor for development of IAH (Table 1).

1. Factors that decrease abdominal wall distensibility

Acute respiratory failure

Abdominal surgery with primary tight fascial closure

Major trauma/ burns

Prone positioning

Obesity

2. Factors that increase intra-abdominal contents

Gastroparesis

Ileus

Colonic pseudo-obstruction

Hemoperitoneum/ pneumoperitoneum

Ascites

3. Factors that increase capillary leak/ fluid overload

Acidosis (pH<7,2)

Hypotension

Hypothermia (<33°C)

Polytransfusion (> 10 U packed red blood/24 h)

Coagulopathy

(platelets< 55000, KPTT> 2times normal, INR> 1,5)

Massive fluid resuscitation

(> 5 L/24 h) or positive fluid balance > 3,5 L/24 h

Peritoneal dialysis

Sepsis/ bacteremia

Major trauma / burns

Damage control laparotomy

Table 1. Risk factors for IAH

It is clear that AP may present various of these conditions. Furthermore, in AP, it is necessary to add local factors that contribute to increase IAP (Table 2).

Retroperitoneal inflammation
Peripancreatic inflammation and edema
Ascites formation
Retroperitoneal hemorrhage
Ileus
Fluid collections
Edema of abdominal wall

Table 2. Local factors that increase IAP in Acute Pancreatitis

Some studies have explored specific risk factors for development of IAH in AP. Ke et al found a significant correlation between the serum calcium level, 24 h fluid balance and number of fluid collections in computed tomography and the risk of IAH. Patients who developed IAH had lower calcium levels, higher fluid balance and major number of collections [10].

Dambrauskas et al showed clinical scores APACHE II >7, MODS > 2 and Glasgow-Imrie > 3 were predictive of development of ACS, thus suggesting that IAP should be recorded in these patients [11].

Bezmarevic et al investigated the role of procalcitonin (PCT) as predictor of IAH in acute pancreatitis. They found a significant correlation between PCT values and IAP levels at 24 h of admission and between maximum PCT and IAP levels [12].

6. Clinical relevance of IAH and ACS in acute pancreatitis

Since recognition of IAH/ACS as a frequent complication in AP, various clinical case series have been published. Even though different criteria have been used to define severity of AP, all reports show IAH/ACS as a complication of SAP. It is possible that exists some bias in estimating the incidence of IAH in AP given that patients who present with mild clinical features are not subjects for urinary catheterization and record of IAP. Incidence of IAH is estimated between 60-80%, whereas incidence of ACS has been reported in 10-56% [13,14].

As systematically reported, IAH/ACS are early complications of SAP that present in the first week of the illness and are usually accompanied by other organic failures. Up to 70% of patients present IAH at ICU admission.

Evolution of IAP in the subsequent days is also important. Survivors evolve with progressive decrease of IAP during the first days meanwhile nonsurvivors maintain high IAP values during the first week. A decrease in IAP could be considered as a good prognostic factor in

SAP. On the other hand, it is likely that part of early mortality, typically attributed to multi-organ failure in setting of inflammatory response, is related to ACS. ACS should be considered as a tentative cause underlying fulminant presentation because it requires a very different approach.

Morbidity and mortality are clearly affected by the presence of IAH/ACS. Mortality related to IAH in AP has been reported 30-40% and 50-75% for ACS [15,16]. Hospital stay is longer in patients affected by IAH and the risk of infection of pancreatic necrosis is also greater in this patients may be due to increased bacterial translocation [17].

In our experience (partially communicated at The European Pancreas Club Meeting 2009) in a group of 28 patients with SAP (mean APACHE II score 16,6), 22 (78%) developed IAH. In this subset, 5 patients evolved with ACS. All the patients received medical treatment to lower IAP. 15 patients underwent percutaneous drainage of collections. One patient with IAP 33mmHg and anuria required decompressive laparotomy with immediate relief of IAH and recovery of renal function. 2 patients died later, not related to IAH/ACS [18].

7. Prevention

It is likely that prevention is the most important issue to consider, given that therapeutic interventions may increase IAP. As previously mentioned, fluid overload is a very important cause of IAH in any setting, and particularly in AP. Management of fluids represents a real challenge, since patients severely ill, usually require resuscitation in the first hours and the appropriate amount and rate of infusion has not been clearly defined.

Circulatory abnormalities in AP, similar to septic shock, can lead to oxygen debt that compromises organ perfusion, mainly in splanchnic territory, leading to multiorgan dysfunction. Local microthrombosis further impairs oxygen supply to the pancreas, contributing to necrosis and release of cytokines. Randomized trials have shown that patients who receive more than 4L fluids in the first 24 h have an increased risk of respiratory failure and IAH/ACS. The same consequences have been reported in patients that receive rapid fluid expansion, defined as 10-15ml/kg/h compared to 5-10 ml/kg/h [19,20].

Another issue is the type of fluid used to restore intravascular volume. Zhao et al showed that patients with SAP treated with a combination of normal saline with hydroxyethylstarch and glutamine reached hemodynamic stability more quickly, with less fluid volume and less risk of ACS [21] and the trial by Du et al revealed that patients randomized to hydroxyethylstarch versus Ringer lactate, also received less volume and had lower IAP levels [22]. Recently, a randomized trial by Bu et al compared normal saline with Ringer lactate resulting that patients in the Ringer group had less systemic inflammation assessed by C-reactive protein levels. The authors suggested that Ringer lactate offers better local pH homeostasis [23].

The American College of Gastroenterology Guidelines suggest that aggressive hydration, defined as 250 – 500 ml per hour of isotonic crystalloid solution, should be provided to all patients in the first 12 – 24 h. After that, fluid requirements should be periodically reassessed.

Clinical end points should be restorage of diuresis, normalization of BUN and correction of hemoconcentration [24].

Enteral nutrition has systematically shown to improve evolution in acute pancreatitis. Enteral nutrition was tested as a measure to prevent development of IAH. Patients who received early enteral nutrition (in the first 48 hours from admission) had a significantly lower risk of IAH than patients fed after 8th day [25].

8. Management of IAH/ACS

General interventions that are useful to diminish IAP can be classified according to their mechanism of action. These therapies are recommended for the WSACS with different levels of strength according to the quality of the evidence (Table 3). All these measures can be applied in patients with AP in order to prevent development of IAH or progression of IAH to ACS and even to treat ACS.

Therapies to improve abdominal wall compliance

Sedation and analgesia
Neuromuscular blockade
Consider supine position < 20°- Avoid prone position
Remove constrictive dressings and abdominal eschars

Therapies to evacuate intraluminal contents

Nasogastric/colonic decompression
Proton pump inhibitors
Enemas
Colonoscopic decompression

Evacuation of abdominal collections

Percutaneous drainage
Paracentesis

Management of fluids

Restriction of fluids/ permissive hypotension in trauma
Negative fluid balance
Use of diuretics/albumin
Hemodialysis/ultrafiltration

Maintain APP > 60 mmHg

Fluids/Vasoactive drugs

Table 3. Medical treatment of IAH/ACS

Cytokines released from immune cells are fundamental in the pathophysiology of systemic inflammatory response that leads to multiorgan dysfunction in AP. Continuous hemofiltration and hemodiafiltration have been evaluated in SAP to test if diminishing blood cytokine levels improves evolution and reduces the risk of IAH.

Xu et al treated a group of patients with SAP and IAH and elevated levels of TNF- α at admission with continuous veno-venous hemofiltration (CVVH). 24 hours after initiating CVVH they noted a significant decrease in TNF- α levels and IAP. There was a positive correlation between levels of TNF- α and IAP [26]. Oda et al obtained similar results with early continuous venous hemodiafiltration in SAP. Hemodiafiltration decreased levels of IL-6 accompanied by a reduction in IAP [27]. A retrospective analysis of 10 years experience by Pupelis et al, shows that continuous hemofiltration/hemodiafiltration improves fluid balance, reduces cytokine plasma levels and reduces IAP, without changes in mortality [28].

Finally ACS not responding to medical interventions is an indication for decompressive laparotomy. In the last decades it has been clear that surgery should be avoided or at least delayed in AP, since this approach offers a better prognosis and reduces mortality. Nevertheless, one of the few remaining indications for surgery is refractory ACS, given that natural evolution in this situation is almost always fatal.

A retrospective review of 12 patients who were treated with decompressive laparotomy for ACS in the setting of SAP showed a mortality of 50%. Median time to decompression was 4,5 days and five patients underwent exploration of lesser sac in this group. However it is relevant to consider that these patients were treated between 2000 and 2009 and critical care in AP has evolved substantially in recent years [29].

Robin-Lersundi et al presented 5 patients with SAP complicated with ACS who underwent decompressive laparotomy with a mean IAP of 28 mmHg. A bilateral subcostal laparotomy was done with a temporary abdominal closure with a polytetrafluoroethylene mesh. The abdominal wall defect was repaired later. Four of five patients survived [30].

An intermediate alternative to surgical decompression is percutaneous drainage of fluid collections. In our series, 15 of 22 SAP patients with IAH were treated successfully in this way.

With existing information it is not possible to issue an emphatic recommendation about abdominal decompression in pancreatitis related ACS. It is not expected that randomized trials will come up and this decision-making will always suppose a big challenge where multidisciplinary confrontation of the patient is warranted.

In conclusion IAH/ACS are very frequent complications in patients with SAP. They represent a factor that worsens prognosis and complicates management. It is clear that we must increase our awareness to detect IAH, including in our clinical praxis algorithms to prevent, diagnose and treat this life threatening complication.

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