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Intra-Abdominal Hypertension and Abdominal Compartment Syndrome in Critically Ill Surgical Patients (Special Findings in Severe Acute Pancreatitis)

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„…a clinical entity that had been ignored for far too long"
(Ivatury RR-Sugerman HJ)

1. Introduction

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are very frequent findings in patients with severe acute pancreatitis. The causing factors are the retroperitoneal inflammation, paralytic ileus, ascites and the serious visceral edema due to massive fluid resuscitation, which leads to increased intra-abdominal pressure (IAP), early organ disfunction with IAH and finally ACS. Several publications conclude that this clinical entity can appear in high-risk surgical patients (severe acute pancreatitis) within the first 12 hours of the admission to intensive care unit. In these cases the mortality rate is extremely high. That is why we have to pay attention to make the correct and early diagnosis, treatment and follow-up of the severe acute pancreatitis (1-2).

The ACS is a rediscovered life threatening clinical entity. The aim of this chapter is to show the definitions, ethiology, pathophysiology, diagnosis and treatment of this serious, not only surgical problem.

The mortality due to the abdominal compartment syndrome is extremely high (38-71%). It can be defined as adverse physiologic consequences that occur as a result of an acute increase in the IAP. The most common causes are retroperitoneal haemorrhage, visceral oedema, pancreatitis, bowel obstruction, tense ascites, peritonitis, tumor. The affected systems are cardiovascular, pulmonary, renal, central nervous systems and splanchnic organs. The gold standard diagnostic method is the continuous intra-abdominal pressure monitoring. The fundamental ways of the treatment are the adequate fluid resuscitation and surgical decompression (1-2).

Finally we would like to show the special findings of the IAH and ACS according to the problem of severe acute pancreatitis.

2. Historical highlights

There are three big eras in the history of ACS.

The first is the evolution in the understanding of the pathophysiology of the compartment syndrome in general. (The first case of the muscular compartment syndrome was described by Hamilton in 1850.)

The second was the era of the experimental studies for measure the IAP. (different devices and techniques by different locations, for example through the bladder (uterus or rectum).

The third is the era of the understanding and management of the basic problem which was started with the work of Sir Heneage Ogilvie who performed the first laparostomies and described the beneficial effect of this process in the management of giant abdominal war wounds (3).

The real story of ACS started in the second part of the XIX. century (4-5). First, in 1863 Marey published his experiences about the increased abdominal pressure's effects. In 1865 the first intra-abdominal pressure measurement via rectum was performed in Germany. Between 1870 and 1900 numerous attempts were carried out to measure IAP and to study its influence to vital functions (Bert, Schroeder, Schatz, Wendt, Quinke, Heinricius).

In 1911 Emerson published his article titled "The intra-abdominal pressure" (6). In 1940 the first laparostomy was performed to reduce the increased IAP (Ogilvie). The first description of the importance of staged abdominal closure was published in 1948. (Gross). In 1951 Baggot claimed that abdominal closure in case of distension can cause death.

In the 70's several teams carried out investigations on laparoscopy and effects of pneumoperitoneum on IAP. In 1982 Harman and his team proved the harmful effects of IAP on renal function and significance of decompression in these cases. In 1984 Kron was the first who described the compartment syndrome but didn't use the definition itself (4). Also he used the method of pressure measurement via urinary catheter that became commonly used in 1989 though the base of this method was described by Oderbrecht 100 years earlier. Later this technique was developed by several scientists (Iberti, Sugrue, Malbrain, Balogh and their colleagues) (7-11). In 1989 terminus technicus "abdominal compartment syndrome" was created by Fietsam and his team (5). The two articles that had opened the decade of ACS were published in 1995 and 1996 (12-13). In the previous ten years several teams started to investigate the monitoring techniques of intra-abdominal pressure. The first worldwide conference was organized 6-8 December 2004, in Noosa (Queensland) in Australia and WSACS (World Society of Abdominal Compartment Syndrome) was established at the same time. This society now counts numerous members from all over the world.

3. Definitions

3.1 Consensus definitions

To understand perfectly the question of the ACS it is very important to make clear of the basic concepts. The below mentioned consensus definitions were made by the 2004 International ACS Consensus Definitions Conference of the WSACS firstly in December of 2004 in Noosa (Australia) and they were rediscussed in 2007 in the Antwerp (Belgium) during the Third World Congress of the Abdominal Compartment Syndrome (WCACS):

Intra-abdominal pressure (IAP): is the steady-state pressure concealed within the abdominal cavity. (14-15)

Intra-abdominal hypertension (IAH): is defined by a sustained or repeated pathological elevation in IAP ≥ 12 mmHg. (14-15)

Abdominal Compartment Syndrome (ACS): is defined as a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg, where APP = MAP - IAP and APP = abdominal perfusion pressure; MAP = mean arterial pressure) that is associated with new organ dysfunction / failure. (14-15)

(The normal range of the IAP is between the 0 and 5 mmHg, and it significantly depends on Body Mass Index (BMI) (9,16).)

3.2 Classification

IAH is graded as follows:

- grade I. 12-15 mm Hg
- grade II. 16-20 mm Hg
- grade III. 21-25 mm Hg
- grade IV. IAP > 25 mm Hg

(IAP may be defined with several units. To avoid misunderstanding IAP is measured in mmHg according to the international consensus (1 mmHg = 1.36 H_20cm = 0.13 kPa)). (1-2)

3.3 Etiopathogenesis

Primary ACS: is a condition associated with injury or disease in the abdominopelvic region that frequently requiers early surgical or radiological intervention. (for example: peritonitis, pancreatitis, bowel obstruction, haemorrhage, trauma, etc.) (1-2)

Secundary ACS: conditions that do not originate from the abdominal cavity, generaly caused by surgical activity (for example: abdominal closure under distension, extremely large abdominal hernias, operation of bowel obstruction). (1-2)

The incidency of IAH is 2-30% and of the ACS is 1-16% in general surgical syndromes and in the ethiopathogenesis are acute and chronic causes:

Acute (1-2):

- **spontaneous:** peritonitis, abdominal abscess, bowel obstruction, ruptured aortic aneurysm, tension pneumoperitoneum, acute pancreatitis, mesenteric thrombosis
- **postoperative:** peritonitis, abscessus, bowel obstruction, intra-abdominal haemorrhage
- **trauma:** intraperitoneal and retroperitoneal haemorrhage, visceral oedema after cardiopulmonary resuscitation
- **iatrogenic:** laparoscopy, abdominal closure under distension

Chronic (1-2):

ascites, enlarged intra-abdominal cyst, long term peritoneal dialysis, pregnancy, extreme obesity

Chronic and slow increasing of IAP can be compensated by the human organism and the abdomen adaptates for increased load, so ACS won't develop in this case.

Incidency

Incidency of IAH is 2 – 30 % (10,18) in general surgical syndromes and the incidency of ACS is 1 – 16 % (17-19). If intensive care unit (ICU) treatment is necessary, the development of ACS is expectable in the first 12 hours (11,18).

Risk factors for IAH / ACS (14-15)

Diminished abdominal wall compliance:

- acute respiratory failure, especially with elevated intrathoracic pressure
- abdominal surgery with primary fascial or tight closure
- major trauma / burns
- prone positioning, head of bed > 30 degrees
- high body mass index (BMI), central obesity

Increased intra-luminal contents:

- gastroparesis
- ileus
- colonic pseudo-obstruction

Increased abdominal contents:

- hemoperitoneum / pneumoperitoneum
- ascites / liver dysfunction

Capillary leak / fluid resuscitation:

- acidosis (pH < 7.2)
- hypotension
- hypothermia (core temperature < 33 C^o)
- polytransfusion (> 10 units of blood / 24 h)
- coagulopathy (platelets < 55000 / mm^3 or prothrombin time (PT) > 15 seconds or partial thromboplastin time (PTT) > 2 times normal or international standardised ratio (INR) > 1.5)
- massive fluid resuscitation (> 5 l / 24 h)
- pancreatitis
- oliguria
- sepsis
- major trauma /burns
- damage control laparotomy

4. Pathophysiological changes during the increased IAP

IAH can cause serious complications in any organ (**Table 1**). Without effective therapy / intervention it can occurr multi organ failure (MOF) by affected cardiovascular, respiratory, central nervous system and renal function. De Waele and his colleagues published that there was a 94% incidence of respiratory, 94% cardiovascular and 89% of renal failure among patients with IAH (where IAP > 12 mmHg). (1,2,12,13,16,18):

Cardiovascular system

The increased IAP reduces the cardiac functions. The increased pressure lowers the end-diastolic volume as well as the venous return from the inferior caval vein, portal vein and superior caval vein. The systemic vascular resistency increases because of the compressed capillary system and the afterload will be consequently elevated. All of these effects result cardiac insufficiency and compensatoric tachycardia (19,20).

IAP = 0-9 mmHg	**IAP = 10-15 mmHg**	**IAP = 16-25 mmHg**	**IAP = 26-40 mmHg**
cytokines release	circulation of the abdominal wall decreases with 42%	significant reduction of splanchnic circulation and venous return	„hemodinamic collapse"
increased capillary permeability	significant reduction in the circulation of other abdominal organs	increase in systemic vascular resistance (SVR), central venous pressure (CVP), peak airway pressure (PAWP)	fatal acidosis
fluid content of the „third space" expands	local acidosis	total respiratory capacity (TRC), vital capacity (VC) lowered due to pulmonary compression	hypoxia hypercapnia
decreased venous return as well as preload	free radical release	hypoxia hypercapnia	anuria
early effects on the central nervous system	bacterial translocation throught intestinal mucosa	circulation of intestinal mucosa decreases with 61%	circulation of coeliac artery is reduced to 58%
		increasing acidosis	circulation of superior mesenteric artery is reduced to 39%
		renal failure: oliguria, anuria	circulation of renal artery is reduced to 30%
		disturbance of central nervous system	circulation of abdominal muscles lowered with 80% (infection, wound-healing disturbances)

Table 1. Physiological changes during increased IAP (Wolfe, 2005)

Respiratory system

Due to the increased IAP the diaphragm will be shifted to cranial direction on both sides causing the reduction of pulmonary volume and cardiac function. Without intervention acidosis and hypoxia will occur (12,19,20).

Renal function

The renal perfusion decreases related to the reduced cardiac function and direct compression of renal arteries and kidneys, with consequent oligo- and anuria. IAP 15 - 20 mmHg results in oliguria, and above 30 mmHg it causes anuria (13,19,20,21).

Splanchnic circu1ation

The splanchnic, mesenteric and hepatic perfusion decreases above 15 mmHg of IAP. It is a well-known and proved fact that splanchnic and hepatic circulation has an autoregulation. The basis of this autoregulation is the renin-angiotensin system, the HABR (hepatic arterial buffer response) and vasopressin. Though numerous clinical studies proved that this complex system could compensate the consequencies of insuffient arterial circulation, venous return, decreased preload due to the increased IAP only for few hours. When it worns out it causes irreversible destruction of the intestinal mucosa and the liver. This is why decompression is essential in the ACS's therapy! The reduced circulation results in a mucosal ischaemia and bacterial translocations. Intestinal bacteria and their toxic products result sepsis and multiple organ failure (MOF) (22-24) (**Figure 1**). Endotoxins or exotoxins cause a chain reaction of mediators that can damage either the pathogen and the human organism and if it becomes irreversible can cause death. This progressive clinical syndrome is a manifestation of multiple organic dysfunction or failure (ARDS - Adult Respiratory Distress Syndrome, renal failure, DIC -Disseminant Intravascular Coagulation). If it progresses the symptoms of multiple organ failure and septic shock with hypotonia will appear. Since this phenomenon occurs as a consequence of not only infections but pancreatitis, burning and ACS, the definition of SIRS (Systemic Inflammatory Response Syndrome) was introduced in the 90's (22).

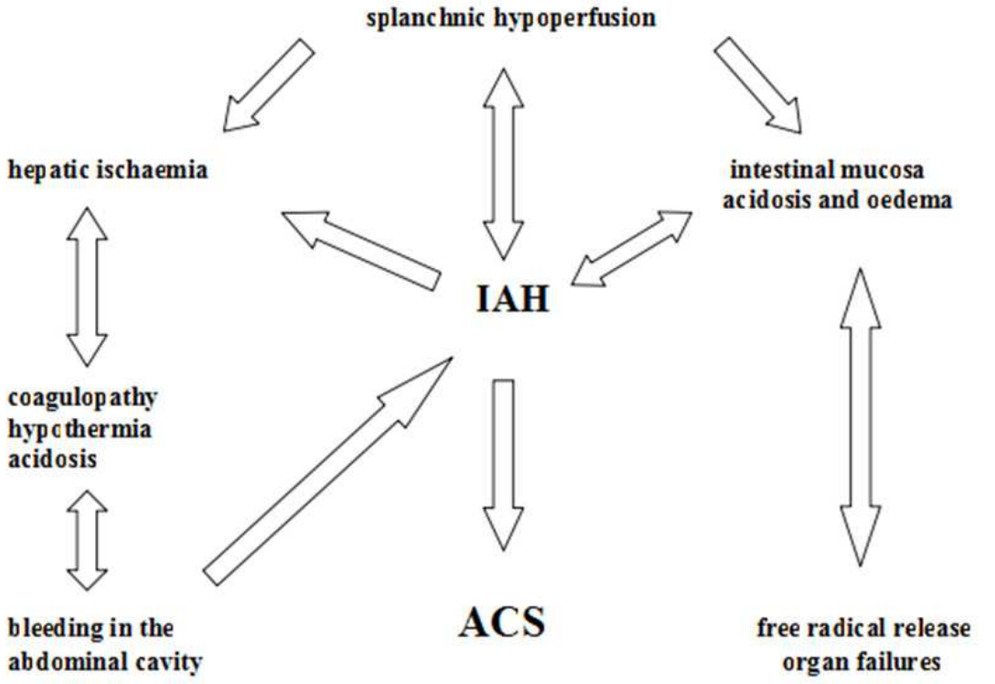


Fig. 1. Effects of increased intra-abdominal pressure on the splanchnic circulation (IAH = intra-abdominal hypertension, ACS = abdominal compartment syndrome)

Abdominal wall circulation

The reduced abdominal wall circulation results in insufficient wound-healing, higher possibility of inflammation and dehiscency (25).

Central Nervous System

The increased IAP lowers the intracranial pressure secondarily due to the compression of jugular venous system and of the thoracic region (26).

5. Laboratory changes of inflammatory markers during the increased IAP in patients with acute pancreatitis

The technique of IAP measurement is accurate, precise, reproducible and cost-effective. However laboratory measures for monitoring of IAH have not been defined. The immunological changes were examined in a study group of 65 patients with acute pancreatitis (IAP > 12 mmHg) in one of our studies. Serum adenosine, IL-1β, IL-2, IL-4, IL-10, TNFα and IFNγ were measured. Significant correlations were found among IAP-adenosine values and IAP-IL-10 values providing new tools for the laboratory monitoring of IAH as well as further understanding of the pathomechanism contributing to ACS.

We present here the historical diagram **(Figure 2)** from one of our preliminary studies which shows the very important correlation between IAP and adenosine:

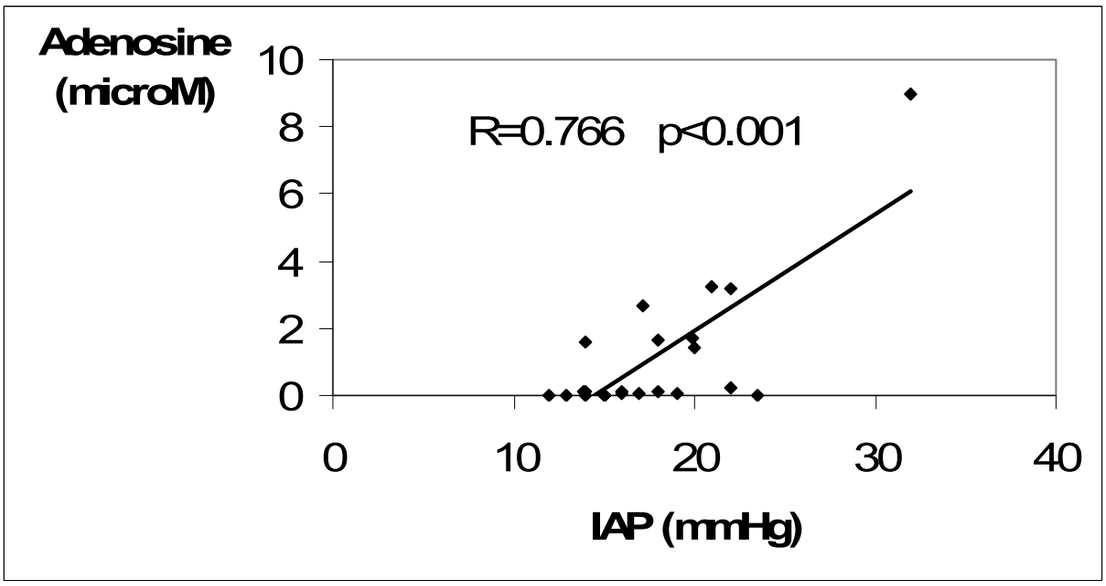


Fig. 2. Significant correlations between IAP and adenosine level

The pathophysiology of IAP is still poorly understood, including the contributions of adenosine and cytokines (27). Adenosine contributes to the maintenance of hepatosplachnic blood flow (28) and hypoxia results in increased serum adenosine concentrations (22,29). Such alterations may result in enlargement of the abdominal organ blood volume and

potentially contribute to the pathology associated with increased IAP and ACS. In addition to its contribution to maintenance of hepatosplanchnic blood flow, adenosine is an endogenous regulator of cellular functions including neurotransmission (30), local circulation (31) and the modulation of inflammation (32-35).

Previous studies in porcine and rabbit models demonstrated that increased IAP was associated with reduced gastric intramucosal pH (36) and increased levels of IL-1β, IL-6, TNFα, and CRP (37).

In our own studies, we investigated the associations of serum levels of adenosine, C-reactive protein (CRP) and various cytokines (interleukin 1β [IL-1β], IL-2, IL-4, IL-10, tumour necrosis factor α [TNFα] and interferon γ [IFNγ]) with IAP in surgical patients with or without elevated IAP.

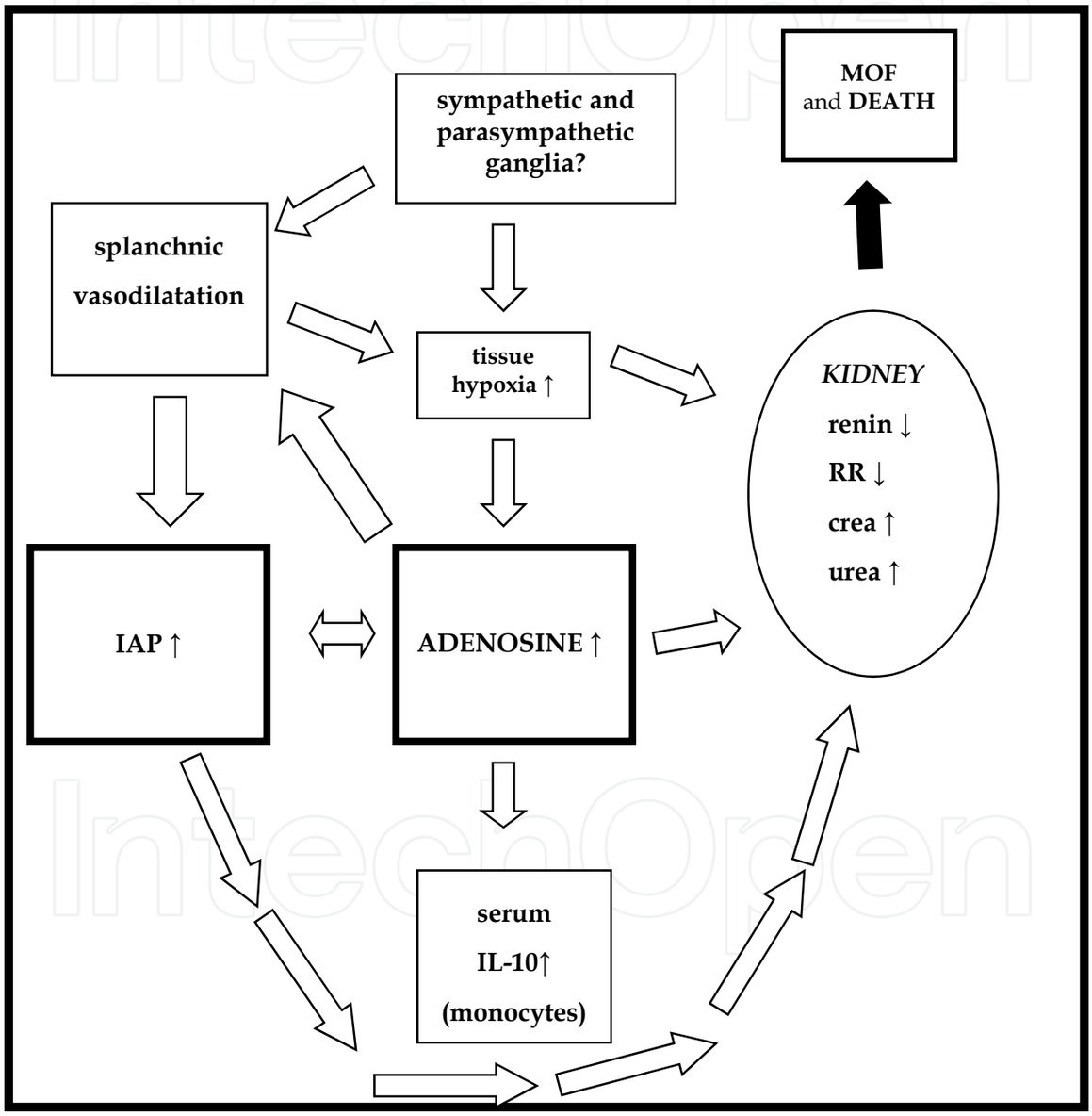
Our observations derive from surgical patients being treated in an intensive care unit and therefore, likely represents the greater degree of individual variation expected in human populations as compared to homogeneous experimental models. Thus, while cytokines such as IL-1β, IL-6, TNFα and CRP may have been significantly associated with IAP in animal models (37), they were not significantly related in our study. On the other hand, our results showed a highly significant relationship of both IL-10 and adenosine with IAP in a heterogeneous human population, suggesting that they may be better indicators of IAP in this case.

We found a robust linear correlation between IAP ≥15 mmHg and serum levels of adenosine and IL-10. While we believe that this relationship may be helpful in monitoring the effects of IAP lowering therapies, we also believe that the increase in serum adenosine level may directly contribute to the development and maintenance of IAH-ACS.

We propose that a strong relationship may exist between the advancement of hypoxia and splanchnic ischemia, inducing the release of adenosine from the hypoxaemic tissues of the gut (22,28,29), resulting in splanchnic vasodilatation and subsequent increase in IAP. This relationship is cyclic in nature and if left undisturbed, results in a 'vitious circle' leading ultimately to ACS. Thus, while adenosine itself is a very potent anti-inflammatory molecule (33-35), in the development of IAH-ACS, elevated adenosine concentrations cause renal arterial dysfunction (38), ultimately causing collapse of kidney function manifested by decreased renin production and blood pressure and increased blood urea and creatinine concentrations (39-40). In this context, the renal failure resulting from increased adenosine concentrations is a main cause of multi-organ failure (41-42) and damage of vegetative splanchnic ganglia (43-44). The elevated serum IL-10 levels appear partly to be the direct consequence of adenosine stimulating monocyte secretion of IL-10 (34), but IL-10 can be produced also by other inflammatory cells. In the **Figure 3** we propose a model describing the potential role of adenosine in the pathomechanism of IAH-ACS.

Our observations show that the laboratory determination of serum adenosine and/or IL-10 may be helpful for initial screening or grading of IAH as well as for monitoring progress in therapeutic reduction of IAP. As the determination of IL-10 by ELISA is an easily automated method, it may be preferred to the measurement of adenosine by HPLC, particularly, during follow-up of surgical, trauma or medical patients with high risk of IAH or ACS.

In conclusion, we report that plasma/serum concentrations of adenosine and IL-10 are strongly and linearly correlated to the values of IAP >15 mmHg (Grade II IAH) in surgical patients. Thus, monitoring of serum adenosine and IL-10 concentrations may offer significant insights into the progression and treatment of IAP, particularly, in patient populations at risk of IAH and ACS. The role of adenosine in the pathomechanism of IAH-ACS offers a new insight into this severe clinical syndrome (45-46).



(RR = Riva Rocci (blood tension), Crea = creatinine, MOF = multi organ failure)

Fig. 3. The role of adenosine in the proposed model of the pathomechanism of ACS

6. Diagnostic methods

Numerous investigations proved that only the physical examination of the abdomen to recognise ACS is inappropriate (11). Detection of ACS and elevated intra-abdominal pressure is based on standardized measurements of IAP. Practically IAP is detectable in every region of the abdomen (12,16,47,48).

Direct technique:

- during laparoscopy (laparo-insufflator)
- with an intraperitoneal cateter and transducer
- with a metal cannula lead into the intraperitoneal space and connected to a manometer

lndirect technique:

- pressure measurement of inferior caval vein
- measurement throught nasogastric tube
- measurement of vaginal pressure
- measurement of rectal pressure
- measurement of urinary bladder's pressure

It seems that the most reliable and the less invasive method is the transvesical technique (7,8,13). It is based on the fact that IAP is equal with intravesical pressure. Filling the urinary bladder with 50 ml saline serum ensures that the pressure will be translocated to the catheter wich is clamped during the procedure. The pressure inside the catheter measurable by connecting a T-tube or inserting a sterile needle (Sugrue technique) (12). The T-tube is connected to a monitor via a transducer or to the system used to measure the central venous pressure (pressure of the liquid column). The intravesical technique's results correlated with the direct (laparoscopic insufflator) method. The intravesical way of measuring was more accurate than the gastric or rectal way which are very position depending. Animal-testing proved that the pressure of inferior caval vein is correlating with the vesical pressure but it is much more invasive and can be the source of serious complications - like other direct methods. Additionally urinary catheter is required in all critically ill patients. The intravesical method for measuring IAP was originally described by Kron (4), and then validated by Iberti and his team (8). It was simplificated by Sugrue and his team: a T-tube was inserted to the catheter. Since in this method it is unnecessary to prick the aperture, the number of infectious complications decreased. This method is simple but may be time consuming - because in case of critical patients approximately 7 minutes are needed for the procedure at least four times a day. Having intermittent character the standard IAP measuring is unable to give information about the length of IAH. To eliminate insufficiencies (like labour-intensive, fluctuating character) Balogh and his colleagues developed and validated the continuous abdominal pressure measurement (CIAPM) technique (11). Continuous intra-abdominal pressure measurement requires only a three-way catheter and a transducer, and it is unnecessary to clamp the catheter and fill the urinary bladder („Balogh-Sugrue technique”). This method is used successfully to follow up critically ill patients in our department.

7. Treatment

To solve the problem of this serious clinical entity there are two opportunities: non-surgical (evacuate intraluminal contents and intra-abdominal space occupying lesions, improve

abdominal wall compliance, optimize fluid administration and systemic/regional perfusion) and surgical (early decompression) management.

Non-surgical management (14-15)

The treatment of ACS always means surgical decompression, but sometimes or if we have to treat only IAH we have the possibility for non-surgical way. The success is strongly depends on the etiology of the current IAH / ACS.

The main fields of the conservative management are:

- **evacuate intraluminal contents:** insert nasogastric and / or rectal tube
  initiate gastro- / coloprokinetic agents
  minimize enteral nutrition
  administration of enemas
  consider colonoscopic decompression
  discontinue enteral nutrition
- **evacuate intra-abdominal space occupying lesions:** abdominal ultrasound to identify
  abdominal CT to identify
  percutaneous catheter drainage
  consider surgical evacuation
- **improve abdominal wall compliance:** ensure adequate sedation / analgesia
  remove constrictive dressings
  avoid prone position, head of bed > 20 degree
  consider reverse Trendelenberg position
  consider neuromuscular blockade
- **optimize fluid administration:** avoid excessive fluid resuscitation
  aim for zero to negative fluid balance by day 3
  resuscitate using hypertonic fluids, colloids
  fluid removal through judicious diuresis
  consider hemodialysis / ultrafiltration
- **optimize systemic / regional perfusion**: goal-directed fluid resuscitation
  maintain APP (APP > 60 mmHg)
  hemodynamic monitoring to guide resuscitation
  vasoactive medications to keep APP > 60 mmHg

If IAP > 25 mmHg (and/or APP < 50 mmHg) and new organ dysfunction is present, patient's IAH / ACS is refractory to medical management. Strongly consider surgical abdominal decompression.

During the non-surgical management the CIAPM is strongly recommended!

Surgical management (14-15)

The surgical management of the IAH / ACS always means decompressing laparotomy with temporary abdominal closure.

If the patient has etiology for IAH / ACS it is very important to check the IAP continuously. When we have the criteria of ACS (see in the consensus definitions) we have to make the urgent decompression.

From now on it is important to continue with medical treatments and fluid resuscitation to reduce IAP and stabilize our patient. Having APP more than 60 mmHg and IAP < 12 mmHg consistently means that IAH has resolved.

If we have APP > 60 mmHg and IAP > 12 mmHg we have to go on with the non-surgical way. But in case of APP < 60 mmHg we have to perform / revise decompression again.

During the non-surgical management the CIAPM is strongly recommended!

Reconstruction after open abdomen management

After decompression it is necessary to consider the way and the time of closure.

The main possibilities (13,16,49,50,51):

- **primer closure**
- **primer skin or fascial closure** (Towel Clip Closure, direct suture)
- **temporary abdominal closure (TAC) technique**: let the abdomen open replacing it with tissue friendly mesh or other material temporarily (Bogota bag, VAC-Pac / vacuum-assisted closure, Gore-Tex patch, Whitman patch, biological patches, skin graft)
- **covering the abdominal organs with omentum** which may be epithelized in the future
- **staged abdominal closure** (elective hernias)

The closure is possible when the general condition is stable (24-48 hours) or after the definitive recovering (sometimes 6-12 months later).

The aim is to prevent abdominal distension, though an enormous elective hernia will be generated.

8. Special field: Pancreatitis and the ACS

In the last few decades the intra-abdominal hypertension and the abdominal compartment syndrome has arrived to get a special attendence in non-traumatologic patients. One of these cases is the ACS caused by severe acut pancreatitis. By the recent publications the number of acute and chronic pancreatitis is increasing. On one hand it is due to evaluation of the radiological diagnostic techniques, on the other hand we have more possibility to treat the severe cases. In spite of the fact, the successfull management of severe acut pancreatitis still needs an interdiscipline cooperation.

Clinical course

The aim of this chapter is not describing the clinics, diagnosis and treatment of acute pancreatitis, it is done by the authors of other chapters. But to understand the relation between IAH / ACS and pancreatitis I would like to shortly summarise the leading findings in the clinical picture:

The main symptom is the abdominal pain that usually starts in the gastric region but rapidly spreads to the complete abdomen. Often accompained with nausea, vomiting and paralytic ileus. Treating with minor analgesics often fails. In serious cases signs of shock like hypotension, tachycardia and cold sweat can be present. The paralytic ileus starts from the duodenum and jejunum and expand to the colon due to intensive fat necrosis and venous bowel congestion. Liberation of active pancreatic enzymes causes alterations in liver, kidneys and lungs and as the consequence of shock we face with acute renal failure.

A serious complication of pancreatitis is acute lung failure. The frequency depends on the severity of the basic disease.

Patients with imminent and manifest severe cases must be under continuous observation in the intensive care unit. The circulatory parameters, blood pressure and pulse rate, palpation and auscultation of the abdomen, supervision of fluid intake and output, central venous pressure and laboratory values of haemoglobin and haematocrit, the number of leucocytes, amylase levels in the serum and urine and/or lipase in the serum, serum calcium, blood glucose daily profile, serum potassium, urea, creatinine in the serum, arterial pO_2 pressure, acid-base-balance and sonography of the upper abdomen, the latter in 1-3 day intervals, are especially suitable for patient monitoring.

Although the acute necrotizing inflammation (except biliary causes) can be controlled by conservative treatments in most cases, surgical intervention often becomes necessary under emergency conditions.

The indication for early surgery applies upon failure of conservative treatment: the leading clinical symptoms during therapy inferred are: peritonitis, ileus, septic shock, MOF, incontrolable pain.

Due to an increase in the intra-abdominal volume the diaphragm is pushed upward. Compression of the thoracic space results in a restrictive ventilatory disorder. (And we are facing with Abdominal Compartment Syndrome!!!)

As the transverse colon is particulary affected and toxins from the large bowel can have adverse affects on the course of the disease.

In these cases of the ACS urgent laparostomy is recommended. The laparotomy remains wide open and is only covered by a foil (Bogota Bag or VAC-Pac). This allows repeated peritoneal lavage and necrosectomy too. The prolonged inflammatory process in the abdomen and the risk of recurrent ACS favors the use of gradual closure or delayed reconstruction of the abdominal wall. The early recognition of the syndrome and the wide decompression are crucial factors of the treatment of this very serious clinical enthity (52).

Although the above mentioned clinical findings of the severe acute pancreatitis are almost the same as we can see in patients suffering from ACS but do not confuse ACS with acute pancreatitis while it is only one of the possible causes of this serious symptoms. The two entities are very similar, this is the cause why the ACS is often late diagnosed and untreated which leads to early organ failure.

The current estimate of the prevalence of IAH in severe acute pancreatitis is about 40%, with about 10% overall progressing to ACS associated with increased hospital mortality rates. In the majority of cases, the development of IAH is rapid. The agressive fluid resuscitation and the inflammatory process in the retroperitoneum leading to the development of visceral edema and pancreatic ascites within days or even hours from admission.

The agressive fluid resuscitation is a „doble-edged weapon" : without strict control can induce the progress to ACS itself but it is proved that in the early phase the correct fluid therapy combined with prophylactic antibiotic treatment, surgical intervention, monitoring and management of organ dysfunctions, enteral nutrition and early endoscopic

spincterotomy in patients with common bile duct gallstone-induced pancreatitis have improved survival.

Loosing patients in the early phase is frequently due to not recognised and not treated ACS.

The presence of IAH can also be used as a predictor of the severity of acute pancreatitis, because by some clinical trials there is correlation with the increased IAP and severity of pancreatitis, mortality, peripancreatic infection rate, and need for surgical intervention (53).

The true prevalence of IAH in patients with severe acute pancreatitis is not known. We can find numerous studies in the bibliography of the prevalence of IAH among the patients treated by severe acute pancreatitis. De Waele and his team described in their study that 44% of the 41 study patients had IAP levels higher than 12 mmHg, and 4 patients (10%) had IAP levels higher than 25 mmHg with severe organ disfunction and undergoing surgical decompression (54). Keskinen treated 37 patients in the ICU for acute pancreatitis and 27% of the patients (n=10) had IAP levels higher than 25 mmHg (55). In a study of 297 patients from China presented that the overall incidence of IAH was 36% (56).

Severe acute pancreatitis is one of the most common diseases associated with IAH in the ICU environment.

Thus, it can be estimated that the overall prevalence of IAH in patients with severe acute pancreatitis is about 40%, and the frequency of ACS requiring surgical decompression is about 10% (53-56).

In a study comparing patients with or without ACS (IAP > 25 mmHg) treated in the ICU for severe acute pancreatitis the hospital mortality rate for patients with ACS was 50% compared with 15% in patients without ACS (55).

The following case reports from patientes treated in our department are to point out the importance of measurement of IAP:

First patient:

33-year-old man was taken to ICU with signs of acute pancreatitis after alcoholic abuse. Conservative treatment was started but on the second day the conditions of the man were getting worse. IAP measurement was started and we found IAP = 12 mmHg which rised to 26 mmHg during the next 3 days and developed MOF. During the intervention we found the tipical clinical picture of acute necrotizing pancreatitis with retroperitoneal necrosis. Although the decompression with Bogota Bag insertion was performed we lost the patient 2 days later. Concluding this case it is very important to pay attention to the early signs of IAP / ACS and the timing of the decompression.

Second patient:

37-year-old woman was taken to our hospital after alimentary abuse and with consequent abdominal pain. Examinations proved acute pancreatitis. Despite the appropriate conservative therapy her condition worsened, IAP reached 20 mmHg, respiratory distress and oliguria developed.

Laparotomy, lavage, drainage was performed. To prevent abdominal distension the abdomen was left open and organs were covered with a sterile urinary drainage bag (Bogota Bag). In the ICU 12 operations were performed during 108 nursing days. The IAP was

monitored permanently and when it reached the critical value necrectomy and lavage was performed. On the 116th nursing day she was sent home without complaints.

Her abdomen was covered with epithelized omentum which will be solved by a further operation (1-2).

9. Discussion

IAH and ACS are one of the major causes of organ failure and increased mortality (ACS with extremely high 38-71% mortality) among a wide variety of patient populations.

The pathophysiology of IAH and ACS is based on the chain reaction of physiological processes generated by the increased abdominal pressure which affects almost every organ and it could be fatal without correct diagnosis and treatment. It was originally noticed in traumatological cases (gunshot and stab wounds, intra-abdominal haemorrhage) when the extreme abdominal distension was related with a rapidly worsening condition and ended in ARDS, MOF and toxic shock. The same process is taking place in general surgical patients in spite of the different etiology. The leading etiological factor among general surgical critically ill patients is the acute pancreatitis. The cause of this phenomenon is the extremely increased IAP. The diagnosis was supported by the improved methods of monitoring IAP because the physical examination of the abdomen is far from accurate with a sensitivity of only 40%. The gold standard of the IAP monitoring is the CIAPM ensures diagnosis of ACS and the appropriate indication of the operation. The treatment consists of adequate fluid resuscitation and surgical decompression.

Intra-abdominal hypertension and abdominal compartment syndrome are frequent clinical findings among acute general surgical patients. Patients with comparable demographics and acute severity of illness are more likely to die if intra-abdominal hypertension or abdominal compartment syndrome is present. We conclude that the early recognition and surgical decompression is urgent.

„We must study and learn from the past and, at the same time, proactively „invent" the future. The future of IAH and ACS is in our hands. It is time to pay attention." (Cheatham, Ivatury, Malbrain, Sugrue) (53)

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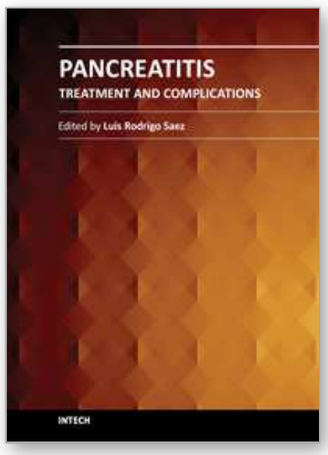

Pancreatitis - Treatment and Complications
Edited by Prof. Luis Rodrigo

ISBN 978-953-51-0109-3
Hard cover, 212 pages
Publisher InTech
Published online 02, March, 2012
Published in print edition March, 2012

Pancreatitis may be acute or chronic. Although they can be caused by similar aetiologies, they tend to follow distinct natural histories. Around 80% of acute pancreatitis (AP) diagnoses occur as secondary to gallstone disease and alcohol misuse. This disease is commonly associated with the sudden onset of upper abdominal that is usually severe enough to warrant the patient seeking urgent medical attention. Overall, 10 to 25% of AP episodes are classified as severe, leading to an associated mortality rate of 7 to 30%. Treatment is conservative and consists of general medical support performed by experienced teams, sometimes in ICUs. Although most cases of acute pancreatitis are uncomplicated and resolve spontaneously, the presence of complications has significant prognostic importance. Necrosis, hemorrhage, and infection convey rates of up to 25%, 50%, and 80% mortality, respectively. Other complications such as pseudocyst formation, pseudoaneurysm formation, or venous thrombosis increase morbidity and mortality to a lesser degree. The presence of pancreatic infection must be avoided.

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