

Abdominal Compartment Syndrome in Severe Acute Pancreatitis – When to Decompress?

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Abstract

Intra-abdominal hypertension is increasingly reported in patients with severe acute pancreatitis, and is caused by several factors, including visceral edema and ascites associated with massive fluid resuscitation, paralytic ileus and retroperitoneal inflammation. There is a strong relation with early organ dysfunction and mortality in these patients, which makes intra-abdominal hypertension an attractive target for intervention. Several reports conclude that this phenomenon occurs within the first 5 days after admission, and that the kinetics of IAH is important: patients with persistent IAH seem to be at the highest risk for mortality. Several strategies to reduce IAP have been developed, and given the pathophysiology, percutaneous drainage of ascites is a first logical step. However, if conservative measures fail to reduce IAP in a setting with ongoing or worsening organ dysfunction, abdominal decompression is recommended.

Key Words

Acute pancreatitis · Necrotizing pancreatitis · Intra-abdominal pressure · Intra-abdominal hypertension · Abdominal compartment syndrome · Decompressive laparotomy

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Introduction

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been described most often in patients with abdominal trauma or after emergency abdominal surgical procedures such as aortic aneurysm repair [1]. The IAH is defined as a

sustained or repeated pathologic elevation of the intra-abdominal pressure (IAP) above 12 mmHg. The ACS is described as the sustained elevation of IAP above 20 mmHg in combination with newly developed organ dysfunction [2]. Three different types of abdominal compartment syndrome are described: (a) *primary ACS* is caused by a disease process in the abdominopelvic region, and comprises most of the typical cases of ACS; (b) *secondary ACS* is caused by conditions that do not originate from this region, and is increasingly recognized in various condition associated with critical illness, most often after large volume resuscitation such as burns or severe sepsis [3]; (c) *recurrent ACS* develops after treatment of prior ACS.

Although the typical symptoms of ACS, i.e. rapidly evolving multiple organ dysfunction syndrome (MODS) (most often a combination of respiratory failure, hemodynamic compromise and acute renal failure) are often found in patients with severe acute pancreatitis, it was not until recently that the importance of IAH has been recognized. Several strategies to reduce IAP in those patients have been studied, but the obvious question remains: *when should I decompress?*

In this review, we will first give an overview of the current understanding of IAH and ACS in the development of complications in patients with severe acute pancreatitis, because insight in the natural history of IAH in those patients is essential to answer this question, and then we will discuss the role of strategies aimed at reducing the IAP in patients with IAH and severe acute pancreatitis.

Epidemiology of IAH in Severe Acute Pancreatitis

Insights in the true incidence of IAH in patients with severe acute pancreatitis are hampered by the lack of uniform definitions of IAH and ACS until recently.

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Also, guidelines for reliable IAP monitoring have changed, as instillation of larger volumes of saline for IAP monitoring may lead to falsely elevated values [4], and in most studies selection bias cannot be completely excluded, as routine measurement of IAP is rarely performed [5]. Nevertheless, the high incidence rates of both IAH and ACS, and the mortality associated with both the problems are significant. A clear causal relationship could not be demonstrated, but the case reports and case series reporting excellent outcomes in pancreatitis patients undergoing abdominal decompression that will be discussed below [6–11], suggest that IAP may be a target for early intervention. Table 1 gives an overview of the incidence of IAH and ACS according to the definitions used in recent studies.

Severe acute pancreatitis is responsible for a considerable part of the cases of ACS described in a dedicated 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%) [12]. In a study reporting on the problem of secondary ACS in medical patients, severe acute pancreatitis was the most prevalent cause of secondary ACS (5/13 patients, 38%) [13]. Obviously, the incidence may vary according to the type of ICU, and more importantly, the incidence of acute pancreatitis in the population, which may differ considerable from country to country.

When it develops, IAH develops early in the course of the disease. Pupelis et al. reported an elevated IAP during the first 3–5 days, and in patients with a favorable outcome a decrease from day 5 onwards [14]. De Waele et al. reported that IAH developed after a median of 1 day, and was maximal on the second day. IAH persisted for 6 days (interquartile range 3–8 days) [9]. Keskinen et al. found that in non-survivors, IAP remained elevated during the first week; survivors had high IAP on day 1, but showed a progressive decrease in IAP in the following days [15]. Apparently, the first 5 days after ICU admission seem

to be critical. All authors found that IAP developed within this time frame, with a progressive decrease in IAP in patients who survived, and a parallel improvement in organ dysfunction. From this, it can be concluded that organ dysfunction related with IAH is reversible if the duration of IAH is limited to maybe 2 or 3 days.

The IAH may also develop later in the course of the disease, when infectious complications arise, but data on this problem are non-existent so far.

The outcome of severe acute pancreatitis patients with IAH or ACS is poor. In all studies on this topic, mortality was higher in patients with IAH. 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 [15]. There was a clear correlation between the maximum IAP value within the first 2 weeks and the mortality rate. Also Pupelis et al. found that in patients with IAP higher than 25 mmHg had a significantly increased mortality from 0 to 36% [16].

Pathophysiology

The development of IAH in patients with severe acute pancreatitis is initiated by the inflammatory process in the retroperitoneum leading to the development of pancreatic and visceral edema, peripancreatic acute fluid collections and ascites. The combination of capillary leakage and large volume fluid resuscitation further increases the development of intra-abdominal edema. Additionally, paralytic ileus and upper gastrointestinal tract obstruction by the pancreatic collections may also aggravate IAH. Reduced abdominal wall compliance due to edema may also play a role.

In some patients, complications related to the pancreatitis may contribute to IAH; arterial or venous bleeding from the necrotic and inflamed tissue can contribute to acute increase of IAP in patients with

Table 1. Incidence of IAH and ACS and reported mortality. IAP: intra-abdominal pressure; IAH: intra-abdominal hypertension; ACS: abdominal compartment syndrome; NA: not available.

First author	Publication year	Definition of IAH	Incidence of IAH	Mortality in IAH vs. non-IAH	Definition of ACS	Incidence of ACS (n)	Mortality in ACS vs. non-ACS
Pupelis [14]	2002	NA	NA	NA	IAP > 25 mmHg	18/71	28 vs. 2%*
De Waele [9]	2005	IAP > 15 mmHg	21/27 ^a	38 vs. 16%	NA	NA	NA
Keskinen [15]	2007	IAP > 12 mmHg	31/37 ^a	NA	IAP > 20 mmHg + new organ dysfunction	18/37 ^a	NA

* p value < 0.05

^aSelection of patients with IAP monitoring (based on clinical suspicion)

severe acute pancreatitis. It should be mentioned that this rarely occurs early, but IAP monitoring may add to the diagnosis of a new intra-abdominal complication when IAP elevation occurs later in the course of the disease.

IAH may also contribute to the development of pancreatic necrosis. Elevated IAP causes intestinal hypoperfusion even at levels as low as 8–12 mmHg [17]. In the setting of severe acute pancreatitis, pancreatic perfusion is affected by the disease process itself, and IAH may add to the development of pancreatic hypoperfusion and eventually pancreatic necrosis. Moreover, the observed problem of bacterial translocation in patients with IAH and abdominal compartment syndrome [18] may apply to patients with severe acute pancreatitis, which may imply that IAH also may induce subsequent infection of the necrosis. Animal studies have shown an increased rate of bacterial translocation in acute pancreatitis [19], but the exact role of IAH in humans remains to be elucidated; however, based on these data it can be hypothesized that IAH is involved to some extent in both the development of necrosis and infection of the necrosis, two major determinants of outcome [20].

Recently, a subset of patients with severe acute pancreatitis who develop early MODS has been described [21, 22]. Typically, these patients develop MODS within a few days after the start of symptoms and require aggressive fluid resuscitation. This clinical picture of “early severe acute pancreatitis” has been associated with increased mortality [21, 22]. Although the exact mechanisms of early MODS are not completely understood, IAH may be a potential contributing factor to the development of early organ failure seen in patients with severe acute pancreatitis as first suggested by both Pupelis et al. [23] and Gecelter et al. [6] in 2002. Tao et al. reported an incidence of 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 [22].

IAH and IAP as Predictors of Severity

It has been suggested that IAP can also be used as a predictor of the severity of the episode of acute pancreatitis. In a study of 45 patients presenting 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. The development of a maximum IAP > 14 mmHg could also adequately predict mortality (AUROC 0.837) [24].

Although the same authors also demonstrated high maximal IAPs in patients who develop all of the typical complications of severe acute pancreatitis such as peripancreatic collection, infected necrosis and MODS, it is highly questionable whether IAP can be used to guide patient disposition in the emergency room or within the first few hours or days. Although most patients may develop IAH early, it is also caused by fluid resuscitation, something which is rarely done outside an ICU or high dependency unit; therefore IAH is often an epiphenomenon of aggressive ICU treatment. Absence of IAH at admission should not be used to deny ICU admission.

The sensitivity and specificity reported for the development of intra-abdominal collections (sensitivity 78%, specificity 86%) and the need for surgery (sensitivity 88%, specificity 86%) using the same cutoff of 14 mmHg, may be indicators that IAH does play a role in the development of pancreatic necrosis and infection as well.

Clinical Manifestation and Diagnosis

The symptoms caused by IAH in patients with acute pancreatitis are not very different from other conditions associated with IAH. Hemodynamic instability requiring vasoactive drugs, acute renal failure and respiratory failure are the most obvious clinical signs and symptoms that have been associated with IAH. The association between IAH and development of organ dysfunction in severe acute pancreatitis is well documented. De Waele et al. showed that there was a 95% incidence of respiratory failure, 91% cardiovascular and 86% acute renal failure rate in patients with IAP of 15 mmHg or higher [9]. Rosas et al. found higher mean and maximal IAPs in patients who developed MODS, and reported a sensitivity of 86% and specificity of 84% for an IAP $\geq$ 15 mmHg to predict MODS [24].

Treatment Options for IAH and ACS

The development of IAH in patients with severe acute pancreatitis is evidently an important problem, as it is associated with organ dysfunction and mortality. Therefore, IAP should be measured routinely in patients admitted to the ICU with severe acute pancreatitis, and IAP should be considered a target for intervention in all patients [5]. Non-surgical measures to reduce IAP should be considered first, and when ineffective, surgical approaches may be necessary in patients with persistent organ dysfunction [25].

Non-surgical Treatment

In patients with large volumes of pancreatic ascites, percutaneous drainage of the intraperitoneal exudate can lead to a significant drop in IAP, as reported by Sun et al. In a prospective trial, IAP decreased in 45 patients assigned to the intervention group from 29 to 14 mmHg one day later by draining a mean volume of 1,817 ml ascites, but unfortunately, the data of the control group are not reported [26]. APACHE II scores on days 2 and 5, as well as the mortality rate was significantly lower (10 vs. 21%).

The CT scan with intravenous contrast is indicated in patients with hemodynamic instability in order to evaluate the extent of necrosis and fluid exudation for possible drainage and can detect a major bleeding source. Angiography and endovascular occlusion therapy can be used in patients with a visualized bleeding.

Oda et al. studied the effect of continuous hemodiafiltration in 17 patients with severe acute pancreatitis who had a mean IAP of 14 mmHg. The authors found that IAP decreased after initiation of continuous venovenous hemodiafiltration, and did not develop organ dysfunction, but it remains unclear if the observed effect was caused by the treatment itself [27]. Other strategies aimed at reducing IAP such as nasogastric suction, or the use of neuromuscular blockers may be considered [28, 29].

As fluid resuscitation is one of the elements that contribute to IAH, the use of crystalloids for fluid resuscitation can be questioned. Although no prospective data are available, there are some parallels between the development of IAH in burn patients and patients with severe acute pancreatitis. Two prospective trials in burn patients have shown reduced incidence of IAH and less organ dysfunction in patients who were fluid resuscitated with albumin or hypertonic saline [30, 31].

Surgical Treatment

Decompressive laparotomy has been shown to effectively reduce IAP and reverse the symptoms typically associated with ACS [32]. In patients with severe acute pancreatitis and IAH, intra-abdominal fluid collections may not always be present and therefore decompressive laparotomy and temporary abdominal closure is the most effective way of decreasing IAP [7, 15, 33].

The most common approach to decompressive laparotomy is through a long, vertical midline incision, but a transverse incision to anticipate later pancreatic surgery may also be used. Because of the risk of

introducing infection to the peripancreatic space and the absence of demarcated pancreatic necrosis, it is not necessary and even dangerous to explore the pancreas at this stage. Intractable bleeding complicating the course in a patient who underwent combined decompression and necrosectomy has been described [9]. The open abdomen is covered with one of the various dressings described for covering the open abdomen [34], a topic which is discussed extensively by Lepäniemi in this issue.

Although the role of decompressive laparotomy has not been studied prospectively, multiple case series report impressive survival rates when the abdomen is timely decompressed. In one of the largest series on the topic with 18 patients who were decompressed, Tao et al. reported survival rates as high as 84% in patients with MODS and severe acute pancreatitis [33].

A promising new method based on subcutaneous linea alba fasciotomy preserving the skin as a cover has been described, and combines the advantage of decreasing the IAP and avoiding the disadvantages associated with the open abdomen [10]. Utilizing three transverse 2–4 cm long skin incisions placed about 10 cm below the xiphoid and about 5 cm above and below the umbilicus, the subcutaneous tissue is incised and the linea alba divided vertically in the midline under visual control using a scalpel and scissors. In most cases, the diastase created by the linea alba fasciotomy is about 8–10 cm, and the decrease in IAP about 10–15 mmHg.

If decompression is needed more than 2–3 weeks after the onset of the disease and there is evidence of extensive necrosis on a CT scan or established infection of peripancreatic necrosis, it is feasible to perform a necrosectomy in conjunction with the decompressive laparotomy. In selected patients with extensive retroperitoneal fluid collections, a lumbotomy may provide access to the retroperitoneal space, and allow evacuation of pancreatic necrosis as well.

The management of the open abdomen following decompression in severe acute pancreatitis is challenging. The best currently available technique is the utilization of the vacuum-assisted closure technique aiming for gradual closure of the abdominal wall. The use of a vacuum assisted closure system guarantees a perfect seal of the peritoneal cavity, avoiding possible superinfection of the pancreatic or peripancreatic necrosis.

Conclusion

IAH seems to have a significant role in contributing to the early MODS, subsequent complications and mor-

tality in severe acute pancreatitis. IAP monitoring is mandatory for all patients who develop organ dysfunction, and IAP should be a target for intervention when IAH and organ dysfunction persist. Surgical decompression should be considered in all patients with persistent organ dysfunction after 3 days or later.

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