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Intra-Abdominal Hypertension in Acute Pancreatitis

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Abstract The incidence of intra-abdominal hypertension (IAH) in patients with severe acute pancreatitis (SAP) is approximately 60–80%. It is usually an early phenomenon, partly related to the effects of the inflammatory process, causing retroperitoneal edema, fluid collections, ascites, and ileus, and partly iatrogenic, resulting from aggressive fluid resuscitation. It also can manifest at a later stage, often associated with local pancreatic complications. IAH is associated with impaired organ dysfunction, especially of the cardiovascular, respiratory, and renal systems. Using current definitions, the incidence of the clinical manifestation, abdominal compartment syndrome (ACS), has been reported as 27% in the largest study so far. Despite several intervention options, the mortality in patients developing ACS remains high: 50–75%. Prevention with judicious use of crystalloids is important, and nonsurgical interventions, such as nasogastric decompression, short-term use of neuromuscular blockers, removal of fluid by extracorporeal techniques, and percutaneous drainage of ascites should be instituted early. The indications for surgical decompression are still not clearly defined, but undoubtedly some patients benefit from it. It can be achieved with full-thickness laparostomy (midline or transverse subcostal) or through a subcutaneous linea alba fasciotomy. Despite the improvement in physiological variables and significant decrease in IAP, the effects of surgical decompression on organ function and outcome are less clear. Because of the significant morbidity associated with surgical decompression and the management of the ensuing open abdomen, more research is needed to define better the appropriate indications and techniques for surgical intervention.

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Introduction

Although intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) were long considered a problem only to occur in surgical patients, they have been identified as a cause of organ dysfunction in several patient categories without apparent abdominal surgical condition, such as burns and sepsis. This category of ACS has been defined as "secondary ACS." "Primary ACS" refers to ACS due to an intra-abdominal cause, and ACS associated with severe acute pancreatitis (SAP) is a common cause of primary ACS.

Whereas the clinical finding of a tense abdomen are well known in patients with acute pancreatitis, the first reports of the association of IAH and pancreatitis were only published in 2002 [1, 2]. Several authors reported small patient series with overt ACS and acute pancreatitis at several stages of the disease, and already at that point, decompressive laparotomy was suggested as a therapeutic intervention in these patients. During the next year, the association between IAH and SAP was confirmed repeatedly in larger studies, and during the last year, two prospective studies of unselected patients with acute pancreatitis were published, providing better insight of the epidemiology of the problem and the dynamics of IAH in pancreatitis. Also, several investigators have studied the effect of various interventions to lower IAP (intra-abdominal pressure) in patients with SAP, using both medical and surgical interventions.

IAH seems to be a frequent problem of SAP. There also is increasing evidence that patients with so-called "early SAP" (patients with early organ dysfunction and high mortality rate) actually suffer from ACS and, therefore, represent a subgroup of patients who require a different approach that specifically targets elevated IAP.

We attempt to provide an overview of the current insights of the epidemiology of IAH in patients with SAP, describe the mechanisms of why patients develop IAH, summarize the clinical effects of IAH in patients with SAP, and review the treatment options for those patients.

Mechanisms of disease

There are several reasons why patients with SAP have increased IAP, although their relative contribution may differ from patient to patient and change over time. Patients with severe acute pancreatitis usually present with an enlarged pancreas, with fluid collections often present in the retroperitoneum. During the days that follow, this inflammatory process extends to the whole retroperitoneum and mesentery, and necrosis may develop, adding to the intensity of the problem. Ascites may develop, sometimes in large quantities, as well as visceral edema, which further increase the intra-abdominal volume. Ileus also is a common finding in SAP, and due to mechanical obstruction of the duodenum, gastric dilation can be very impressive; this increase in intraluminal air contributes to the already increased extraluminal volume increase.

Fluid resuscitation also plays an important role in the development of IAH. Due to capillary leakage and the diffuse intra-abdominal inflammatory process, fluid will accumulate rapidly in the abdomen and further increase IAP. Resuscitation-induced edema of the abdominal wall causes a decrease in compliance, which results in higher IAP when the intra-abdominal volume increases.

IAH itself can initiate a vicious circle, as decreased intestinal perfusion will occur even at IAP as low as 12 mmHg, and as in pancreatitis the microcirculation of the pancreas is already affected, a decrease in arterial perfusion will further compromise oxygen delivery. Also infection of pancreatic necrosis, another much feared complication for patients with SAP, may be related to increased IAP. Animal studies have shown an increased rate of bacterial translocation in acute pancreatitis [3]. Bacterial translocation has been described in patients with ACS, and this may apply to patients with SAP [4]. Recent clinical studies provide new evidence for the association between elevated IAP and infection: in one study, pancreatic infection occurred in 60% of patients with ACS, whereas it was rare in patients with lower IAP [5]. In another study, the maximum IAP was 19 mmHg in patients who developed infected pancreatic necrosis, whereas it was 11 mmHg in patients with an uncomplicated course [6]. Therefore, it is likely that IAH is involved to some extent in both development of necrosis and infection of the necrosis—two major determinants of outcome [7].

At a later stage, complications related to pancreatitis and pancreatic necrosis, such as pancreatic abscess or pseudocysts, or bleeding from venous or arterial origin, may cause IAH. In these cases, monitoring of IAH may help in the early detection of new intra-abdominal problems.

IAH and ACS in SAP: facts and figures

Data on the magnitude of the problem of IAH and ACS in pancreatitis remain scarce. Whereas the first reports of IAH in patients with SAP only included selected patients in whom IAP was monitored for some reason—possibly leading to overestimation of the problem—recent analyses of unselected patient groups has provided better insight about the true incidence of IAH and ACS. In patients admitted to an ICU or HDU, both Chen et al. [5] and Al-Bahrani et al. [8] reported an incidence of approximately 60%, using the current definitions of IAH according to the World Society of Abdominal Compartment Syndrome (WSACS) [9]. Earlier, retrospective studies had reported figures as high as 78–84% in selected patients (Table 1) [10, 11]. There was more discrepancy in the reported incidence of ACS, but this was probably related to the

Table 1 Epidemiology of IAH and ACS in patients with SAP as reported in the literature

First author	Publication year	IAP monitoring	Definition of IAH	Incidence of IAH (%)	Definition of ACS	Incidence of ACS (%)
Pupelis et al. [1]	2002	Selected	NA	NA	IAP > 25 mmHg	18/71 (25%)
De Waele et al. [11]	2005	Selected	IAP > 15 mmHg	21/27 (78%)	NA	NA
Keskinen et al. [10]	2007	Selected	IAP > 12 mmHg	31/37 (84%)	IAP > 20 mmHg + new organ dysfunction	18/37 (49%)
Chen et al. [5]	2008	Unselected	IAP > 12 mmHg	44/74 (59%)	IAP > 20 mmHG + new organ dysfunction	20/74 (27%)
Al-Bahrani et al. [8]	2008	Unselected	IAP > 15 mmHg	11/18 (61%)	IAH plus organ dysfunction	10/18 (56%)

IAP intra-abdominal pressure, *IAH* intra-abdominal hypertension, *ACS* abdominal compartment syndrome, *NA* not available

difference in definition of ACS. When the current WSACS definition was used, the reported incidence of ACS was 27% in the largest study so far—also lower than previously reported [1, 5, 10].

When it occurs, IAH is consistently reported to be an early phenomenon. Up to 70 percent of patients have been reported to have IAH on admission to the ICU, and when it was not present, it developed within the first days. Reported mean IAP values remain high during the first 3 to 5 days after admission, and slowly decrease afterwards. However, in nonsurvivors continued high IAP values have been observed, whereas in patients with a favorable outcome, IAP decreases from day 5 onward [12]. Keskinen et al. [10] found that survivors had a progressive decrease in IAP after admission; in nonsurvivors, IAP remained high during the first week.

IAH may occur at a later stage in the disease, when local complications in the pancreatic region occur, but there are no data so far on this aspect of the problem of IAH in patients with SAP.

Although the reported incidence may vary, the reported mortality rates of patients with ACS are high, despite interventions in some studies to decrease IAP [8]. Some authors found that in IAH patients, mortality is higher than in patients without IAH [11]; however, but Chen et al. found only an increased mortality in ACS patients; IAH patients (i.e., high IAP without organ dysfunction) had similar outcomes as patients without IAH [5]. The most recent studies report a 50% and 75% mortality rate in patients who develop ACS, but it is not clear how the interventions may have played a role in determining outcome [5, 8]. These data are comparable with other patient categories undergoing decompressive laparotomy for ACS [13]. Also, the length of stay is significantly longer in patients who develop IAH [8].

IAH was associated with impaired organ dysfunction in all studies on this topic, irrespective of the scoring system used. Numerous case reports also described improved organ function within hours after interventions to reduce IAP, and the same has been observed in prospective studies; IAP reduction varied from 11–17 mmHg in patients who underwent decompressive laparotomy [5, 8]. This supports the hypothesis that IAH adds to the impaired organ function in patients with SAP; of course, other reasons for organ dysfunction are present in patients with IAH, and it is logical that decompression could not completely reverse this process.

The evolving insight concerning the problem of ACS in SAP also sheds new light on the problem of "early SAP." In a study of 297 patients with SAP, a subset of patients who develop early MODS has been described [14]; typically, these patients develop MODS within a few days after the start of symptoms and require aggressive fluid resuscitation. This clinical picture of "early SAP" is associated with increased mortality [14]. Although the exact mechanisms of early MODS are not completely understood and may involve multifactorial etiologies associated with a severe proinflammatory response, IAH may play an important role in the development of early organ failure seen in patients with severe acute pancreatitis as first suggested by both Pupelis et al. [1] and Gecelter et al. [2] in 2002. Tao et al. [14] reported an incidence of ACS (defined as 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.

Management of IAH in patients with SAP

Because IAH is clearly related to organ dysfunction in acute pancreatitis, and because it is reversible and to some extent preventable, IAH should definitely be considered a therapeutic target. IAP should be measured regularly in patients with SAP, at least every 4 hours or whenever the clinical condition of the patient deteriorates [15]. Nonsurgical measures to reduce IAP should be considered first, and when ineffective, surgical approaches may be necessary in patients with persistent organ dysfunction.

Prevention

Prevention should always be considered, because therapeutic interventions may further increase IAP. Fluid resuscitation is a very important cause of IAH when high volumes are necessary. In these patients, especially the use of crystalloids should be avoided. Although no studies are available for patients with SAP, prospective studies in burn patients have demonstrated that IAP is lower when plasma or hypertonic saline-based resuscitation schemes are used (compared with crystalloids) [16, 17]. Also when IAP is high, further deterioration may be avoided by using crystalloids judiciously or by replacing crystalloids with colloids or albumin.

Nonsurgical interventions

Because ileus and gastric dilatation are frequent in SAP, a first logical step is nasogastric decompression using a nasogastric tube. Although often recommended in other patients with IAH, it is not clear whether the use of prokinetics has any effect on IAP.

Percutaneous drainage of intraperitoneal fluid collections is a simple and more effective way to reduce the intra-abdominal volume. In a prospective study, Sun et al. [18] reported a 15 mmHg decrease in IAP from 29 mmHg in 45 patients assigned to the intervention group by draining a

mean volume of 1817 ml ascites, but unfortunately, the data of the control group are not reported. APACHE II scores on days 2 and 5, as well as the mortality rate, were significantly lower (10% vs. 21%).

Neuromuscular blockers also are an effective means to reduce IAP and also may be considered in this context, but as the deleterious effects of these agents have been well documented, short-term use only is recommended [19].

Because fluid overload undoubtedly contributes to IAH in over-resuscitated patients, any intervention to remove fluid can be beneficial. Administration of loop diuretics often is of little use, because acute kidney injury often is one of the first organ dysfunctions to set in. Removal of fluid by extracorporeal techniques is more effective in rapidly removing excess fluid and seems to have an immediate effect, related to the extent of fluid removal [20]. Oda et al. [21] 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 whether the observed effect was caused by the treatment itself.

All of the above interventions may to some extent help to reduce IAP. In a recent study, Chen et al. found that a combination of nonsurgical interventions was able to reduce IAP in 7 of 20 patients with ACS, thereby avoiding decompressive laparostomy in all of them [5].

Surgical decompression techniques

The most commonly used method for surgical decompression is the midline laparostomy [13]. All layers (skin, fascia, peritoneum) are divided through a vertical midline incision extending from the xiphoideum to the pubis with a few centimeters of fascia left intact at both ends to facilitate subsequent closure or late reconstruction. Alternatively, a bilateral subcostal incision few centimeters below the costal margins can be used to perform a full-thickness laparostomy [22]. A third method utilizes three, short, horizontal skin incisions to perform a subcutaneous linea alba fasciotomy (SLAF) with the peritoneum left intact [23].

Surgical decompression: does it work?

In a critical analysis of 10 articles, including total of 161 patients who underwent surgical decompression via a midline laparostomy, the mean reported IAP before surgical decompression was 34.6 mmHg and decreased to 15.5 mmHg after decompression ($p < 0.001$) [13]. In a case report that described the use of transverse decompressive laparostomy for a patient with severe acute pancreatitis, the IAP decreased from 23 mmHg to 10 mmHg [22].

The original report of two patients utilizing the SLAF method showed a decrease of IAP from 30 mmHg to 14 mmHg and 35 mmHg to 23 mmHg, respectively [23]. Another report showed a decrease from 27 mmHg to 11 mmHg [24].

In an analysis of 26 patients with severe acute pancreatitis who underwent surgical decompression for ACS, 18 patients underwent midline laparostomy, 1 patient had a transverse laparostomy, and 7 patients had SLAF [25]. The median (interquartile range) decrease of IAP was 16 (range, 9–21) mmHg after full-thickness laparostomy and 12 (range, 10–13) mmHg after SLAF ($p = 0.31$).

It seems that all three techniques described result in a significant decrease of the IAP. It is noteworthy that even if the IAP increases after SLAF, the initial decompressive effect is big enough to maintain adequate abdominal perfusion pressure of >60 mmHg and avoid open abdominal decompression [24].

Surgical decompression: does it help?

The effects of abdominal decompression on organ functions were summarized in a collective review of 250 patients who underwent midline laparostomy [13]. Considering the significant interstudy variation and incomplete data, decompression had a positive effect on hemodynamic, respiratory, and renal function parameters. Filling pressures (central venous pressure and pulmonary artery pressure) decreased, but this was most likely caused by the direct effect of the decrease in IAP on the thoracic cavity. Cardiac function improved in the majority of patients, but the largest study reported no improvement in cardiac index. There was an improvement in PaO_2/FIO_2 ratio and decrease in peak airway pressure, but the respiratory function remained severely impaired in most patients. Significant improvement in the urinary output was observed in all but two papers.

In a single-institution experience of 26 patients from Helsinki University, the median (interquartile range) preoperative SOFA (Sequential Organ Failure Assessment) score was 12 (range, 10–15), with individual organ system scores of ≥3 in 92% patients in respiratory, 88% in cardiovascular, and 54% in renal systems, respectively [25]. The PaO_2/FIO_2 ratio increased in 50% and decreased in 50% of the patients. Daily urinary output increased by >200 ml in seven patients (27%), and three patients avoided renal replacement therapy. However, there was no significant change in the overall SOFA score before and after decompression.

In a recent study from China with 13 patients who underwent percutaneous ($n = 8$) or surgical decompression

($n = 5$), there were statistically significant improvements in cardiovascular, respiratory, and renal parameters, including decreases in base deficit and lactate levels [5].

The overall mortality among the 250 patients from a collective review was 49% [13]. Among the 26 patients from Helsinki University [25], the overall mortality rate was 46% with preoperative renal failure ($p = 0.045$), lower preoperative IAP ($p = 0.039$), and late (median, 7 days) decompression ($p = 0.005$) associated with increased risk of death. It is noteworthy that all eight patients who underwent surgical decompression more than 3 days after admission died.

Surgical decompression: is it safe?

Fatal, uncontrollable hemorrhage from the retroperitoneum is one of the early complications of decompressive laparostomy, especially if associated with simultaneous necrosectomy [11]. Currently, the major source of morbidity after decompressive laparostomy is associated with the management and complications of the open abdomen. Among 26 patients who underwent full-thickness laparostomy or subcutaneous fasciotomy, 1 to 18 (median, 5.5) reoperations were required, including 16 patients (62%) who underwent necrosectomy [25]. Of the 21 patients with laparostomy, 17 survived to abdominal closure. Delayed primary fascial closure was achieved in seven patients (5–31 days postoperatively), and a planned hernia strategy was used in the remaining ten patients with split-thickness skin grafting performed 22–40 days after decompression. Intra-abdominal infection was diagnosed in 18 patients (86%) with open abdomen, but in only 1 of 5 after subcutaneous fasciotomy ($p = 0.01$).

The details of the management of the open abdomen are beyond the scope of this review. It should be noted that the decision to perform surgical decompression for ACS in severe acute pancreatitis invariably results in prolonged course, multiple reoperations, and high risk of complications, which requires adequate knowledge and experience in the treatment options for the ensuing open abdomen [26]. Considering the complicated and often fatal outcome of patients who have persistent open abdomen after multiple reoperations—often with entero-atmospheric fistulae and persistent infection—the "hostile abdomen" scenario, extreme caution, and care should be administered when managing patients with open abdomen [27].

Indications for surgical intervention

There is no uniform consensus on the indications for surgical decompression in ACS associated with severe acute pancreatitis. Despite improvements in several physiological variables, the exact effect on organ dysfunction is not clear. Although it would be tempting to assign a clear IAP threshold above which surgical decompression is indicated, the IAP value is probably not the only parameter that should be considered [14, 26]. When nonsurgical interventions fail to turn around the progressive deterioration of organ dysfunctions in the presence of fulminate ACS, surgical decompression should be considered. It seems that early decompression is associated with lower mortality and subcutaneous fasciotomy with lower risk of subsequent intra-abdominal infection.

Conclusions

The presence of intra-abdominal hypertension is increasingly reported in patients with severe acute pancreatitis, partly related to the effects of the inflammatory process, causing retroperitoneal edema, fluid collections, ascites, and ileus, and partly resulting from our interventions, especially aggressive fluid resuscitation. It seems to be an early phenomenon and associated in some patients with early multiple organ failure, but it also can manifest at a later stage, often associated with local pancreatic complications. With improvement of nonsurgical intervention techniques and understanding of the mechanism of ACS in this patient group, the need for surgical interventions can be decreased. When necessary, early surgical intervention seems to be more beneficial. Despite the improvement in physiological variables and significant decrease in IAP, the effects on organ function and outcome are less clear. Because of the significant morbidity associated with surgical decompression and the management of the ensuing open abdomen, more research is needed to define better the appropriate indications and techniques for surgical intervention.

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