

Management of Abdominal Compartment Syndrome in Acute Pancreatitis

Jan J. De Waele

Department of Critical Care Medicine, Ghent University Hospital De Pintelaan 185, 9000 Gent,
Belgium

e-mail: Jan.DeWaele@UGent.be

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Abstract

The incidence of intra-abdominal hypertension (IAH) in patients with severe acute pancreatitis (SAP) is high (60-80% depending on the population studied). It is typically an early phenomenon, and caused by the inflammatory process in the pancreas as well as ascites, ileus, and aggravated by fluid resuscitation. Deterioration to full-blown abdominal compartment syndrome (ACS), has been reported in about 1 out of 3 patients with IAH. Morbidity and mortality is consistently higher in patients with IAH and mortality in patients developing ACS remains high. Prevention through judicious use of fluid resuscitation is a key factor, and nonsurgical interventions, such as nasogastric decompression, or percutaneous drainage of ascites should be instituted early when intra-abdominal pressure (IAP) increases. Surgical decompression remains debated, but may be beneficial when timed appropriately. Open abdomen management with negative pressure therapy results in acceptable morbidity when managed appropriately. IAH has evolved from an incompletely understood and poorly managed complication in SAP to a preventable and treatable condition that should be understood by all physicians involved in the care of these patients.

1. Introduction

Insights in the diagnosis and management of acute pancreatitis are evolving with many treatment strategies that were once considered the standard of care eventually being discarded as non-beneficial or even harmful (10). Both medical treatment and surgery have advanced significantly but morbidity and mortality of severe acute pancreatitis remains high and the course of the disease is often protracted in severe cases.

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been found to be significant contributors to organ dysfunction in a variety of critically ill patients, and several strategies have been developed to prevent and treat ACS (8).

Patients with severe acute pancreatitis appear to be at an increased risk of IAH due to the several mechanisms that occur in pancreatitis as well as the treatment they receive. Our understanding of both the development of IAH and ACS in SAP has advanced significantly and ACS has evolved from an incompletely understood and poorly managed complication in SAP to a preventable and treatable condition that should be understood by all physicians involved in the care of these patients.

Table 1. Contributors to IAH and ACS in acute pancreatitis

Intra-abdominal volume increase
Pancreatic and peripancreatic edema (often fueled by fluid resuscitation) Ascites Ileus
Abdominal wall compliance decrease
Abdominal wall edema Abdominal pain

2. Definitions

IAH has been defined as an intra-abdominal pressure (IAP) of 12mm Hg or higher (19); this is the threshold at which organ dysfunction may set in, although it is often undetectable unless specifically sought for. Oxygen exchange for instance may be impaired but compensatory mechanisms may be effective and oxygen saturation may not be changed.

In case of ACS, the IAP is 20 mmHg or higher with clinically evident new organ dysfunction; acute kidney injury, cardiovascular instability and respiratory insufficient are the most often encountered organ dysfunctions in ACS. A complete review of IAH and ACS and how it affects organ function (11) falls beyond the scope of this chapter.

3. Pathophysiology

IAH and ACS are typically an early phenomenon in SAP and in most reports IAH develops in the first 3-5 days after hospital admission (12). Several mechanisms lead to increased intra-abdominal pressure (IAP) in these patients (**Table 1**) with pancreatic and peripancreatic inflammation probably only of minor importance as the increase in intra-abdominal volume from the local edema is often minimal (14). The ileus that often accompanies this disease process as well as the development of ascites may further increase the intra-abdominal volume but a major contributor is undoubtedly the fluid resuscitation that is often initiated in patients with severe pancreatitis to compensate for central

hypovolemia due to third spacing. Early aggressive fluid resuscitation is still considered the standard of care in many guidelines; however, fluid overload (although variably defined) has been found to be a major risk factor in several studies and different patient categories (23). Also in studies that specifically looked into ACS in pancreatitis, fluid resuscitation was consistently reported as contributing to both increased intra-abdominal volume and reduced abdominal wall compliance (6).

In the context of acute pancreatitis, the effects of IAH may have an important impact, not only on organ function as a whole, but also on pancreatic perfusion in particular. Animal studies have found that pancreatic perfusion is decreased in IAH (15), which may further increase the risk of pancreatic necrosis. Also bacterial translocation – the presumed pathway for pancreatic infection in SAP – is frequent in IAH; there is a dose dependent relationship with the extent of bacterial infection. As a result, IAH may impact both pancreatic necrosis as well as subsequent infection.

4. Epidemiology

SAP is one of the conditions where IAH and ACS are consistently reported. Using the original Atlanta criteria, the incidence of IAH was between 60 and 80 percent, and ACS developed in roughly 25-50 percent of the patients, according to one study (14). Using the new criteria, the incidence in severe disease may be even higher as some patients with what is now considered moderate pancreatitis were in the original severe category. Acute pancreatitis itself has been identified as a risk factor for IAH in a recent systematic review,

but also several other factors are often present in patients with SAP (16).

IAH and especially ACS has been associated with a worse outcome in all reports on this problem (2, 5, 7, 12, 17). Rosas et al. have even proposed to use IAP as a marker of severity in SAP; in their analysis, using a cut-off of 14mmHg, the Receiver Operating Characteristics curve of IAP was higher compared to the Ranson and Imrie score which could make IAP measurement a simple tool (30); unfortunately no other studies have evaluated IAP for this purpose.

5. Diagnosis of IAH

Diagnosing IAH and ACS in SAP is simple. Clinical examination is notoriously unreliable in diagnosing IAH and ACS, but IAP measurement should be now in the armamentarium of all contemporary ICUs. The bladder is used as a window to the abdomen, and several methods for reproducible IAP measurement are now available. Several reviews describing the techniques for IAP measurement have been published (31). In brief: 25 ml of sterile saline is instilled in the urinary bladder and subsequently the hydrostatic pressure is measured in mmHg (>12mmHg IAH, >20mmHg ACS) IAH grade I 12-15mmHg, grade II 16-20mmHg, grade III (ACS) 21-25, grade IV >25mmHg [34]. Small studies have investigated CT features of ACS patients and found that signs as narrowing of the vena cava, an increased in anteroposterior diameter and bowel wall thickening were associated with ACS (3). These are late signs and IAP measurement should be implemented before these occur.

6. Prevention of IAH in SAP

Now that the contributors to IAH have been better described, several options for prevention can be devised. As in other critically ill patients, fluid resuscitation has been coined as one of the key iatrogenic contributors to IAH and ACS, and in many of these conditions the concept of vigorous

fluid resuscitation should be urgently reevaluated. Several studies have linked overly positive fluid balances to worse outcomes, including ACS (17). In this context, studies have found that patients who were resuscitated less aggressively had lower incidences of ACS and better clinical outcomes. Whether the type of resuscitation fluid impacts this phenomenon remains unclear and given the ban on starches in many countries, crystalloids remain the primary resuscitation fluid. However, Zhao et al. found that patients who were resuscitated with normal saline only had higher intra-abdominal pressure (IAP) and ACS more often than patients treated with a combination of colloids and crystalloids (35).

It is difficult to recommend an appropriate resuscitation endpoint in SAP; conventional parameters such as central venous pressure are not recommended, as they are not predictive of fluid responsiveness, especially in IAH. Urinary output also has drawbacks as it is a typical early indicator of IAH and further fluid loading as a response to oliguria may aggravate rather than solve the problem. Dynamic indices such as stroke volume variation may be better tools (33) but can also be affected by IAH so judicious use of any parameter is advisable, and at all points, the requirement for fluids should be balanced against its side effects.

7. Treatment

WSACS – The Abdominal Compartment Society has recently updated the guidelines for managing IAH and ACS, which suggests a stepwise approach to decreasing IAP in patients (**Figure 1**) (19). In the context of SAP, a number of interventions are specifically relevant and these are discussed below. It is very important to realize that there are different non-surgical strategies available and although surgical intervention remains one of the definite treatment modalities, this should only be reserved for therapy resistant ACS.

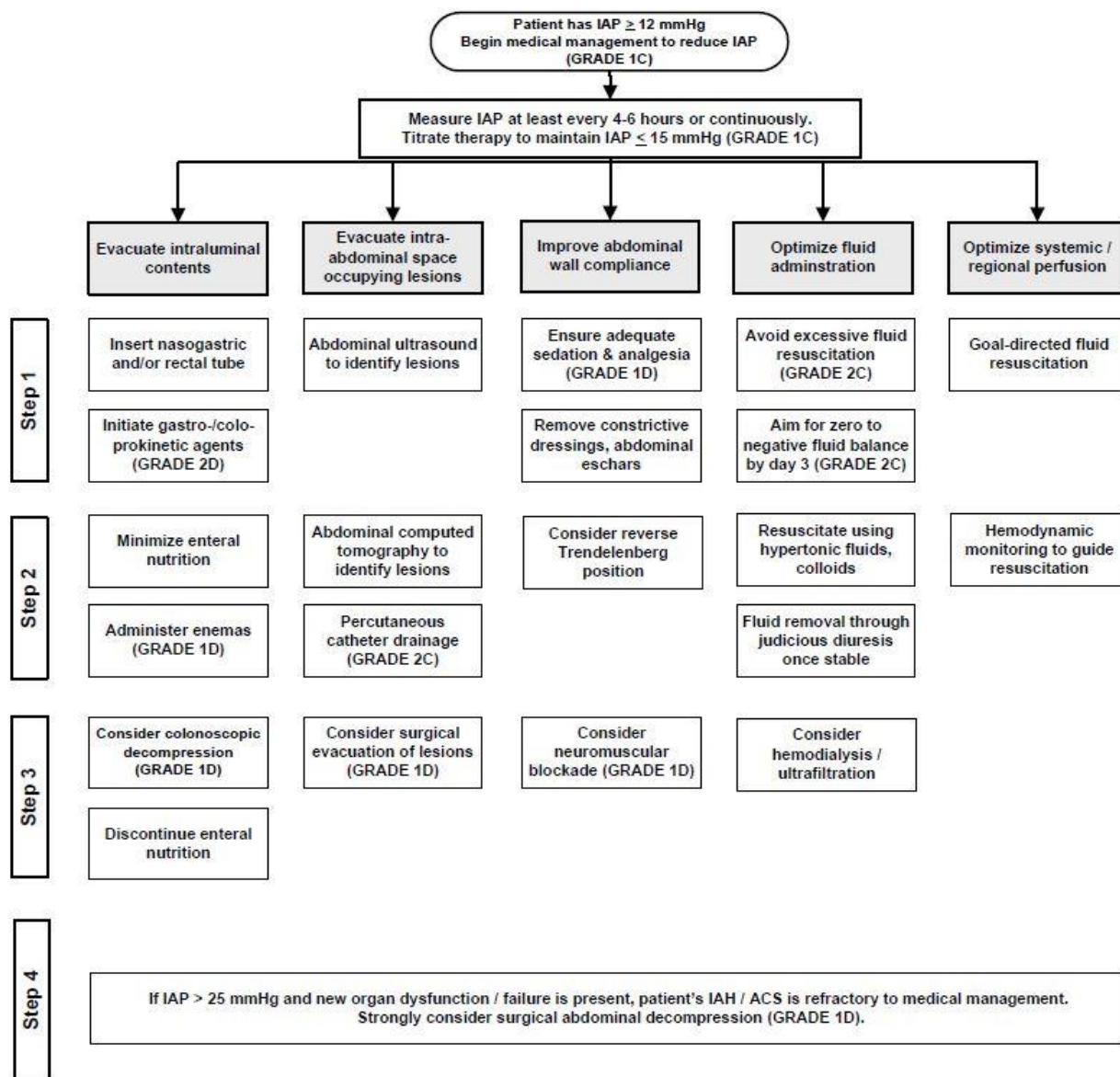


Figure 1. Medical management of IAH and ACS (19).

In most patients medical therapy is the first step, and when applied consistently, this will dramatically reduce the need for decompressive laparotomy. In any case, early and repeated IAP measurement is the first step towards recognition of the problem and therapy as well.

Nasogastric Decompression

As ileus and gastroparesis are often present, reducing the intraluminal volume of the gastrointestinal tract is a logical first step. In case of gastric dilatation, nasogastric decompression can easily be done, and may have an important

impact on IAP. The role of prokinetic drugs remains unclear.

Percutaneous Drainage

More frequently however, percutaneous drainage of ascites is a more useful, minimally invasive treatment option, that can be done at the bedside under ultrasound guidance. In the largest study to date in acute pancreatitis, Sun et al. describe a decrease in IAP from 29 to 14 mmHg after draining a median of 1800mL of ascites (32).

Also percutaneous drainage of retroperitoneal fluid collections or pseudocysts may reduce IAP and improve organ function (26).

Neuromuscular Blockers (NMB)

As in other conditions associated with IAH, improving abdominal wall compliance through NMB may be used (9). Although often used as a bridge to abdominal decompression, this may be continued for a short time (2-3 days) when necessary.

Fluid Removal and Hemofiltration

Small studies have focused on extracorporeal techniques to remove fluid overload. In a retrospective analysis Pupelis et al. found that hemofiltration was effective in removing fluid overload and reducing IAH, and was associated with improved outcomes (28). Also Oda et al. claimed improved outcomes after early hemofiltration to prevent IAH (25), but its exact role remains to be defined. Diuretics may be ineffective as patients often suffer from acute kidney injury with oligo- or anuria.

Surgical Decompression and Open Abdomen Therapy

Surgical decompression – usually through a full midline laparotomy - may be required in deteriorating patients with ACS who do not respond to medical therapy. Decompressive laparotomy is very effective in reducing the IAP in patients with ACS irrespective of the underlying cause (13), and also in SAP this has been documented. The role of decompressive laparotomy remains controversial and many surgeons are reluctant to operate in patients with SAP early in the course of the disease as many studies have found that early surgery in SAP was harmful. It is crucial that the pancreas not be touched during a decompressive laparotomy.

Alternatives to median laparotomy have been described for patients with SAP. Leppaniemi et al. have introduced the subcutaneous linea alba

fasciotomy through small skin incisions on the anterior abdominal wall (22). Although effective to avoid median laparotomy in many patients (20), the resulting giant hernia is definitely a downside of the technique. Fascial closure rates after open laparotomy are increasing because of improved temporary abdominal closure techniques. In the context of SAP, some surgeons may prefer a transverse incision to facilitate access to the pancreas later (21).

Timing of surgical decompression is a particular interesting topic. In a series of patients treated with decompression in Finland, the authors reported a 100% mortality rate in patients who were decompressed later than 5 days after the start of symptoms (24). It should not be surprising that in cases of prolonged exposure to high IAP, organ dysfunction is irreversibly damaged. But the exact time frame within which decompressive laparotomy can be successful is difficult to determine. Ke et al. found in an animal study that early intervention (as early as 6 hours after onset of ACS) was more effective (18).

The resulting open abdomen should be managed appropriately. Whereas this once used to be the surgeons' nightmare, negative pressure therapy has become the standard of care for the open abdomen, with the lowest complications and the highest primary fascial closure rates (4). Also in patients with SAP this method has been used successfully (27, 29). Using a mesh-based technique has been found the most successful method in achieving early abdominal closure (1) and can also be applied in SAP (29).

8. Conclusions

IAH and ACS are frequent findings in patients with SAP, and as in other settings, relevant contributors to organ dysfunction. IAP monitoring allows early detection of IAH and is recommended in all patients with severe disease. As fluid overload is an important risk factor for IAH this should be avoided. When IAH develops, percutaneous drainage of fluid collections is an

effective strategy to reduce IAP, but other medical treatment options can be considered and should be used selectively. If medical therapy fails, decompressive laparotomy may be an appropriate option to reduce IAP and restore organ function.

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