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IAH/ACS: The Rationale for Surveillance

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Abstract Surveillance for intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) should be implemented in every intensive care unit (ICU), because it has been demonstrated that surveillance is effective. Several criteria that have led to the conclusion that IAH/ACS monitoring is of value: First, IAH is a frequent problem in critically ill patients that directly affects function of all organ systems to some degree, and that is associated with considerable mortality. Furthermore, simple tools for intra-abdominal pressure (IAP) monitoring are available, and it can be safely applied without the need for advanced tools. Finally, both ACS and IAH can be treated with either medical or surgical interventions. Treatment for IAH/ACS should be selected on the basis of the severity of symptoms and the cause of IAH. IAP monitoring should also be incorporated in the daily ICU management of the patient.

Introduction

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been described for a long time, but it was not until recently that the deleterious effects of elevated intra-abdominal pressure (IAP) in ICU patients were described. In 2004, the World Society of the Abdominal Compartment Syndrome (WSACS) was founded by a group of international physicians and surgeons who recognized the need for a cohesive approach to promoting research, fostering education, and improving the survival of patients with IAH and/or ACS.

Nevertheless, these problems remain underappreciated by many clinicians [1, 2], and routine IAP monitoring has not been established in all ICUs. Surveys have shown that a considerable number of clinicians still think that IAH is not an issue in their patients or that IAH cannot be treated.

There is an overwhelming body of evidence that IAH and ACS does occur in critically ill patients, that it has an undisputed effect on organ function, and that it may have fatal consequences if left untreated. The WSACS advocates monitoring IAP in patients with risk factors for IAH/ACS [3].

The question, of course, remains whether surveillance—i.e., systematic screening for IAH in large groups of patients—is meaningful. There are a number of conditions that need to be fulfilled for surveillance to be advantageous. First, a simple tool must be available that allows monitoring. Second, the problem should be frequent, with considerable consequences for the patients in terms of morbidity and mortality, and for the daily management of the patients. Finally, one or more treatment modalities ought to be available, and a window of opportunity in which intervention is possible must be established.

In this review, we show that for IAH and ACS, every condition that is required to make surveillance meaningful has been fulfilled. Routine IAP monitoring is necessary in patients at risk for IAH and ACS for the following reasons, all of which are detailed in the sections that follow: (1) IAH is a frequent problem in critically ill patients; (2) IAH is an

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important problem, as it causes morbidity and mortality; (3) IAH can be detected at an early stage; (4) IAH and ACS can be treated; (5) daily IAP measurement is necessary for optimal patient management in IAH/ACS.

IAH is a frequent problem in critically ill patients

Abdominal compartment syndrome can be diagnosed when there is increased IAP with evidence of end-organ dysfunction. While multiple causes of acute cardiopulmonary, renal, hepatosplanchnic, or neurologic deterioration can be documented in the intensive care unit (ICU), it is important to recognize IAP as an independent risk factor. Timely recognition of underlying risk factors that lead to IAH is extremely important. Indications for IAP monitoring can be based on the presence/absence of these risk factors. Many conditions are reported in association with IAH/ACS, and they can be classified into four categories: (1) conditions that decrease abdominal wall compliance; (2) conditions that increase intraluminal contents; (3) conditions related to abdominal collections of fluid, air, or blood; and (4) conditions related to capillary leak and fluid resuscitation (Table 1).

Abdominal compartment syndrome can develop both in nonsurgical patients and surgical patients. Massive volume resuscitation after a "first hit" for any reason (e.g., burns, pancreatitis, trauma, hemorrhagic shock) can lead to increased IAP, particularly postoperatively or in a septic patient. The "second hit" probably results from the effects of capillary leak, shock with ischemia-reperfusion injury, and the release of cytokines combined with massive increases in total extracellular volume [4].

Mixed ICU patients

In a recent multicenter study, 265 consecutive patients (mean APACHE II score 17.4) who stayed more than 24 h in one of the 14 participating ICUs were followed until death, hospital discharge, or for a maximum of 28 days [5]. Medical patients accounted for 46.8%. On admission, 32.1% had IAH and 4.2% had ACS. Importantly, the occurrence of IAH during ICU stay was an independent predictor of mortality. Independent predictors of IAH were liver dysfunction, abdominal surgery, fluid resuscitation, and ileus. Other studies confirmed these results [6, 7].

Burn patients

The occurrence of ACS is significantly correlated with burn size (% total body surface area [TBSA]) and the amount and type of crystalloid fluids infused during the burn resuscitation; it does not require abdominal injury,

Table 1 Risk factors for the development of IAH and ACS [37]

A. Related to diminished abdominal wall compliance
Mechanical ventilation, especially fighting with the ventilator and the use of accessory muscles
Use of positive end-expiratory pressure (PEEP) or the presence of auto-PEEP
Basal pleuropneumonia
High body mass index
Pneumoperitoneum
Abdominal (vascular) surgery, especially with tight abdominal closures
Pneumatic anti-shock garments
Prone and other body positioning
Abdominal wall bleeding or rectus sheath hematomas
Correction of large hernias, gastroschisis, or omphalocoele
Burns with abdominal eschars
B. Related to increased intra-abdominal contents
Gastroparesis
Gastric distention
Ileus
Volvulus
Colonic pseudo-obstruction
Abdominal tumor
Retroperitoneal/abdominal wall hematoma
Enteral feeding
Intra-abdominal or retroperitoneal tumor
Damage control laparotomy
C. Related to abdominal collections of fluid, air, or blood
Liver dysfunction with ascites
Abdominal infection (pancreatitis, peritonitis, abscess,…)
Hemoperitoneum
Pneumoperitoneum
Laparoscopy with excessive inflation pressures
Major trauma
Peritoneal dialysis
D. Related to capillary leak and fluid resuscitation
Acidosis (pH below 7.2)[a]
Hypothermia (core temperature below 33°C)[a]
Coagulopathy (platelet count below 50,000/mm^3 OR an activated partial thromboplastin time (APTT) more than 2 times normal OR a prothrombin time (PTT) below 50% OR an international standardised ratio [INR] more than 1.5)[a]
Polytransfusion/trauma (>10 units of packed red cells/24 h)
Sepsis (as defined by the American–European Consensus Conference)
Severe sepsis or bacteremia
Septic shock
Massive fluid resuscitation (>5 l of colloid or >10 l of crystalloid/24 h with capillary leak and positive fluid balance)
Major burns

[a] The combination of acidosis, hypothermia, and coagulopathy has been put forward in the literature as the "deadly triad."

operation, or even the presence of abdominal wall burn eschar [8–11]. The cumulative ACS incidence ranges from 20% (in patients with 46% TBSA) to 70% (57% TBSA). Interestingly, in one study, two types of ACS were seen: ACS developing within the first 24 h after admission (related to the initial fluid resuscitation) and ACS developing weeks later (related to sepsis) [12].

Postoperative patients

The cumulative incidence of ACS in postoperative patients ranges from 10% in severe acute pancreatitis [13], over 31% in liver transplant patients [14], and 40% after major abdominal surgery [15]. Only one study really looked at the incidence of IAH [14], whereas other studies investigated the association of postoperative IAH with renal insufficiency [15, 16] or gastric intramucosal pH [17]. Emergency surgery carries a higher risk for postoperative IAH [16].

Trauma patients

The incidence of ACS ranges from 2% in all trauma patients to 14% in major torso trauma to 40% in patients undergoing damage-control laparotomy [18]. Hemostasis is achieved by placing packs around the liver, allowing for further correction of coagulopathy before the end of the operation [19, 20]. Differences in the proportion of patients who receive abdominal packs may account for the difference in IAH incidence between groups of comparable patients: in the study by Ertel et al. only 20% required abdominal packing [21], compared to 67% in the study by Meldrum et al. study [18]. Primary fascial closure is associated with a higher risk of IAH than temporary closure [22–25]. However, the application of a temporary abdominal closure does not preclude the occurrence of IAH in 25% of patients with penetrating abdominal trauma [22]. In a large review of the charts of trauma patients without abdominal injury, ACS occurred very early in the time course of fluid resuscitation [26]. The average time from admission to decompressive laparotomy was 18 h in all patients, 3.1 h in survivors, and 25 h in nonsurvivors. Supranormal resuscitation, compared with normal resuscitation (to an oxygen delivery index of respectively >600 ml/min/m^2 and > 500 ml/min/m^2), was associated with an increased incidence of IAH and ACS [27].

IAH is an important problem, as it causes morbidity and mortality

Increased IAP has been shown to affect the function of organ systems both within and outside of the abdominal cavity. A central concept in understanding IAH-induced organ dysfunction is the observation that part of the increased IAP is transmitted to the thorax as a consequence of cephalad displacement of the diaphragm. This leads to increased intrathoracic pressure (ITP) and ultimately to increased jugular venous pressure, decreased venous return from the brain, and possibly increased intracranial pressure in patients at risk for intracranial hypertension [28]. This mechanism has been described in animal studies, and some small human studies have confirmed that decompressive laparotomy can be successful in treating refractory intracranial hypertension after other interventions have failed.

Intra-abdominal hypertension also has profound effects on the respiratory system, most importantly decreased functional residual capacity of the lungs and decreased compliance of the thoracic wall. These changes can result in a clinical picture of secondary (or extrapulmonary) acute respiratory distress syndrome (ARDS), which requires advanced ventilatory strategies [29].

The deleterious effects of IAH on the cardiovascular system are multiple and can be summarized as decreased venous return to the heart (due to compression of the inferior cava vein), decreased cardiac contractility (probably due to direct compression of the heart), and increased afterload due to compression of the arterial tree [30].

The most frequent form of IAH-induced organ dysfunction is kidney injury, which has been described extensively. The exact mechanism is not completely understood, but compression of the renal vein and increased renal venous pressure seem to be the primary mechanisms involved [31].

Given these effects of IAH on all organ systems, it comes as no surprise that it has been associated with mortality in critically ill patients. Although historically this has been studied mainly in trauma patients with ACS [26] and in patients after abdominal surgery [16, 32], recent studies have found that IAH itself—meaning a reduction in IAP—has detrimental effects on outcome. Malbrain et al. reported in 2005 that IAH at admission was associated with organ dysfunction in a mixed medical-surgical ICU population, and that the occurrence of IAH during ICU admission was an independent predictor of mortality [5]. Other investigators have found similar effects of IAH in mixed ICU patients [6], burn patients [33], patients with severe sepsis [34], and patients with severe acute pancreatitis [35]. Cheatham and Safcsak reported a reduced need for decompressive laparotomy and improved patient survival after implementation of a strategy aimed at early treatment of IAH [36].

IAH can be detected at an early stage

An important issue in effective surveillance is to have a simple and affordable tool that can be used in critically ill

patients. Although direct intraperitoneal pressure measurement is the gold standard for IAP measurement [37], extensive research has shown that it can reliably be measured via the transvesical route.

In 1984 Kron et al. [38] described a technique that is in wide use today, with modifications introduced by Cheatham and Safcsak [39]. In this set-up, a three-way stopcock is inserted between the bladder catheter and the collector, which is connected to a pressure transducer and allows measurement of the intravesical pressure after instillation of saline in the bladder. Originally, large volumes of saline were used for instillation, but it has been demonstrated that this may lead to overestimation of the IAP [40]. The WSACS now recommends limiting the amount of fluid to 25 ml or less in children [37]. Several companies have developed a preassembled kit that further simplifies IAP measurement (AbViser AutoValve series, Wolfe Tory Medical, Salt Lake City, UT; Intra-abdominal Pressure Monitoring Device, Bard, Covington, GA). An often suggested drawback of this technique is the fact that the sterile circuit may be disrupted, with an inherent risk of urinary tract infection. Cheatham et al. studied the incidence of UTI in patients with IAP monitoring, and concluded that this fear was unfounded [41].

Alternatively, IAP can be measured using the patient's own urine as a pressure medium. Initially described by Harrahill [42], a commercial device has been developed that employs the same principle (FoleyManometer, Holtech, Charlottenlund, Denmark; UnoMeter, Unomedical, Roskilde, Denmark). This system does not need a pressure transducer or monitor and can therefore be used in non-ICU environments as well.

More advanced systems also allow continuous IAP measurement, either via the bladder or the stomach (Spiegelberg, Hamburg, Germany; CiMON device, Pulsion Medical Systems, Munich, Germany) [43]. These offer additional advantages, such as online monitoring of the abdominal perfusion pressure (APP, which is the mean arterial pressure minus the IAP). Although these may be better suited for monitoring in selected patients, they are not be used for screening purposes.

IAH and ACS can be treated

Although often physicians may argue that IAP measurement is useless as there are no treatment options, we will show that this statement no longer holds true. Treatment options for IAH and ACS can be divided in two categories: medical and surgical. These can be used in a stepwise approach for patients with IAH; however, in patients with ACS and rapidly deteriorating organ dysfunction, urgent surgical intervention should be considered. Better than treatment, of course, is prevention, which specifically applies to abdominal surgery patients. In some patients, such as those who have suffered abdominal trauma or a ruptured aortic aneurysm, abdominal closure at the end of the procedure may be difficult. When this is the case, open abdomen treatment with a temporary abdominal closure (TAC) system is recommended. Abdominal closure can be attempted as soon as the physiology of the patient allows.

Medical treatment options are aimed at either reducing the intra-abdominal volume or increasing compliance of the abdominal wall [44]. Nasogastric decompression and colonic decompression can be attempted with a nasogastric or rectal tube, with decompressive colonoscopy, or pharmacologically, with prostigmin or prokinetics. Intraperitoneal fluid collection can also contribute to increased intra-abdominal volume; percutaneous drainage is an elegant method that significantly reduces IAP, as has been demonstrated in patients with acute on chronic liver failure. Neuromuscular blockers target the compliance of the abdominal wall and have been demonstrated to lower IAP [45]. In patients who have received massive fluid resuscitation, extensive edema both of the abdominal wall and intra-abdominal organs may cause ACS. In those patients fluid removal using renal replacement techniques may help to reduce IAP [46].

Surgical treatment for ACS most often involves a full midline laparotomy, which results in a significant drop in IAP in most patients [47]. At the time of surgery, an intra-abdominal cause of IAH can be treated when present. Except in patients who require extensive resection of intra-abdominal tissue or in whom large quantities of fluid are drained, the abdomen will be left open and a TAC system will be applied; different methods are available, and are discussed elsewhere in this issue by Ivatury et al. A transverse incision may be used in selected patients, depending on the underlying disease. Recently, endoscopic anterior fasciotomy has been described by Leppaniemi et al. as an interesting alternative to a full laparotomy [48].

IAP measurement is necessary for daily patient management

Most pathological alterations associated with IAH are nonspecific and can occur secondary to a variety of other conditions. However, in many cases, these alterations do require a different therapeutic approach in the presence of IAH. Therefore a thorough understanding of the underlying mechanisms of IAH-induced organ dysfunction is essential in providing optimal treatment [49].

The main issue in hemodynamic management of the patient with IAH is the impaired reliability of hemodynamic monitoring. As mentioned above, an increase in ITP

results in a rise in central venous pressure (CVP), which no longer reflects cardiac preload. More advanced, preferably volumetric, monitoring devices also present pitfalls, but are generally more reliable.

The cornerstones of ventilatory management of the patient with IAH are measurement of higher positive end-expiratory pressure (PEEP) levels and lung protective ventilation, as described by the ARDS Network. It must be remembered that higher inspiratory pressures can be tolerated to compensate for the decreased compliance of the thoracic wall.

In patients at risk for intracranial hypertension, it is important to be aware of the association between IAP and intracranial pressure (ICP) and to avoid increases in IAP, e.g., during laparoscopy. In patients with IAH and refractory intracranial hypertension, strategies to lower IAP may have a beneficial effect.

All these measures are aimed at improving organ support in the presence of IAH and do not improve the underlying cause of the organ dysfunction. When higher levels of IAP are recorded in the presence of progressive organ dysfunction, the only definitive solution is to lower IAP.

Conclusions

Intra-abdominal hypertension is a frequent problem in critically ill patients, and it causes significant morbidity with potentially fatal outcome when left untreated. Given the fact that there are multiple treatment options that can be used in a stepwise approach, and given that IAP measurement can change certain aspects of daily ICU care, we conclude that the simple procedure of IAP monitoring is necessary in all patients at risk for IAH.

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