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Abdominal compartment syndrome among surgical patients

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Abstract

Abdominal compartment syndrome (ACS) develops when organ failure arises secondary to an increase in intraabdominal pressure. The abdominal pressure is determined by multiple factors such as blood pressure, abdominal compliance, and other factors that exert a constant pressure within the abdominal cavity. Several conditions in the critically ill may increase abdominal pressure compromising organ perfusion that may lead to renal and respiratory dysfunction. Among surgical and trauma patients, aggressive fluid resuscitation is the most commonly reported risk factor to develop ACS. Other conditions that have also been identified as risk factors are ascites, hemoperitoneum, bowel distention, and large tumors. All patients with abdominal trauma possess a higher risk of developing intra-abdominal hypertension (IAH). Certain surgical interventions are reported to have a higher risk to develop IAH such as damage control surgery, abdominal aortic aneurysm repair, and liver transplantation among others. Close monitoring of organ function and intra-abdominal pressure (IAP) allows clinicians to diagnose ACS rapidly and intervene with target-specific management to reduce IAP. Surgical decompression followed by temporary abdominal closure should be considered in all patients with signs of organ dysfunction. There is still a great need for more studies to determine the adequate timing for interventions to improve patient outcomes.

Key Words: Intra-abdominal hypertension; Abdominal compartment syndrome; Intra-abdominal pressure; Open abdomen treatment; Multiple organ failure; Surgical decompression

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Core Tip: Abdominal compartment syndrome (ACS) is a complication of several surgical and medical conditions that increase the intra-abdominal pressure (IAP) and cause organ hypoperfusion. Diagnosis is made by adequately measuring IAP and identifying the presence of intra-abdominal hypertension (IAH) with secondary organ dysfunction. IAH may progress to ACS when blood flow to multiple organs is compromised. Medical management aiming to decrease IAP should be started promptly to improve outcomes. Signs of organ hypoperfusion or evident organ failure warrants a rapid surgical evaluation for abdominal decompression. Close monitoring and rapid interventions are the key to improve the outcome in this complex condition.

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INTRODUCTION

Abdominal compartment syndrome (ACS) and intra-abdominal hypertension (IAH) may be underdiagnosed and are associated with increased morbidity and mortality in medical patients^[1]. Intraabdominal hypertension (IAH), defined as a persistent intra-abdominal pressure (IAP) > 12 mmHg, can lead to multiple organ failure and induce ACS when IAP rises above 20 mmHg^[2]. Knowledge of the pathophysiologic changes with IAH and in-time diagnosis, by identifying the underlying condition for proper management, is the landmark in treatment success.

BRIEF HISTORY

In 1811, Richard Volkmann described compartment syndrome in limbs. Years later, the relation of increased IAP and respiratory function was described by Etienne-Jules^[3].

The IAP was initially estimated by the intrauterine or rectal pressure using a manometer. In 1875, Odebrecht was the first to assess the IAP by measuring bladder pressure^[3].

Before 1911, IAP was not fully understood, and depending on the method used to measure it could be reported as negative or positive^[4]. Studies in animals were first done to determine the factors that could play a role in IAP. In 1984, Kron *et al*^[5] reported a case series of patients after abdominal surgery and concluded the pressure was more likely to be positive. They also presented their technique for IAP measurement in humans, the values associated with renal insufficiency, and the values associated with re-exploration in the postoperative period.

In the last three decades, the interest in ACS has raised with a significant increase in the literature published^[3].

In 2004, the World Society of the Abdominal Compartment Syndrome (WSACS) was founded after a multidisciplinary critical care team of healthcare workers met at a conference and noted a lack of uniformity among definitions of IAH, ACS, and IAP monitoring^[6]. Their final consensus was reported in 2006 and updated in 2013^[2,6].

DEFINITIONS

Intra-abdominal pressure

Normal IAP in adults is < 5 mmHg. In critically ill patients, IAP is considered normal between 5 mmHg and 7 mmHg^[2].

Many trials have been conducted to measure and determine the normal IAP in adults and the differences among average values in obese patients^[7,8]. Higher pressures in obese patients are thought to be related to a direct effect of adipose tissue. Values of IAP from 7 mmHg to 14 mmHg could be expected in obese patients^[4].

Intra-abdominal hypertension

IAH was defined by the WSACS as a persistent IAP > 12 mmHg. This should be measured at end-expiration in the supine position. IAH is graded according to IAP (Table 1).

IAP may also be divided in hyperacute, acute, or chronic^[9]. Hyperacute IAH is a brief elevation of IAP during sneezing, laughing, coughing, or Valsalva maneuver. Acute IAH is the sustained elevation of IAP which can progress to ACS. Elevation of IAP over months or years secondary to pregnancy or obesity is considered chronic IAH^[9].

Abdominal perfusion pressure

APP is the difference between mean arterial pressure (MAP) and the IAP. $APP = MAP - IAP$. It can be used as an indicator of gastrointestinal organ perfusion and studied as a survival predictor better than other resuscitation markers such as arterial pH, lactate, base deficit, and urinary output in patients with ACS^[10]. The normal APP is 60 mmHg^[11].

Abdominal compartment syndrome

In general, ACS is defined as an IAP higher than 20 mmHg associated with organ failure, however, in the clinical setting ACS is essentially IAH with signs of new organ dysfunction. The cut-off value of IAP for ACS has been established for research purposes, in the clinical setting no reliable IAP cut-off can diagnose ACS, since IAP and organ failure relation may vary from patient to patient^[2].

ACS is further divided into primary, secondary, or recurrent.

Primary ACS is associated with localized abdominopelvic pathology causing an increase in IAP and usually requires an early intervention with decompressive surgery or percutaneous drainage by interventional radiology^[2].

Secondary ACS is the result of an increase in IAP from an etiology outside the abdominal cavity such as aggressive fluid resuscitation, mechanical ventilation, major burns, shock, or sepsis^[2].

Recurrent ACS is the development of IAH or ACS after a previously treated primary or secondary ACS. This can present even in an open abdomen or after closure and resolution of the initial ACS episode^[2,12].

The elevation in pressure from two different anatomical compartments (head, chest, abdomen, and extremities) is referred to as a poly-compartment syndrome^[2,13].

EPIDEMIOLOGY/MORTALITY

The prevalence of IAH and ACS differs vastly among the literature. For IAH, the intensive care unit (ICU) has been reported up to 54.4% in medical patients and 65% in surgical patients^[14]. Two recent prospective studies reported an IAH admission prevalence of 30%-34%, and 15%-16.5% of patients developed IAH during the ICU stay^[15,16]. They found no significant difference in admission prevalence among medical or surgical patients. ACS was reported in 3% and was most common in medical patients^[17]. The highest prevalence of ACS development has been noted in patients with acute pancreatitis, orthotopic liver transplantation, and abdominal aorta surgery^[18]. A prospective study with 503 patients admitted to a mixed ICU found an increased risk for IAH/ACS development in patients with emergency abdominal surgery (56.7%) and non-surgical patients (highest prevalence in acute pancreatitis) compared with elective abdominal surgery (18%)^[18].

Mortality is associated with the grade of IAH (Grade I: 10%-25%, Grade-II: 15%-45%, Grade III-IV: 50%-60%), and studies have found IAH to be an independent predictor of mortality^[17,19]. ACS carries a mortality risk reported by 75%-90%^[19].

ETIOLOGY AND RISK FACTORS

IAH can be a result of a variety of factors. The abdomen is a closed cavity in which the IAP is determined by internal volume (gas, fluid, fat, organs) and wall compliance^[20]. An increase in intra-abdominal volume or a decrease in abdominal wall compliance can increase IAP. Intra-abdominal volume can increase due to ascites, hemoperitoneum, bowel distention, large tumors, and third space of fluids resulting in tissue edema^[12].

Table 1 Intra-abdominal hypertension grading^[2]

| Grade | Intra-abdominal pressure |
|-----------|--------------------------|
| Grade I | 12-15 mmHg |
| Grade II | 16-20 mmHg |
| Grade III | 21-25 mmHg |
| Grade IV | > 25 mmHg |

Mechanical ventilation, severe burns, external constraints, edema, abdominal wall surgeries, among others, can impair abdominal wall compliance^[12].

Critically ill patients are at higher risk of IAH and ACS due to conditions predisposing to aggressive fluid resuscitation and positive fluid balance, causing visceral edema^[21]. This risk may be increased when using a large volume of crystalloid fluids and could decrease with colloid or hypertonic resuscitation^[22]. Additional factors associated with the risk of developing IAH during the ICU stay are: Resuscitation with > 3 L of crystalloids pre-ICU admission, body mass index > 27 kg/m², abdominal distension, absence of bowel sounds, and positive end-expiratory pressure greater than 7 cmH₂O^[15].

Any abdominal surgery or abdominal trauma has the possibility to increase IAP, however, the procedures reported with a higher risk for IAH development are orthotopic liver transplantation, damage control surgery, abdominal aortic aneurysm repair, and massive incisional hernia repair^[2,18,23].

Among surgical and trauma patients, the most commonly reported risk factor for ACS is excessive fluid resuscitation^[22].

McNelis *et al*^[24] conducted a case-control study with surgical patients, they found 24-h fluid balance and peak airway pressure as predictors for ACS development and created a predictive equation with these variables to identify postoperative patients who will develop ACS.

PATHOPHYSIOLOGIC CHANGES

IAP elevation results not only in gastrointestinal function impairment but also in multiple organ damage leading to ACS. Primary damage results from impairment in tissue perfusion caused by increased pressure in a closed space (Table 2).

CLINICAL PRESENTATION

Patients with ACS will usually be critically ill and unable to provide history and symptoms. On physical exam, patients present with a distended abdomen. However, palpation and abdominal circumference are not reliable for the diagnosis of ACS^[25].

A prospective study in postoperative ICU patients showed physicians have less than a 50% chance to identify IAH by clinical examination^[25]. The clinical abdominal exam as IAP assessment has an estimated sensitivity of 56%-60% and specificity of 80%-87%^[25,26].

Signs of ACS will present as the end-organ effect from the physiologic changes (Table 2). The most notorious signs are usually abdominal distention, oliguria, high ventilatory pressures, diminished cardiac output, and metabolic acidosis^[26].

DIAGNOSIS

For ACS diagnosis, IAP needs to be measured. Multiple methods for IAP estimation have been described and can be divided into direct and indirect. Direct measurement is considered more accurate but requires invasive techniques to access the abdominal cavity through a catheter^[20]. Indirect measurement can use intravesical, intragastric, rectal, and intrauterine pressure to estimate IAP^[20]. The standard technique for IAP is the estimation through the intravesical pressure (IVP), which has been proven to be effective, minimally invasive, simple, and low cost^[6]. The technique used was initially described by Kron and later modified by Cheatham and Safcsak^[27].

Table 2 Pathophysiologic changes and end-organ effect of intra-abdominal hypertension

| System | IAH mechanism of adverse effects | End-organ effect |
|---|---|--|
| Central nervous system ^[42] | Obstruction of venous outflow | Increase in intracranial pressure |
| | Increase in jugular venous pressure | Decrease in cerebral perfusion pressure |
| | Increase in inflammatory markers with disruption of the blood-brain barrier | |
| | Decrease lumbar venous plexus blood flow | |
| | -Increase cerebral blood flow | |
| Cardiovascular system ^[43] | Elevation of diaphragm and elevation in intrathoracic pressure with cardiac compression | Increase in right ventricular afterload |
| | Compression of inferior vena cava | Decrease in cardiac output |
| | | Decrease in ventricular compliance/contractility |
| Pulmonary system ^[36,44] | Pulmonary compression | Increase in peak airway pressure |
| | Alveolar atelectasis | Increase in plateau pressure |
| | Decreased pulmonary capillary blood flow | The decrease in pulmonary compliance |
| | Lymphatic drainage impairment | A decrease in tidal volume |
| | | A decrease in functional residual capacity |
| | | Ventilation-perfusion mismatch |
| | | Hypercarbia |
| | | Lung edema |
| Gastrointestinal system ^[12] | Direct impairment of arterial and venous blood flow | Ileus |
| | Intestinal perfusion impairment | Bowel edema |
| | Obstruction of lymphatic flow | Bowel ischemia |
| | | Bacteria translocation |
| | | Lactatemia |
| | | Decrease gastric intramucosal pH |
| | | A decrease in filtration gradient |
| Renal system ^[45,46] | Renal artery and vein compression | Oliguria |
| | Parenchymal compression | Acute renal failure |
| | Glomerular and tubular function impairment | |
| | Activation of the renin-angiotensin system | |
| Vascular system ^[47] | Increased systemic vascular resistance | Peripheral edema |
| | Inferior vena cava compression | Venous stasis |
| | | Increase the risk of venous thrombosis |

IAH: Intra-abdominal hypertension.

During the IVP measurement, the patient should be at end-expiration and in the supine position to avoid muscle contractions, which could decrease pressure accuracy. Head-of-bed elevation to 30-40° may increase IAP by 4-9 mmHg^[4]. The maximal installation volume used is 25 mL of sterile saline since the installation of more volume may artificially increase IAP measurement^[6,28].

IVP measurement is contraindicated in patients with cystectomy or traumatic bladder injury, and other techniques may be used^[20].

Other innovative techniques for IAP measurement include wireless motility capsules, near-infrared spectroscopy, and point-of-care ultrasound^[12]. Although the most widely accepted and used measure for diagnosing abdominal hypertension is IAP, a retrospective study showed APP could be superior for patient survival prediction^[10].

Iyer *et al*^[29] developed a screening tool for ICU patients at risk for IAH and ACS to be used within the first 24 h of admission and identify patients who may benefit from IAP monitoring. Obesity, hemoperitoneum/pneumoperitoneum or fluid collection, abdominal distention, Sequential Organ Failure Assessment score > 4, lactate > 1.4 mmol/L, and more than 2.3 L of IV fluids were independent predictors of IAH and used for the screening tool^[29].

Imaging studies are not considered part of the standard diagnosis algorithm of ACS. However, some computed tomography (CT) signs have been considered for early identification of ACS. Among the significant CT findings related to IAH are the peritoneal-to-abdominal height ratio > 0.52 and the round belly sign, which is a ratio > 0.8 of maximal anteroposterior to transverse abdominal diameter^[30].

Other suggested CT findings related to IAH are bowel wall thickening (> 3 mm), the diaphragm's elevation, narrowing < 3 mm of inferior vena cava, and large intra-abdominal fluids^[30].

MANAGEMENT

ACS treatment is focused on decrease IAP and improve end-organ perfusion^[31].

The management is always patiently individualized. Along with the value of IAP, the etiology of ACS, and the impact on organ dysfunction need to be considered^[32].

IAH management/secondary ACS

Patients with IAP > 12 mmHg should be started in supportive medical care to reduce IAP and avoid ACS with progressive organ failure. Measurement of IAP is recommended every 4 h^[2]. Efforts to decrease IAP should be promptly started, the duration of IAH is an independent prognostic factor of 60-d mortality in patients with IAH^[33].

Fluid balance and hemodynamic support

The goal for fluid balance should be to avoid excessive use of intravenous fluids and maintain a neutral or negative fluid balance^[2]. When possible, the use of colloids and hypertonic solutions should be considered. The use of diuretics and hemodialysis may be used. However, there is no clear evidence of is a benefit, and there is no recommendation for its use in the WSACS guidelines^[2].

Continuous hemodynamic monitoring will guide goal-directed fluid resuscitation and the use of damaged control resuscitation as needed^[2]. However, clinicians should keep in mind that parameters like central venous pressure, pulmonary capillary wedge pressure, ultrasound compression of inferior vena cava, mean arterial pressure, among others, may not be reliable during ACS^[19].

Wall compliance

A reduction in abdominal wall muscle tone may improve abdominal wall compliance and help reduce IAP. Agitation, pain, and use of accessory muscles for ventilation can be avoided using sedation, optimal analgesia, and brief trials of neuromuscular blockade^[2]. Head of bed (HOB) elevation should be avoided, and maintain the patient in the supine position. HOB elevation at 15 and 30 degrees increases IAP to 1.5-3.7 mmHg, respectively^[34].

Reducing intraluminal volume

Intraluminal contents such as gas or fluid can cause IAH (gastric/bowel obstruction, ileus, or colonic pseudo-obstruction). As a first step, the WSACS recommends inserting a nasogastric tube and rectal tube for enteral decompression^[2]. In the case of gastroparesis, metoclopramide or erythromycin could be considered and limit enteral nutrition^[19]. The use of neostigmine is also recommended as a colo-prokinetic agent followed by enemas^[2].

Reducing extraluminal volume

An increase in extraluminal volume can originate from the free fluid (hemoperitoneum, ascites), gas (gastrointestinal perforation), abscesses, or large tumors^[19]. Ultrasound or CT scan can help to recognize these conditions and plan proper management. Percutaneous catheter drainage (PCD) is used either as a definitive treatment for some of these conditions or as a supportive or bridge therapy to surgical decompression^[2,35].

Ventilatory support

Along with proper sedation and neuromuscular blockade, low tidal volume and a limited pressure mode may optimize ventilation and lung protection^[32]. Higher positive end-expiratory pressure can be used in IAH to prevent end-expiratory lung collapse and reduce ventilation-perfusion mismatch^[36].

ACS Treatment

Management of established ACS starts with the identification of the underlying condition. In the presence of primary ACS, the WSACS guidelines recommend performing PCD for fluid removal when possible and use of abdominal decompression with laparotomy as the standard of care in formal ACS^[2]. Surgical management with laparotomy decreases IAP immediately and improves organ function^[37,38]. The procedure is performed in the operating room or the ICU for selected cases^[38]. Delay in decompression may increase mortality^[38]. The decompressive laparotomy, depending on the underlying etiology of ACS, can result in an open abdomen (OA), which carries a risk for infections, hypercatabolic state, fluid loss, and enteric fistulas but reduces the risk for recurrent ACS^[19]. Surgical decompression needs to be followed by a clear closure plan^[39]. Decompressive laparotomy can result in ischemia-reperfusion syndrome and supportive measures need to be considered along with fluid balance and improvement in wall compliance to avoid recurrent ACS^[32].

Open abdomen in ICU

OA increases morbidity significantly and primary closure may not be achieved during the same decompressive procedure. Temporary abdominal wall closure with “Bogota bag” or negative pressure wound therapy (NPWT) helps to decrease the risk for infections, enteroatmospheric fistula, fluid loss and prevents fascial retraction, thus ease an early primary fascial closure^[19]. Re-explorations may be needed in 24-48 h with attempts for fascial closure or at least continuous fascial traction depending on several factors^[40]. Factors that may delay definitive closure are coagulation disorders, shock, sepsis, and persistence of IAH^[19].

The WSACS recommends the use of NPWT and an early same hospital stay abdominal fascial closure^[2]. The primary fascial closure should be attempted in the first 4-7 d following decompressive laparotomy. Failure to close OA within the first 8 d is a risk factor for fistula formation and frozen abdomen^[41]. An ICU multidisciplinary care is essential for physiologic optimization and nutritional support which are determinants for an early and successful OA closure^[40].

CONCLUSION

ACS is a complex and dynamic condition that warrants close monitoring and prompt interventions to improve patient outcomes. The adequate measurement of IAP is essential in establishing a diagnosis of IAH and ACS since imaging findings and signs/symptoms are not enough to make a diagnosis. Once IAH is determined close observation is mandatory. The presence of organ dysfunction establishes an equivocal diagnosis of ACS and patients should be rapidly evaluated for surgical decompression. Additional supportive medical management includes adequate fluid balance, pain/sedation control, hemodynamic and ventilatory support. There is no general consensus on a specific IAP threshold for surgical decompression, however, the general agreement among clinicians is that a prompt intervention may improve outcomes. The measure of abdominal perfusion pressure is an additional value that may be used for decision making. There is still a gap of knowledge regarding the timing of surgical decompression in ACS and patient outcomes, establishing the need for more studies.

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