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RESEARCH **REVIEW ARTICLE**

Abdominal Compartment Syndrome: Definitions, Epidemiology, and Management

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INTRODUCTION

Abdominal compartment syndrome (ACS) stands as a complicated and potentially life-threatening medical condition that requires our attention in the field of critical care medicine. This syndrome, characterized by increased intra-abdominal pressure, has undergone a transformative journey in its understanding and management.

The history of ACS is a story of progressive discovery and understanding. It began when Richard Volkmann described compartment syndrome in limbs in 1811 (1), highlighting how increased pressure within fascial spaces led to reduced muscle blood flow and contractures. In the 19th century, Etienne-Jules Marey and Paul Bert made early connections between elevated intra-abdominal pressure (IAP) and its effect on respiratory function. In 1951, M.G. Baggot emphasized the significance of IAP in abdominal closure (2). "ACS" as a term was first used in 1989 (3); since then, ACS has gained substantial attention, with the establishment of the World Society of Abdominal Compartment Syndrome (WSACS) in 2004, formalizing interest and research in this field (1). As medical knowledge evolved, it became evident that ACS extends beyond traumatic contexts. It can manifest in non-traumatic conditions underscoring its multifaceted nature. This expanding view of ACS necessitates a holistic approach to its diagnosis and management, with a focus on early identification of at-risk patients to prevent devastating consequences.

Despite advancements in critical care, a notable deficiency persists in the current literature surrounding ACS. Many aspects of ACS, including its precise pathophysiology, optimal diagnostic criteria, and management strategies, remain subjects of ongoing research and debate. This deficiency in understanding can result in delayed recognition and treatment, potentially compromising patient outcomes. We aim to provide a summary of the current literature on ACS, focusing on the management approach and identifying knowledge gaps to guide future research.

CLASSIFICATION

Intra-abdominal pressure (IAP) pertains to the pressure within the abdominal cavity, typically averaging between 0 and 5 mmHg. However, in severely ill individuals, it can rise to as much as 7 mmHg.

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Abdominal perfusion pressure (APP) is calculated by subtracting the intra-abdominal pressure (IAP) from the mean arterial pressure (MAP) and represents the effective force propelling blood circulation to the abdominal organs. A normal APP is generally considered to be approximately 60 mmHg or higher. APP has been suggested as a more precise gauge of visceral perfusion and a potential goal for resuscitation efforts (1).

Intra-abdominal hypertension (IAH) is identified by a prolonged elevation in intra-abdominal pressure (IAP) beyond the threshold of 12 mmHg. In contrast, abdominal compartment syndrome (ACS) is characterized by a persistent elevation in IAP exceeding 20 mmHg, either with or without a decrease in abdominal perfusion pressure (APP). ACS is closely associated with the emergence of fresh organ malfunction or failure, as specified by the World Society of Abdominal Compartment Syndrome (WSACS) (4).

IAH is further classified according to pressure, acuteness, and cause. Based on a study by Malbrain et al. (5) IAH is categorized into four grades: Grade I, with an IAP ranging from 12 to 15 mmHg; Grade II, with an IAP ranging from 16 to 20 mmHg; Grade III, with an IAP ranging from 21 to 25 mmHg; and Grade IV, with an IAP exceeding 25 mmHg. The timeframes for the development of IAH are classified as follows: hyper-acute IAH, which occurs within seconds; acute IAH, which develops over hours, particularly in surgical and trauma patients and can often progress to ACS; sub-acute IAH, which develops over several days and is the most common form; and chronic IAH, which develops over years due to factors like obesity, pregnancy, or cirrhosis. Chronic IAH places critically ill patients at significant risk of developing ACS and necessitates monitoring (5).

Ultimately, abdominal compartment syndrome (ACS) is divided into three categories: primary ACS, stemming from injuries to the abdomen or pelvis; secondary ACS, arising from conditions unrelated to the abdomen or pelvis; and recurrent ACS, which occurs after previous management of ACS, regardless of whether it was initially primary or secondary ACS (6).

EPIDEMIOLOGY & RISK FACTORS

The prevalence of ACS varies across different patient populations and clinical settings. While exact prevalence rates may differ due to variations in diagnostic criteria and patient populations, a meta-analysis has reported ACS prevalence ranging from 0.0% to 36.4% with a mortality rate ranging from 0.0% to 100% with a pooled value of 50% approximately after reviewing 80 publications (7).

In another prospective, observational, single-center cohort study of 503 patients admitted to the ICU, it was found that 33% developed intra-abdominal hypertension and 3.6% developed ACS: with pancreatitis patients having the highest prevalence of ACS (57%)(8). Similar results were present in a prospective observational study done in 15 ICUs worldwide which included 491 patients showing the prevalence of IAH was 34% upon ICU admission (9). Individuals who possess pre-existing conditions, such as obesity, intra-abdominal tumors, or ascites, are particularly susceptible to the development of ACS due to their increased risk of experiencing IAH (10).

Among the most prevalent risk factors for ACS is the administration of large amounts of fluid during resuscitation, typically exceeding 3 liters of crystalloid or non-crystalloid solutions (6). Severe abdominal trauma resulting from blunt or penetrating injuries, or even major surgery can lead to internal bleeding, organ harm, or significant tissue swelling, thereby increasing the likelihood of ACS. Furthermore, conditions such as sepsis, pancreatitis, and prolonged mechanical ventilation have been identified as additional factors that can elevate the risk of developing ACS (10)(11).

PATHOPHYSIOLOGY

Due to the relatively limited space within the abdomen and the restricted capacity of the abdominal wall to stretch, heightened pressure can swiftly result in organ damage (12). The underlying mechanism of abdominal compartment syndrome (ACS) involves a series of events that ultimately undermine the blood flow and functioning of organs. Initially, elevated intra-abdominal pressure (IAP) leads to the compression of the inferior vena cava and renal veins, resulting in diminished venous return and impaired renal blood circulation. In this context, insufficient renal perfusion pressure (RPP) and renal filtration gradient (FG) have been identified as critical factors contributing to renal failure induced by elevated IAP. The renal filtration gradient (FG) refers to the mechanical force exerted across the glomerulus and can be quantified as the disparity between the glomerular filtration pressure (GFP) and the proximal tubular pressure (PTP) (6). When intra-abdominal pressure is present, it is assumed that PTP is equivalent to IAP, meaning that GFP can be esti-

mated as the discrepancy between mean arterial pressure (MAP) and IAP (13). Hence, variations in intra-abdominal pressure (IAP) can exert a more pronounced influence on renal function and urine production compared to fluctuations in mean arterial pressure (MAP). Consequently, a reduction in urine output, referred to as oliguria, is frequently among the initial observable signs of intra-abdominal hypertension (IAH) (6,13). The degree of kidney damage is directly related to the rise in intra-abdominal pressure (IAP). As a result, individuals with normal or elevated blood pressure need a more substantial increase in IAP to trigger renal dysfunction (14).

Additionally, elevated IAP can impede arterial blood flow to the abdominal organs, causing ischemia and impaired tissue oxygenation. As IAP continues to rise, it can lead to decreased cardiac output and increased systemic vascular resistance, further compromising organ perfusion. The pressure exerted by the contents within the abdomen can hinder respiratory mechanics, resulting in an imbalance between ventilation and blood flow, leading to respiratory difficulties (13). ACS may negatively affect gastrointestinal function, causing reduced gut motility, heightened bacterial translocation, and an inflammatory response throughout the body. A study by M. Smit et al. analyzed the presence of IAH in 59 patients with severe acute pancreatitis. 29 patients had IAH, 13 (44.8%) of which developed ACS and 8 out of 13 (61.5%) had bowel ischemia. The results demonstrated high mortality rates for ACS complicated by bowel ischemia (15) (16).

DIAGNOSIS

Accurate identification of ACS necessitates the assessment of IAP, which should be conducted with a low threshold of suspicion. To determine IAP, there are several indirect measurement methods available, such as utilizing catheters placed in the intragastric, intracolonic, intravesical, or inferior vena cava sites (17).

The standard approach to screening for IAH and ACS involves measuring pressure within the bladder. The process involves the following steps (18–20):

1. The patient's Foley catheter drainage tube is clamped.

2. Normal saline, usually approxi mately 25 mL in volume, is introduced into the bladder via the Foley catheter's aspiration port, ensuring proper and sufficient filling of the catheter with fluid.

3. An 18-gauge needle, linked to a pressure transducer, is inserted into the aspiration port. In certain contemporary Foley catheter configurations, a needle-free connection mechanism can be employed for this task.

4. The pressure is documented during the patient's exhalation phase while lying flat on their back, ensuring the absence of any abdomina muscle contractions. It is essential to calibrate the transducer at the midaxillary line level.

Precise bladder pressure measurement can face challenges like intraperitoneal adhesions, pelvic hematomas, pelvic fractures, abdominal packing, or a neurogenic bladder (17).

The measurement of bladder pressure is best done when the patient is both intubated and under the effects of chemical paralysis. By temporarily immobilizing the patient's respiratory efforts and muscle activity through intubation and chemical paralysis, the measurement of bladder pressure becomes a highly reliable diagnostic tool (17).

6.MANAGEMENT

SUPPORTIVE MEASURES AND INTERIM INTERVENTIONS

The objectives of providing supportive care for individuals experiencing IAH involve appropriate body positioning, enhancing the flexibility of the abdominal wall (such as managing pain, administering sedation, inducing paralysis, utilizing mechanical ventilation), and decreasing the volume within the abdomen through abdominocentesis if necessary (21–24).

PATIENT POSITIONING

Special consideration should be given to the placement of the patient, ensuring they are positioned supine. It's important to note that elevating the head of the bed beyond 20°, a practice often employed to mitigate the occurrence of ventilator-associated pneumonia, can elevate IAP and affect the accurate measurement of IAP (25,26).

PARALYSIS AND VENTILATORY SUPPORT

Excessive peak and mean airway pressures can pose challenges. To address this, it may be necessary to implement measures such as reducing tidal volume, utilizing a pressure-limited mode, and/or allowing permissive hypercapnia. In cases of severe hypercapnia, pharmacologic paralysis

may be required to relax the abdominal wall and reduce carbon dioxide production, thus facilitating improved ventilation. Positive end-expiratory pressure (PEEP) may reduce ventilation-perfusion mismatch and improve hypoxemia (27).

REDUCTION OF INTRA-ABDOMINAL VOLUME

To decrease the volume within the abdomen, several strategies can be employed. These include preventing a positive fluid balance following initial resuscitation, removing intraluminal contents, evacuating ascites or hematomas occupying the intra-abdominal space whenever possible, and relieving bladder pressure.

ABDOMINAL DECOMPRESSION

Surgical decompression is used when supportive care fails and is considered the definitive management (28). The established method involves performing a midline incision along the linea alba to access the abdominal cavity, followed by employing temporary abdominal wall closure to keep the abdomen open (29). Surgical decompression should be considered for patients with an intra-abdominal pressure exceeding 25 mmHg (30,31).

Several clinicians recommend considering surgical decompression at a lower IAP range (e.g., 15 to 25 mmHg) in the setting of organ dysfunction, based on their belief that this would lead to enhanced organ perfusion, improved patient outcomes, and prevention of ACS (28,31–33). The decision for surgical decompression is not light as it comes with potential complications. These include the increased risk for abscess or fistula formation, and abdominal wall hernia formation (15)(34).

Alternative perspectives among clinicians suggest that the decision for surgical decompression should be based on the APP. According to a retrospective study, an APP below 50 mmHg demonstrated higher sensitivity and specificity in predicting mortality compared to either the mean arterial pressure or the intra-abdominal pressure alone (35).

IMPACT OF THE NEW RECOMMENDATIONS ON INCIDENCE AND PREVALENCE OF IAH/ACS

The WSACS introduced new definitions and guidelines for managing ACS in 2013 (36). The reported incidence of IAH in critically ill patients has displayed significant variability, ranging from 31% to 59% (37–42). Older studies reported rates of ACS between 8% and 12% (38–41), whereas more recent reports indicate significantly lower rates, ranging from 1.1% to 4% (43,44). This discrepancy can be attributed to the use of diverse definitions and measurement methods for IAH.

One of the most comprehensive epidemiological studies on IAH and ACS prevalence was conducted by Iyer et al in 2014. They included all ICU patients who had a urinary catheter inserted in their study. The results revealed a higher incidence of IAH at 39% with a similar incidence of ACS at 2%. The study aimed to develop a screening tool for detecting IAH, consisting of 6 risk factors (obesity, hemoperitoneum/ pneumoperitoneum or intra-peritoneal fluid collection, resuscitation >2.3L, abbreviated SOFA > 4, lactate > 1.4 mmol/L). Iyer et al found that the presence of at least three risk factors, as per their screening tool, in an ICU-admitted patient had the highest kappa value. This combination provided sensitivity and specificity rates of 75% and 76% for all grades of IAH, along with sensitivity and specificity rates of 91% and 62% for grades II and above (45).

In the year 2012, Kim and colleagues examined the occurrence of IAH within a cohort of 100 consecutive patients in the intensive care unit (ICU). Their findings revealed that 42% of the patients experienced IAH, while 4% developed ACS. The study also pinpointed certain risk factors linked to IAH, including a BMI exceeding 30, elevated central venous pressure (CVP), the presence of abdominal infection, and sepsis upon admission (43).

Blaser et al. aimed to investigate whether an expanded criteria for IAH monitoring would enhance the detection rate. Blaser's study categorized patients into three groups according to the time point of data collection. Their analysis showed that the expanded criteria did not have a significant impact on the rate of IAH detection. IAH occurred in 19.9% of patients in the first period, 20.3% in the second period, and 20.1% in the third period. The study also reported an ACS rate of 1.1% (46), which could be attributed to its exclusive focus on mechanically ventilated patients, unlike previous literature.

According to the latest guidelines established by WSACS, it is advised to commence the monitoring of Intra-Abdominal Pressure (IAP) whenever any recognized risk factor for IAH/ACS is present (36). The 2007 WSACS guidelines stipulated that IAP measurement should be conducted if the patient is critically ill, experiencing deteriora-

tion, and exhibits two or more supplementary risk factors connected to IAH (47). Iyer et al. showcased the effectiveness of their screening tool, which comprises six closely linked risk factors. Their findings indicated that having a minimum of one risk factor results in a sensitivity of 99% and a specificity of 15%. (48)(45). Iyer's methodology, resembling the 2007 recommendations, underscored the advantages of implementing a more stringent approach aimed at reducing the frequency of unnecessary IAP measurements. Consequently, it calls for a reassessment of the risk factors linked to IAH and the criteria that guide us in deciding when to monitor IAP.

LIMITATIONS OF THE WSACS ALGORITHM AND FUTURE DIRECTIONS IN THE MANAGEMENT OF ACS

Since the WSACS algorithms were concluded almost 10 years ago, for many, they may be considered outdated and in need of reformation. They mainly rely on Intra-Abdominal Pressure (IAP) measurements, which doesn't consider the possibility for rapid deterioration that may complicate ACS. It is worth mentioning however that these algorithms state the need for an experienced clinician for the clinical judgment and in some sense acknowledge their own limitations. Because ACS can lead to many potential complications, both surgical and non-surgical, creating one-size-fits-all guidelines for such a complex condition is probably destined to be incomplete and will unlikely completely replace physician judgment.

Despite this, those algorithms can definitely be enhanced to incorporate a more holistic and dynamic approach that moves beyond just checking Intra-Abdominal Pressure (IAP) into using imaging such as ultrasonography and computed tomography and labs more effectively. Such approach will also provide us with more imaging archives which can serve as a library to derive research from to advance our knowledge of this condition. This new algorithm has the potential to be personalized for the specific patient risk factors and may benefit from artificial intelligence to calculate scores to determine the overall risk for ACS. In conclusion, ACS management is a rich field and is envisioned to undergo major advancements after updating current management algorithms.

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The authors report no proprietary or commercial interest in any product mentioned or concept discussed in this article.

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