CHAPTER 2

ABDOMINAL COMPARTMENT SYNDROME

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INTRODUCTION

Abdominal compartment syndrome (ACS) is a condition in which intra-abdominal pressure (IAP) is >20 mmHg, leading to intra-abdominal hypertension (IAH) that is associated with a new organ failure or dysfunction. IAH is defined as a steady IAP ≥ 12 mmHg. ACS has significant relevance in the surgical practice and the care of critically ill patients, because it has tremendous effects on multiple organ systems. ACS is classified as primary, secondary and recurrent. Mortality rate of ACS is reported between 40 and 100%. Recent studies have demonstrated a high prevalence of this condition (4-12%) in medical/surgical patients admitted to the ICUInterest in IAH and ACS as causes of significant morbidity and mortality among the critically ill patients has increased exponentially over the last decades. Early recognition and appropriate management of IAH and ACS significantly decreased morbidity and mortality in recent years. In this chapter definition, etiology and risk factors, pathophysiology, diagnosis, medical and surgical treatment of ACS is discussed.

DEFINITIONS

Intraabdominal Pressure (IAP)

Intraabdominal pressure is the pressure in the abdominal cavity. An increase in the volume of retroperitoneal or abdominal contents leads to an increase in IAP. IAP is defined as a steady-state pressure concealed within the abdominal cavity (Papavramidis et al., 2011). IAP varies with respiration. IAP increases with diaphragmatic contraction (inspiration) and increases with diaphragmatic relaxation (expiration) (Park and Han, 2015). IAP is expressed as mmHg and measured in a patient in the supine position in absence of abdominal muscle contractions. According to Pascal's law, IAP measured at one point in the abdomen is assumed to represent the IAP throughout the abdomen (De Laet and Malbrain, 2007). The intermittent indirect IAP is measured through transduction of the pressure within the bladder, while the continuous indirect IAP is measured

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with a continuous bladder irritation or with a balloon-tipped catheter. Abdominal perfusion pressure (APP) is calculated as mean arterial pressure – IAP.

Normal IAP ranges from sub-atmospheric to 0 mmHg. Chronic IAP elevations are seen with certain physiological conditions including pregnancy and morbid obesity (Papavramidis et al., 2009). IAP is frequently elevated to 5-7 mmHg in critically ill patients. IAP has been associated with recent abdominal surgery, organ failure, sepsis, changes in body position and the need for mechanical ventilation (Lerner, 2008). Significantly elevated IAP causes intra-abdominal hypertension (IAH), which profoundly affects local and systemic tissues and organ systems that if left untreated, may lead to lethal organ failure (Pereira, 2019).

Intraabdominal Hypertension (IAH)

The upper limit of IAP is generally accepted as 12 mmHg, which reflects an increase in normal pressure from clinical conditions such as chronic obstructive pulmonary disease (COPD) and obesity (Lambert et al., 2005). On the other hand, IAH is defined as a repeated and sustained pathologic increase in IAP>12 mmHg (Carlotti and Carvalho, 2009). In another definition, an IAH ≥12 mmHg recorded by three standard measurements with 4 to 6 hours intervals or an APP ≤60 mmHg recorded by two standard measurements with 1 to 6 hours intervals (Milanesi and Caregnato, 2016). Based on the level of IAP, IAH is graded as seen in Table 1.

Table 1. Grading of IAH

Intraabdominal Pressure (mmHg)	Grade
12 – 15	I
16 – 20	II
21 – 25	III
> 25	IV

IAH can also be classified according to its duration as hyperacute, acute, subacute or chronic IAH. *Hyperacute* IAH lasts for seconds and is resulted from coughing, sneezing, laughing etc. *Acute* IAH lasts for hours and results in abdominal trauma or intraabdominal hemorrhage. Subacute IAH lasts for days and is caused by septic shock or severe acute pancreatitis. Chronic IAH lasts for months or years and is caused by pregnancy or morbid obesity (De Waele et al., 2009).

Abdominal Compartment Syndrome (ACS)

According to the World Society of Abdominal Compartment Syndrome (WSACS),

ACS is defined as a sustained IAP>20 mmHg (Grade III and Grade IV IAH), with or without APP<60 mmHg, that is associated with new organ failure or dysfunction (Rajasurya and Surani, 2020). It is unclear whether ACS is a phenomenon, which leads to multiorgan failure, or an epiphenomenon, which develops in conjunction with other organ dysfunction (Sosa et al., 2019). Since patients suffering from ACS are usually critically ill, organ dysfunction caused by ACS may be mistakenly attributed to the primary illness of these patients (Kimball, 2021).

Based on the duration and cause of IAH-induced organ failure, ACS is classified as primary, secondary and recurrent. *Primary* ACS occurs as a result of an injury or disease that develops within the abdomen. *Secondary* ACS is caused by conditions outside the abdomen including sepsis, major burns, bacteremia, acute respiratory failure, capillary leak and pneumonia that require fluid resuscitation (Chabot and Nirula, 2017). *Recurrent* ACS occurs after prophylactic or therapeutic surgical treatment of primary or secondary ACS. Recurrent ACS reflects the resurgence of ACS after resolution of a previous episode (Eggers and Lindahl, 2017).

HISTORY OF ACS

The compartment syndrome was first described in limbs by Richard Volkmann in 1811 (Volkmann, 1881). Volkmann described a condition in which increased pressure within a closed fascial cavity decreases blood perfusion to the muscles, leading to a contracture (Volkmann, 1881). The association between increased IAP and respiratory function was presented for the first time by Etienne-Jules Marey in 1863. In this study, it was stated that the effects of respiration on the thorax are the inverse of the effects on the abdomen (Marey, 1863). In 1911, H. Emerson conducted experimental studies in dogs and concluded that IAP elevates with the contraction of the diaphragm and decreases with muscle paralysis and that elevated IAP may result in mortality due to cardiac failure (Emerson, 1911). In 1951 M.G. Baggot described abdominal dehiscence as the major factor increasing IAP (Baggot, 1951).

However, the recognition of the abdomen as a compartment and the concept of IAH that leads to ACS have recently attracted interest. The development of laparoscopic surgery provided a significant contribution to observation of the complications in the cardiovascular and respiratory systems related to IAH. ACS was described for the first time by I. Kron, P.K. Harman and S.P. Nolan in 1984. The authors stated that the direct measurement of IAP through an indwelling transurethral bladder is a safe and simple diagnostic method to determine ACS (Kron et al., 1984). In 2004, the WSACS was founded and the raising interest on ACS became formal. WSACS consisted of international experts of anesthesiology,

trauma and general surgery, critical care, vascular surgery, and other specialties and defined the terms of IAH and ACS (Kirkpatrick et al., 2015). In the past two decades ACS has been recognized in a wide range of surgical patients and treated aggressively.

ETIOLOGY AND RISK FACTORS

ACS develops as an acutely and significantly increased IAP due to several factors. Determination of the etiology of IAP enables selection of the best method for treatment of ACS. An initial elevation in IAP up to 18 mmHg following abdominal hernia repair may be tolerated and could be just observed (Petro et al., 2015). However, the same value of IAP in patients with acute pancreatitis requires massive fluid resuscitation in order to preserve organ perfusion and poses a risk for developing ACS. In this condition, immediate action and measures should be taken including sedation and muscle relaxation to control IAP (De Laet et al., 2020).

Etiological factors for spontaneously increased IAP include intraabdominal abscess, peritonitis, intestinal obstructions, acute pancreatitis, acute mesenteric ischemia and ruptured abdominal aortic aneurysm. Postoperative causes of ACS include peritonitis, ileus, intraperitoneal hemorrhage and abscess, while post-traumatic causes include intra – and retro-peritoneal hemorrhage, and post-resuscitation visceral edema. Iatrogenic etiologies include laparoscopic procedures, abdominal packing and abdominal closure under tension (De Laet et al., 2020). In addition, the most common etiologies of ACS can be divided into intra-abdominal and extra-abdominal causes (Table 2) (Cheatham, 2009).

Table 2. Etiologies of ACS

ACS Etiology	
Intra-abdominal Causes	**Extra-abdominal Causes**
Pancreatitis	Burn eschar
Intra – or extra-peritoneal hemorrhage	Antishock trousers
Aortic rupture	Hernia repair
Bowel edema or distension	Laparotomy closure under tension
Abdominal packing	
Ascites	
Mesenteric venous obstruction	

The independent risk factors for developing ACS include a body mass index

(BMI) > 30 Kg/m2, hypothermia (<33°C), massive transfusion and resuscitation with >5000 ml of crystalloids within 24 hours (Hecker et al., 2016). These factors are essential in many conditions including wounds, burns, and pancreatitis that require fluid resuscitation and can induce ACS. Risk factors for developing ACS include increased intra-abdominal volume, decreased abdominal wall compliance and increased intra-abdominal contents.

Risk factors for IAH and ACS can be further examined under surgical and medical factors. Surgical factors manifest in the postoperative period end include hemorrhage, edema following extensive dissections, primary closure of abdominal wall defects, damage control laparotomy, ileus, multiple trauma/burns, acidosis, coagulopathy, polytransfusion and visceral edema following fluid resuscitation (Papavramidis et al., 2009).

Medical risk factors include ascites secondary to massive fluid resuscitation, peritonitis, intra-abdominal abscess, acute pancreatitis, gastric dilatation, intra-abdominal or retroperitoneal tumor, pneumoperitoneum, gastroparesis, acute respiratory failure, probe positioning and peritoneal dialysis (Papavramidis et al., 2009).

PATHOPHYSIOLOGY OF IAH AND ACS

Increased IAP is determined by intra-abdominal volume and abdominal wall compliance (De Waele et al., 2009). Altered perfusion to tissues due to increased pressure within a fixed volume results in both IAH and ACS (Maluso et al., 2016). ACS has wide effects on different body systems. ACS causes a decrease in cerebral blood perfusion in the central nervous system and increased intracranial pressure (Montalvo et al., 2020). The presence of elevated IAP can be an extracranial factor for occurrence of intracranial hypertension.

Cardiovascular failure and dysfunction are frequently encountered in patients with ACS. In order to restore end-organ perfusion and functioning, preload, contractility and afterload should be accurately assessed and optimized. An elevated IAP can increase systemic vascular resistance, leading to an increased afterload, causing a decrease in cardiac output, resulting in hypotension (Maluso et al., 2016). Compression of the heart due to diaphragmatic elevation alters cardiac function (Cullen et al., 1989). Regarding venous blood return from the lower limbs, IAH increases the inferior vena cava pressure resulting in significantly increased femoral venous pressure (Papavramidis et al., 2011).

In the pulmonary system, an increased IAP causes elevation of the diaphragm and as a result of increased intrapleural pressure, residual functional capacity,

thoracic volume and compliance decrease (Maluso et al., 2016). IAH leads to an extrinsic compression of the pulmonary parenchyma, which can be attributed to cephalic displacement of the diaphragm due to increased intra-abdominal volume. In addition, IAH leads to pulmonary hypertension through an increase in intrathoracic pressure, resulting in direct compression of pulmonary vessels, causing right an left ventricular dysfunction (De Laet et al., 2020). This in turn results in respiratory dysfunction characterized by reduced capillary flow, decreased oxygen transport through the pulmonary capillary membrane and reduced carbon dioxide excretion (Karbing et al., 2020).

The cause of renal dysfunction is multifactorial in ACS. In the urinary system in the presence of normal initial kidney function, renal dysfunction presents as oliguria at an IAP of 15 mmHg and anuria at an IAP of 30 mmHg due to compression of the renal parenchyma, decreased renal perfusion and compression of the renal vein that lead to decreased microcirculation in the renal cortex followed by reduction in urinary output (Mohmand and Goldfarb, 2011). The renal dysfunction include decreased proportional and absolute renal blood flow, increased water and sodium retention and reduced glomerular filtration (Copur et al., 2022).

A change in the micro-perfusion of the mucosa leads to elevated serum lactate that decreases pH, causing mesenteric ischemia (Hecker et al., 2016). On the other hand, intestinal ischemia can present as interstitial edema and/or ileus that raise intra-abdominal volume and worsen ACS. Following 60 minutes of sustained IAP greater than 25 mmHg, mucosal blood flow changes even in well-resuscitated patients (Kirkpatrick et al., 2016).

The liver is particularly vulnerable to injury in the presence of elevated IAP. An increased IAP can lead to hepatic hypoperfusion due to a reduction in blood flow through the portal vein. This may result in liver failure due to altered elimination of lactate in plasma, leading to metabolic acidosis. Increased IAP results in decreased venous portal flow, decreased hepatic arterial flow and increased portacollateral circulation. All of these manifestations exert physiological effects with altered glucose metabolism, altered mitochondrial function and decreased lactate clearance (Pereira et al., 2021).

Poor intestinal perfusion due to decreased splanchnic circulation, decreased venous return as a result of compression of the inferior vena cava and decreased glomerular blood flow can cause systemic acidosis and cardiovascular collapse (Maluso et al., 2016).

DIAGNOSIS

The diagnosis of ACS is based on a high suspicion and recognition of a patient with risk factors, determining clinical syndrome and measured IAP. Early recognition of IAH is the essential part for preventing ACS. In order to determine IAH, IAP is measured by insertion of a catheter into the inferior vena cava or indirectly using an intragastric, intravesical or urinary catheter. Direct measurement of IAP through inferior vena cava is the most accurate method, but it requires access to the peritoneal compartment and poses risks related to invasive abdominal procedures. Therefore, this method is not widely used in the measurement of IAP. Intravesical measurement of IAP is a widely used method for determining the presence of IAH and ACS. This method is performed through the modified Kron technique and has advantages of reliability, sensitivity, ease of use, reproducibility, low cost, less invasion and minimal complications and side effects (Hunt et al., 2014).

Intragastric pressure is an alternative method for indirect IAP measurement. This method is performed through orogastric or nasogastric tube manometry (Sugrue et al., 2002). However, this method can be unreliable because of the confounding effects of the increased pressure due to gastric contractions. Although this limitation can be overcome with continuous measurement through a gastric balloon, there are other potential confounding factors such as the administration of enteral feeds (Maluso et al., 2016).

WSACS recommend using an urinary catheter, because it is easy to perform, accurate, minimally invasive and cost-effective. In this measurement, the patient must be in supine position with the bed at 0^{o} for a correct measurement (Kirkpatrick et al., 2013). According to the WSACS, IAP should be expressed as mmHg at the end of expiration with no abdominal muscle contractions (Rastogi et al., 2014). In the case of IAH, IAP is recommended to be measured every 4-6 hours or even permanently in certain conditions such as critical organ dysfunction.

Sensitivity and positive predictive value of physical examination in the diagnosis of ACS has been reported between 40-60% (Milanesi and Caregnato, 2016). Clinical parameters used in the diagnosis of ACS are shown in Figure 1.

Imaging examinations including plain abdominal X-ray, abdominal CT or abdominal ultrasound have a poor specificity for detecting an increase in IAP (Roberts et al., 2016).

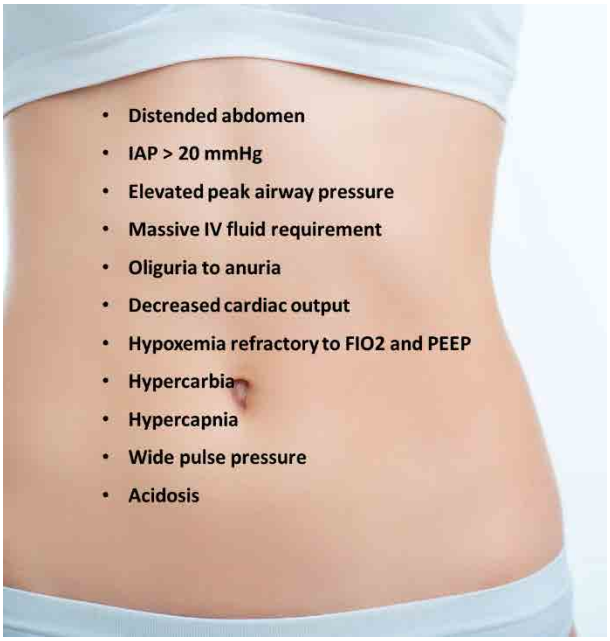

Figure 1. Clinical parameters using in the diagnosis of ACS

MANAGEMENT OF ACS

Medical Treatment

Before considering surgical decompression, less invasive medical therapy options should be considered first, particularly in cases of secondary IAH or ACS. Mild IAH can often just be observed, while medical therapy should always be used in the case of IAH progressing to ACS. Dynamics of IAP and general status of the patients should be carefully monitored for timing of medical treatment in order to avoid ACS. IAP should be measured regularly. The main strategies of medical therapy include improving abdominal wall compliance, evacuation of extraluminal and intraluminal contents and correction of fluid balance in order to improve worsening ACS (De Laet et al., 2020). Options of non-surgical management of ACS are given in Figure 2.

Figure 2. Options for non-surgical management of ACS

Deep sedation, analgesia and neuromuscular blockade can restore abdominal wall compliance by alleviating pain, agitation and accessory muscle use. Ileus is commonly observed in patients with peritonitis, major trauma, pancreatitis, abdominal surgery and following mass fluid resuscitation (Newman et al., 2020). Decompression through nasogastric tube and rectal tube help in reduction of IAP in patients with IAH. Prokinetic agents and bowel enemas can help in evacuation of intraluminal contents and decreasing visceral volume. It has been reported that patients undergone percutaneous drainage placement have improved outcomes compared to open abdominal decompression (Cheatham and Safcsak, 2011). Diuretics and renal replacement therapy for aggressive fluid removal may help in reducing IAP. Other medical treatment procedures to decrease IAP include the use of gastric suctioning, paracentesis, rectal enemas, continuous negative abdominal pressure, prokinetics such as metoclopramide, erythromycin and cisapride, furosemide, sedation and curarization.

Surgical Treatment

When IAP can not be reduced with nonsurgical techniques, surgical abdominal decompression must be initiated promptly since it is the definitive treatment to decrease the risk of mortality from ACS. Presumptive decompression should be considered at the time of laparotomy in patients exhibiting multiple risk factors for IHA and ACS. When combined with negative pressure peritoneal therapy, decompression laparotomy decreases IAP, reduces the transmission of inflammatory mediators into the bloodstream, improves visceral perfusion and reduces the risk of developing multiorgan dysfunction (Kubiak et al., 2010). Open abdomen is a surgical management strategy in which the incision at abdominal wall is left unrepaired at the end of the procedure to relieve IAP. There has been

increased awareness of the devastating effects of IAH in recent years and this has led to more open abdomen in ICUs, which also decreases the risk of developing ACS (De Waele et al., 2006). Most patients tolerate primary fascial closure of the abdomen within 5-7 days if it is decompressed before organ failure occurs.

Although open abdomen and surgical decompression is the definitive treatment method in ACS, it can also lead to several complications including development of fistulas, protein loss through removal of peritoneal fluid, and life threatening hemorrhagic complications such as reperfusion syndrome (Miller et al., 2004). Due to these risks, surgeons should attempt to perform rapid definitive closure as soon as it is possible to reduce postoperative complications and mortality (Hecker et al., 2016).

Surgical methods used to reduce the risk of developing IAH and ACS include open abdomen technique with temporary closure using silo technique (Bogota bag), patch technique, temporary closure using vacuum and skin-only technique using towel clips (Gracias et al., 2002). These are simple solutions for the management of an open abdomen and provide control of fluid loss. Some of these methods are explained briefly below.

Bogota Bag

The Bogota bag consists of a bag with 3 L of saline solution that is used to cover abdominal content and sutured to the skin. This technique has several advantages including ease of application, being accessible, low cost and being not adherent. Its disadvantages are the need for sterilization of the materials, skin loss and insufficient control of fluid leaks (Seternes et al., 2010).

Mesh

The absorbable meshes are composed of polyglycolic acid and polyglactin 910. Both can be secured to the fascia or skin and have great resistance strength. The higher incidence of enterocutaneous fistula is the main disadvantage of this method. Polytetrafluoroethylene and polypropylene meshes are the most widely used nonabsorbable meshes. These meshes are prone to infections and colonization. They should be removed before definitive closure (Petersson et al., 2007).

The Wittmann Patch

This patch is made of polypropylene and polyamide. It has a Velcro zipper. The sheets of the patch are sutured to the fascial edges. Definitive closure can be performed after the patch is removed. It has disadvantages including manipulation of the fascia that may lead to fascial necrosis and reduced fluid leakage control.

As an advantage, it prevents abdominal domain loss and is easy to re-entry (Demetriades and Salim, 2014).

Expanded Polytetrafluoroethylene (ePTFE) Mesh

Decompression laparotomy associated with temporary abdominal closure using ePTFE mesh enables reduction of IAP in patients with severe acute pancreatitis. This method allows early abdominal reconstruction (Robin et al., 2013). ePTFE smesh has several advantages such as absence of adherence, enabling re-examining the abdominal cavity through the mesh and the resistance of the material to high traction pressures. In addition, progressive approximation of the ePTFE mesh can be performed in order to facilitate later definitive abdominal wall closure (Cheatham and Safcsak, 2011).

CONCLUSION

ACS is a life-threatening condition characterized by sustained acute elevation of IAP more than 20 mmHg. Studies have shown a high incidence of IAH and ACS in ICU patients. Inflammatory intrabdominal complications and large volume fluid resuscitations are among the frequent etiologies of ACS. Regular monitoring of IAP in patients at risk is vital for early diagnosis and treatment of IAH and ACS. When IAH is diagnosed, first medical therapy should be initiated with bowel and gastric decompression, paracentesis, evacuating intraluminal content, diuresis and sedation. Surgical abdominal decompression is the definitive treatment method when non-surgical methods fails.

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