Management of Intra_Abdominal Hypertension & Abdominal Compartmental Syndrome in Intensive Care Unit

Essay

Submitted for Partial Fulfillment of Master Degree
In Intensive Care

By
Dina Mohamed Fathi Abdelrahman
M.B.,B.Ch.

Supervised by

Prof. Dr. Sahar Kamal Aboul Ella
Professor of Anesthesia and Intensive Care
Faculty of Medicine - Ain Shams University

Dr. Mohamed Anwar El Shafei
Professor of Anesthesia and Intensive Care
Faculty of Medicine - Ain shams University

Dr. Mohamed Saleh Ahmed
Lecturer of Anesthesia and Intensive Care
Faculty of Medicine - Ain shams University

Faculty of Medicine
Ain Shams University
2015

Introduction

Abdominal compartmental syndrome (ACS) is a sustained intra_abdominal pressure greater than 20mmHg [with or without an abdominal perfusion pressure (APP) <60mmHg] associated with new organ dysfunction or failure. Intra_abdominal hypertension (IAH) and ACS are common associated with substantial morbidity &mortality among critically ill adults. These conditions have been linked with acute & chronic renal failure, multiorgan dysfunction syndrome (MODS), increased length of intensive care unit, hospital stay and elevated mortality. Identifying critically ill patients at risk for IAH&ACS is there for important in order to guide identification of the source population for future treatment trials and to stratify patients into risk groups based on prognosis ***(Malbrain and Cheatham, 2011).***

The World Society of the abdominal compartmental syndrome (WSACS) categorizes conditions that causes ACS as primary (surgical), secondary (medical) & recurrent. The primary conditions are ones that need surgical or interventional radiological treatment. Secondary conditions are due to medical causes that do not require surgery or radiological intervention as an initial therapy. Recurrent conditions are ones in which ACS develop after surgical or medical treatment of the primary or secondary causes of ACS ***(Cheathman and Safcsak, 2010).***

Risk factors for IAH and ACS including diminished abdominal wall compliance, increased intra-luminal contents, increased intra_abdominal contents, increased retroperitoneal contents, capillary leak &fluid resuscitation ***(Kim et al., 2012).***

Medical Management of IAH&ACH is done through improving abdominal wall compliance, evacuating intra luminal contents, evacuating intra_abdominal contents, correcting positive fluid balance & organ support by optimizing systemic &regional perfusion ***(De Keulenaer et al., 2011).***

Presumptive decompression should be considered at the time of laparotomy in patients who demonstrate multiple risk factors for IAH&ACS (but giving the hazards of managing the open abdomen, it is at present not recommended to do presumptive decompression for IAH without organ dysfunction).Surgical decompression should be performed in patients with ACS is refractory to other treatment options ***(De Waele et al., 2010).***

Aim of the Work

To identify the pathophysiology of intra_abdominal hypertension and abdominal compartmental syndrome, and give a spotlight on the new guidelines for its management.

Chapter (1): Intra-abdominal Hypertension and Abdominal Compartment Syndrome

Introduction:

Intra-abdominal hypertension and abdominal compartment syndrome (IAH/ACS) contributes to multiorgan dysfunction syndrome (MODS), failure, and death in surgical critically ill patient populations ***(Ameloot et al., 2012).***

Intra-abdominal hypertension and ACS are conceptually defined by a spectrum of elevated pressures within this enclosed space. Its importance resides in the impairment of its organs' viability. Intra-abdominal hypertension/ACS is categorized under the obstructive type of circulatory failure/shock ***(Ortiz-Diaz and Lan, 2014).***

Despite critical illness, intraperitoneal pressures are no more than 5 to 7 mmHg ***(Sanchez et al., 2001).*** Although other routes exist with the purpose of measuring intraabdominal pressure such as transgastric, intravascular (through the inferior vena cava), or percutaneous, intra-abdominal pressure's criterion standard for measurement and diagnosis is intermittent or continuous bladder pressure measurement with urethral catheterization ***(Sanchez et al., 2001)***.

The World Society of Abdominal Compartment Syndrome has derived a consensus statement with definitions of IAH and ACS in 2006 and 2013 ***(Kirkpatrick et al., 2013).*** Intra-abdominal hypertension is defined as

intra-abdominal pressure exceeding 12 mmHg. By consensus, ACS is defined as an intra-abdominal pressure exceeding 20 mmHg associated with new organ dysfunction ***(Ortiz-Diaz and Lan, 2014).***

History:

For several decades, increased IAP has been increasingly recognized as both cause and consequence of many adverse events in critically ill patients. Increased IAP within the closed anatomic volume of the abdominal cavity can lead to decreased perfusion and ischemia of intra-abdominal organ ***(De Waele et al., 2011)***.

In addition, increased IAP also leads to physiologic changes and organ dysfunction beyond the abdominal cavity because of the close anatomic relationships with contiguous cavities. Depending on the severity of increased IAP and organ function, the condition is defined as intra-abdominal hypertension (IAH) or ACS ***(De Waele et al., 2011)***.

The harmful consequences of increased IAP initially were reported more than 100 years ago, and effects on the kidney were among the first described. In 1876, Wendt reported that an increase in IAP was associated with a decrease in urine output, and in 1947, Bradley and Bradley published a comprehensive experimental article describing the effect of IAP on kidney perfusion and function ***(Gillenwater, 1970; Shenasky, 1972).***

Several investigators have since noted similar effects in animal models and clinical studies in the critically ill. Presumably because measurement of IAP was cumbersome and clinicians were unaware of the dangers, clinical effects of IAP were not reported again until the early 1980s ***(De Waele, 2011).***

It was not until the landmark report by Kron et al., which reported that IAP could be monitored objectively and relatively easily through an indwelling intravesical catheter, that more clinical evidence was rapidly forthcoming concerning the deleterious effects of increased IAP on different organ systems ***(Kron et al., 1984)***.

Since then, the clinical importance of IAH and ACS essentially has been rediscovered, largely by physicians and surgeons taking care of the sickest of the sick in the ICU. This rediscovery also reflects in part an increasing incidence of IAH and ACS in critically ill patients who were treated more aggressively than ever before, both peri- and intraoperatively and in the ICU. However, changes in organ function in patients undergoing laparoscopic surgery have shown that even at lower pressures in the range of 12-20 mm Hg, IAH is relevant and affects organ function ***(Schwarte et al., 2004).***

Intra-abdominal pressure (IAP)

The abdomen may be considered as a closed box with walls that are either rigid (costal arch, spine, and pelvis) or flexible (abdominal wall and diaphragm). The

compliance of these walls and the volume of the organs contained within determine the pressure within the abdomen at any given time ***(Malbrain, 2013).***

Intra-abdominal pressure is defined as the steady-state pressure concealed within the abdominal cavity, increasing with inspiration (diaphragmatic contraction) and decreasing with expiration (diaphragmatic relaxation). IAP is directly affected by the volume of the solid organs or hollow viscera (which may be either empty or filled with air, liquid or fecal matter), the presence of ascites, blood or other space-occupying lesions (such as tumors or a gravid uterus), and the presence of conditions that limit expansion of the abdominal wall (such as burn eschars or third-space edema) ***(Malbrain et al., 2007).***

Hyperacute IAH refers to elevation of the intraabdominal pressure lasting only seconds. It is due to laughing, coughing, straining, sneezing, defecation, or physical activity. IAH with ACS due to gastric over-distention following endoscopy has been described ***(Malbrain et al., 2005).***

Acute IAH refers to elevation of the intraabdominal pressure that develops over hours. It is usually the result of trauma or intraabdominal hemorrhage and can lead to the rapid development of ACS ***(Gestring et al., 2011).***

Subacute IAH refers to elevation of the intraabdominal pressure that develops over days. It is most common in medical patients and can also lead to ACS ***(Gestring et al., 2011).***

Chronic IAH refers to elevation of intraabdominal pressure that develops over months (pregnancy) or years (morbid obesity). It does not cause ACS, but does place the individual at higher risk for ACS if they develop superimposed acute or subacute IAH ***(Malbrain et al., 2005).***

Normal IAP ranges from sub-atmospheric to zero mmHg. In the typical intensive care unit patient, however, IAP is commonly elevated to a range of 5–7 mmHg, while patients with recent abdominal surgery, sepsis, organ failure, or need for volume resuscitation may demonstrate IAPs of 10–20 mmHg ***(Cheatham et al., 2007).*** Normal IAP is ~5–7 mm Hg, with baseline levels in morbidly obese individuals often ranging from 9–14 mm Hg ***(Malbrain et al., 2005).***

Although this degree of IAH may affect organ function in other patients, it often appears to be tolerated in obese individuals. Normal IAP usually is lower in children ***(Cheatham et al., 2007).***

In general, an individual patient's physiologic state must be taken into account when interpreting IAP measurements. IAP typically is expressed in millimeters of mercury and conversion from centimeters of water may be necessary (1 mm Hg = 1.36 cm H2O ***(Malbrain et al., 2006)***.

IAH usually is associated with situations in which either increased abdominal volume or decreased abdominal

compliance may predominate, and often a combination of the 2 is to blame. The World Society of the Abdominal Compartment Syndrome (WSACS) recently listed conditions associated with these situations. Abdominal compartment syndrome — For research purposes, ACS is defined as a sustained intraabdominal pressure >20 mmHg (with or without abdominal perfusion pressure (APP) <60 mmHg) that is associated with new organ dysfunction ***(Malbrain et al., 2006).***

For clinical purposes, ACS is better defined as IAH-induced new organ dysfunction without a strict intraabdominal pressure threshold, since no intraabdominal pressure can predictably diagnose ACS in all patients ***(Moore et al., 2004).***

Patients with an intraabdominal pressure below 10 mmHg generally do not have ACS, while patients with an intraabdominal pressure above 25 mmHg usually have ACS ***(van Mook et al., 2002).*** Patients with an intraabdominal pressure between 10 and 25 mmHg may or may not have ACS, depending upon individual variables such as blood pressure and abdominal wall compliance ***(Cheatham et al., 2005):***

- Higher systemic blood pressure may maintain abdominal organ perfusion when the intraabdominal pressure is increased, since the perfusion pressure (APP) is the difference between the mean arterial pressure and the intraabdominal pressure.

- Abdominal wall compliance initially minimizes the extent to which an increasing abdominal girth can elevate the intraabdominal pressure. But when a critical abdominal girth is reached, abdominal wall compliance decreases abruptly. Further increases in abdominal girth beyond this critical level result in a rapid rise of intraabdominal pressure and ACS if untreated. Increased abdominal wall compliance due to chronic increased abdominal girth (eg, pregnancy, cirrhosis with ascites, morbid obesity) may be protective against ACS ***(De Waele et al., 2011).***

Every inspiration, spontaneous or during positive-pressure breathing, moves the diaphragm downward, increases intraabdominal pressure, and shifts blood volume from the splanchnic system into the systemic circulation. At the same time, venous flow from the lower extremities along the inferior caval vein decreases ***(Hofer et al., 2002; Galstian et al., 2011).***

During expiration, the diaphragm shifts upward, decreases blood flow from the splanchnic system, and increases blood flow from the lower extremities. These cyclic events overall do not dramatically affect venous return (VR) and cardiac output (CO) ***(Hofer et al., 2002; Galstian et al., 2011).***

However, a longer-lasting increase in intraabdominal pressure to any level lower than pressure within inferior caval vein may lead to a simultaneous increase in VR due

to shift of blood volume from the compliant splanchnic venous system toward the right atrium; on the other hand, such an increase in intraabdominal pressure may decrease VR secondary to an increase in venous resistance within the inferior caval vein and to a shift of the diaphragm upward, an increase in intrathoracic pressure (ThorP) with concomitant increase in intramural central venous pressure (CVP) ***(Hofer et al., 2002; Galstian et al., 2011).***

Such an increase in CVP does not reflect the volume status of a patient and may be associated with a decrease in VR, resulting from a decrease in the gradient between mean circulatory filling pressure (MCFP) and CVP. This effect can be modified by an increase in stressed volume (Vs), which in turn can be achieved by an infusion of additional fluid and/or an administration of a vasoconstrictor ***(Odeberg et al., 1994; Andersson et al., 1999; Goldberg, 2001).***

Such intervention is not always needed because of the activation of sympathetic and renin–angiotensin systems; this is associated with an increase of MCFP and maintenance of the needed MCFP–CVP gradient to preserve VR and CO ***(Koivusalo and Lindgren, 2000).*** Anesthetics, sedatives, and other interventions might minimize such a homeostatic response ***(Thorlacius and Bodelsson, 2004).***

Positions (Tilts) Different positions, e.g., head up versus head down, affect systemic hemodynamics

including function of the venous system. A head-up position (e.g., standing up) could be associated with a gravity-induced shift of blood volume from the upper to the lower part of the body ***(Hofer et al., 2002)***.

In healthy, awake patients, head-up or head-down positions do not affect blood pressure, CO, or CVP, because of immediate activation of sympathically mediated reflexes as well as the renin–angiotensin system and release of other vasoconstricting mediators ***(Moller et al., 2004),*** prevent such a dramatic shift of blood volume. However, during anesthesia, the head-up position is practically always associated with a decrease in CVP, CO, and blood pressure because the reflexes are blunted as the depth of anesthesia increases ***(Hofer et al., 2002).***

The head-down (Trendelenburg) position is always associated with an increase in CVP. However, CO and blood pressure may be maintained ***(Moller et al., 2004)*** or decreased ***(Hofer et al., 2002).***

Left ventricular end-diastolic area (reflecting volume) and intrathoracic blood volume are increased. After change from a head-down to a horizontal position in an anesthetized patient, a decrease in blood pressure and CO may occur ***(Reuter et al., 2003).*** It might be due to a failure to increase afterload or to hypovolemia, which may have been misinterpreted as normovolemia or hypervolemia secondary to high CVP with the head-down position ***(Hofer et al., 2002).***

A compartment syndrome exists when increased pressure in a closed anatomic space threatens the viability of the tissue within the compartment. When this occurs in the abdominal cavity it threatens not only the function of the intra-abdominal organs, but it can have a devastating effect on distant organs as well ***(De Laet and Malbrain, 2007).***

Recent animal and human data suggest that the adverse effects of elevated intra-abdominal pressure (IAP) can occur at lower levels than previously thought and even before the development of clinically overt abdominal compartment syndrome (ACS). The ACS is not a disease but truly a syndrome, a spectrum of symptoms and signs that can and mostly does have multiple causes. It is only recently that this condition received a heightened awareness ***(De Laet and Malbrain, 2007).***

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been shown to occur frequently in Intensive Care Units (ICU) patients and have been independently associated with mortality. Unlike many commonly encountered disease processes which remain within the purview of a given discipline, IAH and the ACS readily cross the usual barriers and may occur in any patient population regardless of age, illness, or injury ***(Malbrain et al., 2004).***

As a result, no one specific specialty can represent the wide variety of health care workers who might encounter patients with IAH and/or ACS in their daily practice ***(Malbrain et al., 2004).***

Definitions of IAH and ACS:

The first consensus paper by the world society of abdominal compartment syndrome (WSACS), published in 2006, contains a list of definitions related to IAH and ACS ***(Malbrain et al., 2006).***

These definitions are based on the best available scientific data today, but they are likely to undergo some minor changes in the future. The different methods used for diagnosis of ACS will be discussed here. Several surveys among clinicians show that many of them use clinical examination for the diagnosis of ACS. This has been shown to be unreliable with a sensitivity and positive predictive value of around 40-60% ***(Sugrue et al., 2002).***

The use of abdominal perimeter is equally inaccurate. Radiologic investigation with plain radiography of the chest or abdomen, abdominal ultrasound or CT-scan are also insensitive to the presence of increased IAP. However, they can be indicated to illustrate the cause of IAH (bleeding, hematoma, ascites, abscess…) and may offer clues for management (paracenthesis, drainage of collections…) ***(Sugrue et al., 2002; Kirkpatrick et al., 2000).***