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Elevated intra-abdominal pressure: A review of current knowledge

Elevated intra-abdominal pressure

Abstract

Elevated intra-abdominal pressure (IAP) is a known cause of increased morbidity and mortality among critically ill patients. Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) can lead to rapid deterioration of organ function and the development of multiple organ failure. Raised IAP affects every system and main organ in the human body. Even marginal sustained IAH results in malperfusion and may disrupt the process of recovery. Yet, despite being so common, potentially lethal condition, it still often remains unnoticed. In 2004, the World Society of the Abdominal Compartment Syndrome (WSACS), an international multidisciplinary consensus group, was formed to provide unified definitions, improve understanding and promote research in this field. Simple, reliable and nearly costless standardized methods of non-invasive measurement and monitoring bladder pressure allow early recognition of IAH and, consequently, starting optimized management in a timely manner. The correct, structured approach to treatment can have a spectacular effect and fully restore homeostasis. In recent years, significant progress has been made in this area with the contribution of surgeons, internal medicine specialists and anesthesiologists. Our review focuses on recent advances in order to present the complex underlying pathophysiology and guidelines concerning diagnosis, monitoring and treatment of this life-threatening condition.

Key Words: Abdominal compartment syndrome; Intra-abdominal hypertension; Intra-abdominal pressure; Multiple organ failure

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Core Tip: Elevated IAP is a common, life-threatening condition associated with increased morbidity and mortality. Unrecognized IAH quickly leads to the development of multiple organ failure; therefore, high alertness is necessary in relation to high-risk patients. Monitoring bladder pressure in this group allows early recognition of IAH/ACS and, consequently, timely intervention, which can have a spectacular effect and fully restore homeostasis. Although the progress made in recent years is significant, there are still open questions, leaving room for future research.

INTRODUCTION

Increased intra-abdominal pressure (IAP) has drawn attention and become an accentuated condition in critically ill patients over the last years [1-3]. IAP is defined as pressure concealed within the abdominal cavity, which oscillates adequate to the respiratory phase and abdominal wall resistance. It was first recognized in the 19th century with a recent increase in interest and publications in this field. In 1863, Marey noticed a link between the respiratory system and abdominal pressure for the first time. Promptly after this discovery, a damaging effect on other organs was described, inspiring scientists to explore this phenomenon in animal models in the early 20th century. In the following years, surgeons made a significant contribution to understanding gastrointestinal, cardiac and pulmonary complications and restoring their function through decompression of the abdominal cavity. Accumulation of clinical data throughout the last decades has led to a better understanding of underlying pathophysiology and its consequences. The impact of elevated IAP is not only limited to deterioration of intra-abdominal organs function but rather directly and indirectly

influences different systems and main organs [4]. Untreated pre-existing comorbidities, abdominal trauma or surgical interventions and extra-abdominal diseases like chronic kidney disease, heart failure or impaired respiratory function exacerbate the harmful effects of elevated IAP. In these high-risk subpopulations, even marginal sustained rise in IAP results in malperfusion and potential subsequent multiple organ failure.

In 2004, the World Society of the Abdominal Compartment Syndrome (WSACS), an international multidisciplinary consensus group, was formed from critical care, surgical and trauma experts to provide standardized definitions, promote research in this field and improve survival of patients affected by intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS). WSACS specialists published new definitions and recommendations for IAP measurement and management. According to created guidelines, normal IAP in critically ill adults ranges approximately from 5 to 7 mmHg. IAH is defined as a sustained or repeated rise in IAP above 12 mmHg and is divided into four grades, namely grade I: 12–15 mmHg, grade II: 16–20 mmHg, grade III: 21–25 mmHg and grade IV: > 25 mmHg. ACS is defined as a sustained IAP > 20 mmHg associated with new organ dysfunction or failure [5].

The following review focuses on current knowledge on elevated IAP, its relationship with deterioration of organ function and structured management of this condition, which can be applied in general internal and surgical practice.

EPIDEMIOLOGY AND RISK FACTORS

A series of the latest studies indicate a high prevalence of IAH ranging from 28 to 71% depending on the studied cohort of patients admitted to critical care units. In comparison, ACS developed in 3–12% of admissions. Moreover, IAH was a significant and independent predictor for short- and long-term mortality and was significantly associated with severe organ failures and prolonged stay in hospital [6–8]. Depending on the study, ICU mortality was considerably higher in IAH and ACS group compared to normal IAP, and the grade of IAH was inversely related to outcome. According to the study conducted by Smit *et al*, both intensive care and 90-day mortality were higher in IAH (4.8% and 15.2%, respectively) and ACS (16.7% and 38.9%, respectively) in

comparison to patients with normal IAP (1.2% and 7.1%, respectively) [6]. In addition, mechanical ventilation with positive pressure, obesity, sepsis, positive fluid balance, acute respiratory distress syndrome and anemia were indicated repetitively as risk factors of IAH development [9].

PATHOPHYSIOLOGY

The complicated pathophysiology of raised IAP remains poorly understood. The deteriorated function of organs has been observed for years, but this complex network of interactions is now being rediscovered (Table 1).

The abdominopelvic cavity is the body chamber defined by rigid bone structures and soft tissue. A slow increase in internal pressure causes the walls to expand, but their abilities to compensate are limited. When they are exceeded, a force applied to the diaphragm causes its cephalic shift, first described in 1922. This movement results in a reduced volume of the thoracic cavity and changed distribution of pressures. This concept called abdominal compliance (AC) is defined as a measure of the capability of abdominal expansion, which depends on the elasticity of the abdomen and diaphragm and can be expressed as a change in intra-abdominal volume per change in intra-abdominal pressure. AC depends on both these components and its dynamic of changes over time is thus hard to evaluate AC directly. Nevertheless, it should be considered an important component of intra-abdominal pressure, playing a significant role in organ interactions and end-organ perfusion. Understanding AC helps in applying the right management strategies and preventing the consequences of increased IAP. Particularly patients with IAH and reduced AC can benefit even from a small reduction in IAV [10-11].

Reduction of thorax volume caused by deviation of the diaphragm is accompanied by elevation of the thoracic pressure, which declines the preload and thus cardiac output [12-14]. Such alterations were observed already at pressures of 10 mmHg in animal models [15]. The unfavorable effect can be aggravated additionally by hypovolemia. A rise in IAP followed by higher intrathoracic pressure results in impairment of ventricular compliance and compression of systemic and pulmonary arteries, increasing

afterload [16]. A combination of these factors affects the venous system. Reduction in venous return can be observed due to increased pressure in the vena cava, proportional to the increase in pressure in the abdominal cavity [12]. Data suggest that fluid resuscitation improves venous return and thus diminishes the negative effect of increased afterload [16]. Furthermore, patients are at risk of deep venous thrombosis because of a rise in hydrostatic pressure throughout veins and the formation of peripheral edemas.

Compression of pulmonary tissue results in higher intrapulmonary shunt fraction; therefore, it may contribute to the development of respiratory failure. This effect begins with an IAP >16 mmHg. Alterations in dynamic and static spirometry variables are observed. Decrease in vital capacity, residual capacity and increase in pleural, inspiratory and peak pressure are prevalent [17].

Deterioration of alveolar blood flow, ventilation-perfusion mismatch and atelectasis are presented with hypercarbia and hypoxemia in arterial blood samples and frequently accompanied by infective complications [18].

In the state of elevated IAP, renal function should be considered at all times during therapy. The pressure determining the proper blood flow through the abdominal organs is so-called abdominal perfusion pressure (APP). It is calculated using the mean arterial pressure (MAP) and IAP. APP results from the difference between these two variables.

The organ most exposed to changes in IAP are the kidneys. The derivative parameter from APP that refers directly to kidney function is called the renal filtration gradient (RFG). The RFG, assuming a pressure in the proximal tubule is equal to IAP, can be calculated from the formula $RFG = MAP - 2 \times IAP$. Acute kidney injury induced by declined renal blood flow, venous congestion and parenchymal compression is a common finding among all patients with IAH [19-23].

As the response renin-angiotensin-aldosterone (RAA) axis is chronically activated and an increase in antidiuretic hormone concentration can be observed [24]. A large decrease in urine sodium concentration is demonstrated, which contributes to the development

of diuretic resistance, further disturbing the fluid balance ^[25-26]. The effect of IAH on the kidney is highly dependent on pressure value. With IAP between 15 and 20 mmHg, reduced renal filtration pressure leads to oliguria (2–5 mL/kg/h of diuresis) followed by the development of anuria at 30 mmHg. Also, an IAP of 20 mmHg causes an increase in renal vascular resistance by 500% and 1500% at 40 mmHg ^[27]. These changes shunt blood from the kidney, start glomerular necrosis, cause tubular damage and progress towards renal insufficiency ^[19].

The influence of IAP on the gastrointestinal tract cannot be ignored. Reduced blood flow combined with compression leads to venous hypertension in the visceral vascular system causing edema in the intestinal tissue. Along with it, the pressure in the abdominal cavity also increases, promoting further ischemic changes, malabsorption syndrome, bacterial flora translocation and metabolic acidosis, finally increasing mortality. A specific scale has been developed to predict mortality caused by intestinal dysfunction in patients with IAH ^[28]. Hepatic complications are common as the liver is sensitive to changes in abdominal pressure. In conditions of IAH, both hepatic blood vessel and portal vein blood flow is decreased. As a response to congestion, hepatocellular injury occurs ^[29]. Compromised mitochondrial function results in altered glucose metabolism and increased lactic acid concentration. This effect has been observed at IAP of only 10 mmHg, with preserved normal mean arterial blood pressure and cardiac output ^[13].

Venous blood return from the cerebrum affected by elevated pressure in the abdomen and thorax cause an increase of intracranial venous blood volume, rise in intracranial pressure and decrease in cerebral perfusion pressure ^[12]. These factors lead to cerebral edema and worsening of neurological functions ^[30-31].

DIAGNOSIS

Physical examination has been proven unreliable and imprecise in IAH diagnosis ^[32-34]. Since monitoring IAP is of fundamental importance for profiling patients and guiding therapy, different methods of its assessment have been proposed. The first attempts to measure intra-abdominal pressure were associated with an invasive

peritoneal puncture procedure and direct measurement of IAP using a peritoneal dialysis catheter. In the past, other methods like intramuscular continuous automatic piezoresistive pressure reading were confirmed as precise and consistent with non-direct measurements in porcine models. However, it has not been evaluated for safety and reliability in the ICU environment. Therefore, other, less invasive methods have been developed as an alternative. IAP can be estimated by recording bladder pressure (using Foley catheter), stomach pressure (using nasogastric or gastrostomy tube), rectal pressure (using balloon catheter) or inferior vena cava pressure. Transurethral measurement gained special recognition among them because of its simplicity, reliability, constancy, and low cost. The bladder is a passive reservoir, which reflects IAP accurately [35]. In this technique, a Foley catheter is inserted into a vesicle and filled with 25 mL of saline, which guarantees the transfer of hydrostatic pressure between the bladder wall and the pressure measuring device. As a device, a simple standpipe or conventional electronic transducer can be used. The tip of the pressure tube is connected to the catheter to prevent air from entering the line and avoid damping records. (Figure 1). Recently, commercially produced measurement kits have been available on the market. WSACS recommends measuring IAP every 4 h in the complete supine position, without abdominal muscle activity and at end-expiration phase with transducer zeroed at the mid-axillary line level, at the level of the iliac crest, 30–60 s after installation of body temperature fluid to allow bladder detrusor muscle relaxation to reduce inaccuracy and ensure reproducibility of the measurement. While using standpipe, reading in cmH₂O should be converted to mmHg. Intravesical measurement is a gold standard for indirect measurement proposed by WSACS, although it is only suitable for intermittent use [36–37].

MEDICAL MANAGEMENT

The goal of the treatment is to reduce IAP, restore regular systemic perfusion, reduce risk and avoid permanent end-organ damage. To achieve it, WSACS recommends a strategy that consists of improving abdominal wall compliance, evacuating intraluminal contents and excessive abdominal fluid collections, correcting positive fluid

balance and supporting organs. ⁴ However, it is essential to remember that clinical judgment and physical examination are far from accurate estimating IAP. Therefore, the ACS management algorithm has been developed to help clinicians in the process of decision-making. This integrated approach results in the improved outcome and higher economic efficiency.

As soon as IAH is diagnosed, actions should be taken to lower the pressure inside the abdominal cavity. Underlying etiology has to be identified and treated. To improve wall compliance, sedation and neuromuscular blockade can be initiated. If possible, the head of the bed angle should be under 30 degrees. And intra-luminal contents can be removed by nasogastric or rectal decompression, and prokinetic drugs can be applied. Invasive procedures like paracentesis or percutaneous drainage are recommended for evacuating residual fluid from the abdomen. The options of correcting the fluid balance include diuretics, colloids or hypertonic fluids, hemodialysis and ultrafiltration. If all of the above have failed, surgical intervention might be indicated. Abdominal decompression technique called open abdomen (OA) therapy is proven to enhance renal function and urine output, improve cardiac index and lung compliance [38]. These changes have major repercussions on hemodynamics. Lowered CVP and higher tidal volume are observed, which physicians must be prepared for and adjust resuscitation as well as parameters of mechanical ventilation appropriately. The intensivist should optimize the process, aiming for early fascial closure to avoid delayed extubation, development of fistulas and septic complications of extended OA therapy. Hypothermia, coagulopathy and acidosis are general concerns that should be addressed. Choosing optimal therapy requires a personalized approach. After implementing appropriate treatment, IAP should be monitored with serial assessments every 4 h (Figures 3 and 4).

CONCLUSION

Elevated IAP is a common, life-threatening condition associated with increased morbidity and mortality. Unrecognized IAH quickly leads to the development of

multiple organ failure; therefore, high alertness is necessary in relation to high-risk patients. Monitoring bladder pressure in this group allows early recognition of IAH/ACS and, consequently, timely intervention, which can have a spectacular effect and fully restore homeostasis. Although the progress made in recent years is significant, there are still open questions, leaving room for future research.

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