REVIEW

INTRA-ABDOMINAL HYPERTENSION: EVOLUTION AND CURRENT DEVELOPMENTS

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Abstract

Starting with simple empirical observations and continuing with studies on animals and humans, the medical knowledge of IAH/ACS has been gradually enriched. The subtlety of the signs and symptoms caused by the increase in IAP requires the implementation of a simple and effective and also adaptable method of measuring this parameter as well as careful monitoring of the patients at risk. Moreover, technological advances are oriented to measuring IAP and managing IAH by less invasive and less expensive methods, prophylaxis of this syndrome and prevention of its complications being the ones that prevail. For the purpose of exposing the most relevant historical aspects as well as the most recent developments, we have chosen studies between 1947 and 2017.This review aims at presenting some of the key moments in the evolution of understanding of the consequences of increased IAP, to defining current diagnostic and treatment protocols.

Keywords*: intraabdominal hypertension, measurement of abdominal pressure*

Introduction

The persistent increase over a certain level of the pressure in the abdominal cavity, seen as closed anatomical space, causes intraabdominal hypertension (IAH), which is the precursor stage in the development of abdominal compartment syndrome (ACS). This phenomenon results in impairment of circulation and tissue perfusion and, in the absence of any treatment, the evolution is towards organ failure and death.

The effects of the increased intraabdominal pressure (IAP) on the central nervous system, cardiovascular, renal, respiratory and digestive apparatus and the frequency of this type of complication in critically ill patients have prompted clinicians to develop protocols for effective prevention, diagnosis, treatment and hindering the possible evolution towards ACS. This review presents some of the key moments in the evolution of IAH, from understanding the consequences of increased IAP, to defining current diagnostic and treatment protocols.

Materials and method

For the purpose of exposing the most relevant historical aspects as well as the most recent developments, we have chosen studies

between 1947 and 2017. An extensive search was performed on pubmed database using the following keywords: "intra-abdominal pressure", "intra-abdominal hypertension", "intra-abdominal hypertension treatment".

Discussions

Since 1863, Marey has noticed an association between increased abdominal volume accompanied by increased IAP and increased respiratory frequency, seen as "breath stimulation"[1]. The first rectal measurement of IAP was performed by Braune in 1865, and through the bladder by Oderbrecht in 1875. The existence of a link between IAH and oliguria was first reported in 1876 [2]. The consequences of IAP growth were originally studied in animals and thus, in 1890, Heinricius showed that an increase from 27 to 46 mmHg of IAP determined their death and he associated a number of possible causes such as respiratory depression, hypotension or decreased cardiac diastolic distension [3]. Later in 1911, Esmerson [4] concluded that the main factor responsible for the decrease of the respiratory volume is the reduction in the diaphragm movement. Effects on the cardiovascular system, such as increased peripheral vascular resistance and even death by heart failure following a high IAP, have been noted. The author also provides recommendations regarding some therapeutic approaches such as reducing cardiac effort by removing ascites fluid. Splanchnic and peripheral circulation changes were later studied in 1939 by Bellis and Wangenstten. In 1947, Stanley and Geraldine Bradley detailed the negative effects of IAH on renal function in humans [5].

The unfavorable outcome of patients developing post-operative IAH was first suggested by an anesthesiologist from Dublin. He stated that the air that infiltrates the abdominal cavity by organs and instruments manipulation is responsible for the wound dehiscence and the impossibility of restoring the abdominal wall continuity. He draws attention to some studies conducted to that date showing that inadequate, forced abdominal wall closure (as is the case of patients known for many years with large umbilical hernia) is associated with a high mortality rate several days postoperative [6]. Although these observations were made early, clinicians focused their attention on this topic only after 1980 when they started measuring postoperative IAP and depending on its value, eventual abdominal decompression was attempted [7].

The most practical and efficient method for estimating IAP used today has been implemented by Kron et al. in 1984 and involves bladder pressure measurement after introducing a volume of 50-100 ml of liquid. The authors demonstrated that pressures recorded simultaneously by a transurethral catheter in the bladder and by a peritoneal dialysis catheter were equal over a range of 5 to 50 mmHg during the dialysis solution infusion [7]. The accuracy of the method was subsequently confirmed by direct measurement of IAP through laparoscopy. Although many authors consider Kron's work from 1984 to be decisive in defining ACS, he does not refer strictly to this notion. He brings instead solid data showing that abdomen re-opening in case of IAP greater than 30 mmHg can save the patient's life [7]. Subsequently, it has been demonstrated that insertion of a volume greater than 50 mL of saline into the bladder causes inaccuracy in the estimation of IAP [8]. Regarding this aspect, the most recent WSACS (World Society of the Abdominal Compartment Syndrome) recommendation mentions a maximum of 25 mL of fluid [9]. Fietsam et al. were the first to use the abdominal compartment syndrome notion to describe the alterations occurring in IAH caused by aortic aneurysm. In the four cases of IAP growth following the rupture of an abdominal aortic aneurysm, increased ventilation pressures and central venous pressure and decreased diuresis were observed. Thus, the authors suggest that ACS may be caused by interstitial and retroperitoneal edema, emphasizing that for volemic resuscitation more than 25 liters of fluids were used and that a significant improvement in evolution has been achieved following the abdominal decompression incision [10].

IAP can also be measured using the nasogastric tube or the gastrostomy tube. This method can be applied in patients to whom one cannot insert a Foley catheter, or when the value of bladder pressure might overestimate the IAP,

as is the case of bladder trauma, peritoneal adhesions, pelvic hematoma or fracture, neurogenic bladder and "abdominal packing" [11]. Similar to estimating IAP via the bladder, this technique has been improved over time [12]. Although it does not have clinical significance, IAP can be estimated by measuring both rectal [13] and intrauterine pressure [14].

Another method that allows continuous monitoring of IAP involves measuring the pressure in the femoral vein. Following a study that enrolled 149 patients, De Keulenaer et al. show that this continuous assessment manner becomes important in the prevention of ACS especially when IAP value exceeds 20 mmHg. Thus, the authors of this study recommend the use of the femoral venous catheter in patients at risk of developing IAH grade 3 or 4, but not as a replacement for the bladder estimation [15].

Technological progress makes it possible to imagine some less invasive as well as easier ways to determine IAP. Johan Van Stappen et al. describe the advantages of using a new system of simultaneous measurement of IAP and residual gastric volume [16]. Measurement of parietal tension is a non-invasive variant of IAP determination, initially studied on animals and post mortem patients and subsequently approached by Chen et al. in a study on 51 patients. The authors designed a device through which they monitored the tension in the abdominal wall. The obtained values correlated significantly with those of the bladder pressure and, although more extensive studies are needed in order to standardize the method and evaluate the factors that can influence the results, it can be considered a rapid, accurate and simple way of IAP assessment [17]. Another recent study has shown on pigs that IAP values obtained based on the principle of ultrasound guided tonometry could be significantly correlated with those obtained by bladder measurement. The method employs a pressure transducer in the form of a compressible chamber set at the tip of a linear ultrasound probe applied on the abdominal wall and subjected to various predetermined external pressure levels. Thus, the value of IAP was correlated with the vertical diameter of the pressure chamber [18].

It was found that IAH may occur not only in patients admitted to the intensive care unit, but also after elective surgery involving the abdominal wall reconstruction. In patients with large incisional hernias (with a diameter of more than 10 cm), the volume of the abdominal cavity is reduced due to organ protrusion in the hernia sac. Reducing the sac and repairing the parietal defect may lead to IAH and, subsequently ACS. In order to prevent these complications, Yao et al. have elaborated a study by which they attempted to correlate the ratio between the volume of the hernia sac and the abdominal cavity (obtained by 3D CT reconstruction) with the risk of postoperative development of ACS. Establishing this relation requires further studies, but the investigation of these features can provide valuable information on the management of patients with important abdominal wall defects [19].

Naturally, along with the evolution of methods that highlight IAP changes and their consequences, standardized protocols of patients presenting IAH have been developed. Over time, numerous variants of non-invasive treatment or adjuvant to the decompressive strategy have been evaluated in several studies. Bodnar et al. [20] observed a decrease in IAP parallel to the adenosine levels as well as a significant mortality reduction in patients with IAH who received theophylline versus those who received standard therapy. The same authors have shown that elevated levels of adenosine and interleukin 10 are the new laboratory markers of increased IAP. Horer et al. [21] conducted a study on 13 patients with ACS and multiple organ failure, demonstrating the efficiency of using tissue plasminogen activator to evacuate retroperitoneal hematomas. The fibrinolytic agent was administered via a catheter inserted under the computed tomography guidance in the retroperitoneal space. This method led to a decrease in IAP with an average of 8.5 mmHg, improved diuresis and hemodynamic parameters. It has also been shown that octreotide, melatonin and vitamin C have an important therapeutic effect if administered prior to abdominal decompression [22].

The increased interest in IAP as well as the changes in critically ill patient's management have recently brought progress in the field of IAH and ACS. The creation of WSACS brings consensus to defining and

diagnosis of IAH and ACS and establishes guidelines and recommendations on the management of this pathology with the aim of improving the survival of critical patients [9]. IAH and associated pathologies were extensively debated during several WSACS meetings, following which the definitions (in 2004 and 2006) [23] and the clinical approach (in 2007 and 2013) [9,24] were updated. The most recent guide in force was written in 2013 and was enriched by defining new terms and stating a series of recommendations (Table 1). WSACS proposed a step by step algorithm about the management of patients with an IAP over 12 mmHg (Table 2).

RETAINED DEFINITIONS FROM THE ORIGINAL 2006 CONSENSUS STATEMENTS	NEW DEFINITIONS ACCEPTED BY 2013 CONSENSUS PANEL
1. IAP is the steady-state pressure concealed within the abdominal cavity	**1. A polycompartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures**
2. The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25 mL of sterile saline	**2. Abdominal compliance is a measure of the ease of abdominal expansion, which is determined by the elasticity of the abdominal wall and diaphragm. It should be expressed as the change in intra-abdominal volume per change in IAP**
3. IAP should be expressed in mmHg and measured at endexpiration in the supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line	**3. The open abdomen is one that requires a temporary abdominal closure due to the skin and fascia not being closed after laparotomy**
4. IAP is approximately 5–7 mmHg in critically ill adults	**4. Lateralization of the abdominal wall is the phenomenon where the musculature and fascia of the abdominal wall, most exemplified by the rectus abdominus muscles and their enveloping fascia, move laterally away from the midline with time**
5. IAH is defined by a sustained or repeated pathological elevation in IAP C 12 mmHg	
6. ACS is defined as a sustained IAP[20 mmHg (with or without an APP\60 mmHg) that is associated with new organ dysfunction/failure	
7. IAH is graded as follows Grade I, IAP 12–15 mmHg Grade II, IAP 16–20 mmHg Grade III, IAP 21–25 mmHg Grade IV, IAP >25 mmHg	
8. Primary IAH or ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention	

9.	Secondary IAH or ACS refers to conditions that do not originate from the abdominopelvic region
10.	Recurrent IAH or ACS refers to the condition in which IAH or ACS redevelops following previous surgical or medical treatment of primary or secondary IAH or ACS
11.	APP = MAP – IAP

ACS-abdominal compartment syndrome, APP-abdominal perfusion pressure, IAH-intraabdominal hypertension, IAP-intraabdominal pressure, MAP -mean arterial pressure

Table 1 - Latest WSSCA updates set by the 2013 consensus [9]

	Evacuate intraluminal contents	**Evacuate intraabdominal space occupying lesions**	**Improve abdominal wall compliance**	**Optimize fluid administration**	**Optimize systemic/regional perfusion**
STEP 1	Insert nasogastric and/or rectal tube	Abdominal ultrasound to identify lesions	Ensure adequate sedation and analgesia (GRADE 1 D)	Avoid excessive fluid resuscitation (GRADE 2C)	Goal-directed fluid resuscitation
	Initiate gastro-/colo- prokinetic agents (GRADE 2D)		Remove constrictive dressings, abdominal eschars	Aim for zero to negative fluid balance by day 3 (GRADE 2C)	
STEP 2	Minimize enteral nutrition	Abdominal computed tomogrphy to identify lesions	Consider reverse Trendelenburg position	Resuscitate using hypertonic fluids, colloids	Hemodynamic monitoring to guide resuscitation
	Administer enemas (GRADE 1D)	Percutaneous catheter drainage (GRADE 2C)		Fluid removal through judicious diuresis once stable	
STEP 3	Consider colonoscopic decompression (GRADE 1D)	Consider surgical evacuation of lesions (GRADE 1D)	Consider neuromuscular blockade (GRADE 1D)	Consider hemodialysis/ ultrafiltration	
	Discontinue enteral nutrition				
STEP 4	If IAP>25 mmHg and new organ dysfunction / failure is present, patient's IAH/ACS is refractory to medical management. Strongly consider surgical abdominal decompression (GRADE 1D)				

Table 2. Management algorythm proposed by WSACS (After WSACS guidelines from www.wsacs.org

Conclusions

Starting with simple empirical observations and continuing with studies on animals and humans, the medical knowledge of IAH/ACS has been gradually enriched. The interest in this pathology is justified by the associated high morbidity and mortality and the high frequency occurrence, not restricted to critically ill patients. The subtlety of the signs and symptoms caused by the increase in IAP requires the implementation of a simple,

effective and also adaptable method of measuring this parameter as well as careful monitoring of the patients at risk. Moreover, technological advances are oriented to measuring IAP and managing IAH by less invasive and less expensive methods, prophylaxis of this syndrome and prevention of its complications being the most important aspects.

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