

Abdominal Compartment Syndrome as a Multidisciplinary Challenge. A Literature Review

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ABSTRACT

Abdominal Compartment Syndrome (ACS), despite recent advances in medical and surgical care, is a significant cause of mortality. The purpose of this review is to present the main diagnostic and therapeutic aspects from the anesthesiological and surgical points of view. Intra-abdominal hypertension may be diagnosed by measuring intra-abdominal pressure and indirectly by imaging and radiological means. Early detection of ACS is a key element in the ACS therapy. Without treatment, more than 90% of cases lead to death and according with the last reports, despite all treatment measures, the mortality rate is reported as being between 25 and 75%. There are conflicting reports as to the importance of a conservative therapy approach, although such an approach is the central to treatment guidelines of the World Society of Abdominal Compartment Syndrome, Decompressive laparotomy, although a backup solution in ACS therapy, reduces mortality by 16-37%. The open abdomen management has several variants, but negative pressure wound therapy represents the gold standard of surgical treatment.

Keywords: abdominal compartment syndrome, intra-abdominal pressure, decompressive laparotomy

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INTRODUCTION

Since the early 2000s, Abdominal Compartment Syndrome (ACS) has been accepted as a well-defined clinical entity. Monitoring of intra-abdominal pressure (IAP) represents a necessity, particularly in critically ill patients in intensive care units [1]. Moreover, knowing the risk factors that could lead to an increase in intra-abdominal pressure and progression to ACS, IAP monitoring has made it possible to detect early signs of intra-abdominal hypertension (IAH) in patients being treated in intensive care, surgery, internal medicine and cardiology departments. Besides objective methods of measuring intra-abdominal pressure, detailed anamnesis as well as communication skills with the patients is an important factor in identifying possible risk factors [2]. Early detection of ACS is key to obtaining the best results in treating the syndrome [3]. In addition to IAP measuring, imaging methods such as ultrasound and computer tomography aid in flagging-up characteristic signs that may suggest an increase in IAP [4].

ACS treatment, as recommended by the World Society of Abdominal Compartment Syndrome (WSACS), has undergone several changes over time, and since 2006 both conservative therapy and surgical treatment have been added to their guidelines.

This review reports on the multidisciplinary approach to ACS, from intensive care and surgical perspectives.

The reviews aim is to identify gaps in knowledge and suggested guidelines regarding the diagnosis and treatment of ACS analyzing the results of published studies.

DEFINITIONS

In 2006, the WSACS published a series of definitions they are still valid today [5]. Within these definitions, ACS is defined as an elevated IAP of more than 25 mmHg associated with new organ failure. Trigger factors, located inside of the abdominal cavity, induce a primary ACS, whereas trigger factors, out with the

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abdomino-pelvic cavity, contribute to the development of a secondary ACS. When abdominal scars and adhesions exist, the IAP varies in different parts of peritoneal cavity, resulting in what is described as polyACS.

In addition to well-established definitions, there are a number of recommendations and suggestions that have not been firmly endorsed, and further studies are required to accurately determine the level of implementation. They have been classified according to recommendation or suggestion in grades A to D; A - strongly recommended, D-poorly recommended, with subcategories occurring in each major grouping [5].

■ RISK FACTORS

A number of risk factors for the onset of ACS have been identified from studies, mainly performed in intensive care units. Thus, for primary ACS, the predominant predisposing factors are peritonitis, pancreatitis and abdominal trauma, and for secondary ACS, extra-abdominal sepsis is the major contributing factor (Table I) [5-10].

■ PATHOPHYSIOLOGY

The mechanisms of increased IAP are tissue edema, bowel and mesenteric edema, retroperitoneal space edema, and ascites after capillary leakage. IAH will lead to capillary compression which leads to intestinal ischemia [3,11]. Cardiovascular function is severely affected by the decreased preload due to inferior vena cava compression, which will lead to ischemia and hypoxia in all tissues [11,12]. A high position of the diaphragm and a decreased compliance of the thoracic wall will induce hypoxia, increased pleural pressure and accumulation of intrapleural fluid [13]. Compres-

sion on the inferior vena cava and renal veins leads to alterations of the glomerular filtration rate [11,12]. Liver function is also severely affected by ischemia, which will activate the Kupffer cells and release inflammatory mediators acting on hepatocytes and sinusoidal cells [14-17]. In cases of abdominal trauma and ruptured aortic aneurisms, the high pressure from affected vessels will rapidly lead to the accumulation of blood inside of peritoneal cavity, and a rapid progression to ACS due to the rapid pressure changes of IAP [18,19].

■ DIAGNOSIS

Measurement of IAP was recommended after recognition of severe effects of increased IAP and due to a low sensitivity of the whole series of clinical examinations tests [20]. The main variants of IAP measurement are presented in Table II [21].

The direct method is the most sensitive in determining PIA values. It is an invasive one, not without complications [22]. Laparoscopy allows the direct recording of IAP throughout the surgical procedure and allows the pneumoperitoneum pressure to be controlled. Studies related to the negative impact of intra-abdominal hypertension on body systems have been correlated with establishing the standard value of pneumoperitoneum pressure commonly used in laparoscopic interventions at 12 mmHg - capillary perfusion pressure [23].

The gold standard of IAP's indirect measurement is the monitoring of intravesical pressure, which is currently the most commonly used method [24,25]. There are several methods of measuring IAP by a transvesical approach. The first to be used was that described by Kron (1984) [22] in which the bladder wall was considered to be acting as a membrane pressure transducer. After the intravesical injection of 20 ml saline, the

Table I. Risk factors for the occurrence of primary and secondary ACS

Primary ACS	Secondary ACS
Severe intra-abdominal infection	Sepsis
Pancreatitis	Large-volume fluid replacement
Blunt/penetrating trauma	Burns
Ruptured abdominal aortic aneurysm	Dialysis
Postoperative bleeding	Obesity
Retroperitoneal hemorrhage	
Postoperative closure of the abdomen	
Undertension	
Ascites	
Ileus	
Pregnancy	

IAP is measured by a needle connected to a pressure manometer [26]. Based on this technique, the continuous monitoring system of the IAP was developed. An easier but not so precise technique is the Harahill method, where the IAP values are read from a scale following the same principles as PVC measurement. The column of fluid elevated in a tube perpendicular to the body above the pubic symphysis and connected to the uretrovesical catheter represents the IAP value. In later studies it was concluded that the urinary bladder wall acts as a pressure transducer only when it contains 25 ml of liquid. Continuous monitoring of IAP using the transvesical approach involves the use of a three-way uretrovesical catheter, irrigating the urinary bladder continuously with a steady flow of 25 ml saline solution. The transducer between the urinary catheter and a monitor, displays real-time intravesical pressure [27].

Although theoretical use of the transurethral approach pathway could lead to urinary tract infections, Cheatman (2006) reported on 3108 critical patients of whom 122 had their IAP measured by a transvesical approach. It was found that the technique itself does not lead to the development of urinary infections in a higher proportion to those resulting from the simple catheterization of the bladder [28].

Early detection of ACS by imaging methods has also been reported as being satisfactory. Cavaliere et al. (2011) simulated intraabdominal hypertension in a group of sixteen healthy volunteers and evaluated ultrasonographically the size of the inferior vena cava vein, the flow through it and renal circulation. They concluded that IAH simulation was associated with decreased inferior vena cava cross-section area and an increased resistive index in renal arteries [29]. Pereira et al. (2017) reporting on a group of fifty critically ill patients who developed IAH, showed that the point of care ultrasound (POCUS) proved to be extremely useful in evaluation of bowel activity, identification of large intestinal contents, the identification of patients who would benefit from bowel evacuation as an adjuvant to lower IAP and the diagnosis of moderate to large amounts of free intra-abdominal fluid [30]. Echocardiography

can detect indirect but non-specific signs of ACS, decreased preload as well as dysfunctions of systolic and diastolic ventricular functions [31]. Ignarra et al. (2011), after having performed a CT contrast scanning in fifty patients with IAH, observed some specific signs of elevated intraabdominal pressure. In eight patients there was elevation of the diaphragm, in five patients there was the “round belly sign”, in seven patients there was the presence of free fluid and air in the intraperitoneal and retroperitoneal spaces, in six patients there was collapse of the inferior vena cava, in four patients there was hyperenhancement and thickening of the intestine bowel wall, in another twenty, an elevated hepatic artery resistance index Color-Doppler with reversed diastolic flow [4].

■ TREATMENT

Conservative treatment

ACS conservative therapy should follow the WSACS Guidelines and must be initiated as soon as possible [32]. Reports on the incidence of ACS mortality indicate that without treatment the mortality is higher than 90%, and despite the administration of therapeutic measures, the mortality is between 25 and 75% [33]. The most important therapeutic measures are sedation, 0 or negative balance fluid resuscitation, nasogastric and rectal probe and neuromuscular blockade.

Surgical treatment

Decompressive laparotomy (DL) is of particular importance in the ACS, reducing the mortality by between 16% and 37% [11] (Table III).

An “open abdomen” is a fraught and difficult solution in the treatment of ACS, both in terms of the temporary and the permanent closure. DL is part of the ACS treatment algorithm established by WSACS. [32] However, DL is rarely performed as conservative treatment shows good results in a proportion of patients developing ACS. In Cheatham’s study (2011) on 265 patients with ACS, only sixty-two needed DL, and

Tabel II. IAP measurements methods

Direct IAP measurement methods	Indirect IAP measurement methods
Recording the values transmitted by an intraabdominal catheter	Urinary bladder pressure Intragastric pressure Pressure inside the colon Intrauterine pressure Inferior vena cava pressure

Table III. The ACS mortality rates quoted by different authors in the specific literature after decompressive laparotomy [11,34-37]

Author	Year	No. of patients	Study type	Mortality
J.J De Waale	2006	250	Retrospective	49.2%
J.J De Waale	2010	18	Retrospective	36%
Davis et al	2013	45	Prospective	24%
Divarci et al	2014	150	Prospective	16%
Hwabejire et al.	2015	122	Retrospective	37.7%
J.J De Waale	2016	33	Prospective	36%
Muresan et al	2016	66	Prospective	27.3%

thirty-one underwent percutaneous decompressive laparotomy (Table IV) [38].

The solutions preventing complications of open abdomen (OA) surgery are diverse and have developed concomitantly with the evolution of this new therapeutic concept. Among the many surgery variants for Temporary Abdominal Closure (TAC), only a few have been incorporated into standard medical practice and include closing the skin over the bowels and omentum with clamps, use of a Bogota bag and the Whitman technique [41-43]. The method of wound aspiration by creating negative pressure has been proven to have the best results, accomplishing several of the goals of TAC management. Based on the technique of Brock and Barker (1995) the development of dedicated vacuum therapy kits was introduced. [44,45] The TAC approach using negative pressure wound therapy (NPWT) technique conforms with 1B WSACS recommendation of OA management [32]. The final closure is related to the formerly used TAC. If visceral protection with epi-ploon can be performed, or if the granulation tissue is sufficiently well developed after NPWT, polypropylene meshes can be applied over granulated tissue. Safe al-

ternatives are dual meshes, sutured to the aponeurotic edges and applied over the viscera [46,47]. Biological materials offer a possible solution, but they are laborious and can give rise to postoperative complications due to graft necrosis. Modern cross-linked and non-cross-linked meshes, manufactured in the laboratory, are very expensive at present [48-50].

■ CONCLUSIONS

Despite the new therapeutic protocols recently introduced, ACS remains an entity resulting in a high mortality. Primary ACS occurs most often after the contamination of the abdominal cavity. Decompressive laparotomy is a necessary therapeutic solution in the complex treatment of ACS, improving the prognosis. DL aims both to release the intra-abdominal pressure, and to treat the underlying disease. The open abdomen management is based on eliminating secretions, protecting the viscera and avoiding lateral musculoaponeurotic retraction. Vacuum-assisted wound therapy encounters all above requirements (grade 1B) according with WSACS Guidelines of 2013.

Table IV. The main indications for open abdomen [33, 39, 40]

General condition	Open abdomen strong indication
Trauma	Prevention and treatment of IAH/ACS Need for a “second look” operation Post-injury septic abdomen Loss of abdominal wall
Abdominal sepsis	Peritonitis after perforations Peritonitis after anastomotic fistulas
Severe acute pancreatitis	Necrotizing pancreatitis Infected necrotizing pancreatitis Hemorrhagic necrotizing pancreatitis
Abdominal Compartment Syndrome (ACS)	Primary ACS Secondary ACS Recurrent ACS
Severe ileus	High fixation in frozen abdomen

■ ABBREVIATIONS

ACS: Abdominal Compartment Syndrome

IAP: Intra-Abdominal Pressure

IAH: Intra-Abdominal Hypertension

WSACS: World Society of Abdominal Compartment Syndrome

DL: Decompressive Laparotomy

OA: Open Abdomen

■ CONFLICT OF INTEREST

None to declare.

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