Abdominal Compartment Syndrome in Obese Patients

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12.1 Introduction

The peritoneal cavity can be considered a closed structure characterized by its own compliance and pressure. In obese patients the excess of intra-abdominal adipose tissue can interfere with many internal functions. Abdominal compartment syndrome is a clinical entity recognized in critically ill patients due to an abnormal increase of the intra-abdominal pressure causing severe organ complications and death. This syndrome requires specific medical and surgical treatments that can be effective in many, but not all, patients. Recently, a chronic abdominal compartment syndrome associated with morbid obesity has been identified.

12.2 Intra-Abdominal Pressure, Intra-Abdominal Hypertension, and Abdominal Compartment Syndrome: Background and Definitions

In normal-weight people the physiological value of intra-abdominal pressure (IAP) is 5–7 mmHg. This parameter is strictly related to several internal functions [1, 2].

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In fact, IAP is directly linked to the perfusion pressure of abdominal organs according to the formula:

$$\text{Abdominal perfusion pressure} = \text{Mean arterial pressure} - \text{IAP}$$

There is a close relation between abdominal pressure and renal function as follows [1, 2]:

$$\text{Glomerular filtration rate} = \text{Mean arterial pressure} - (\text{IAP} \times 2)$$

Elevated values of IAP cause a cephalad elevation of the diaphragm, reduce pulmonary gas exchanges, induce a rise in intracranial pressure and a decrease in venous return resulting in lower cardiac output [2]. IAP monitoring is essential in critical patients, since its variations can impair vital functions. However, the increase of IAP is not always connected to organ damage, because physiological mechanisms can balance its effect.

Intra-abdominal hypertension (IAH) is recognized when IAP exceeds 12 mmHg. It is graded as follows: Grade 1 = 12–15 mmHg, Grade 2 = 16–20 mmHg, Grade 3 = 21–25 mmHg and Grade 4 > 25 mmHg.

Abdominal compartment syndrome (ACS) occurs at IAH >20 mmHg when it is associated with one or more organ dysfunction. Usually renal function is compromised first, the kidney being more sensitive to ischemic damage.

Normally a transitional phase without organ impairment between IAH and ACS can be observed. Both IAH and ACS are usually underestimated: their frequency in critical patients varies from 30 to 60% for IAH and from 0.5 to 36% for ACS. The guidelines of the World Society of the Abdominal Compartment Syndrome (WSACS) were updated in 2013 [3, 4].

IAP monitoring is well standardized and provides valuable clinical information [5, 6].

The main risk factors for the development of IAH or ACS can be summarized as follows:

- diminished abdominal wall compliance (i.e., extensive burns, major trauma or prolonged prone position);
- increased intra-luminal content (i.e., mechanical or paralytic ileus);
- increased intra-abdominal content (i.e., hemoperitoneum, pneumoperitoneum or diffuse endoabdominal infections);
- capillary leak with accumulation of liquid in the third space and tissue edema (i.e., during acidosis, hypothermia, septic shock or after massive fluid resuscitation).

According to the classification of the WSACS, IAH and ACS can be divided in three groups:

- ***primary IAH/ACS***, due to diseases of the abdominal/pelvic area that often require emergency surgical/invasive treatments (diffuse peritonitis, hemoperitoneum, retroperitoneal hematomas and ascites);
- ***secondary IAH/ACS***, when the causes are outside the abdominal/pelvic compartment (excessive fluid resuscitation, massive transfusions, acidosis and hypothermia);
- ***recurrent IAH/ACS***, when the increase of intra-abdominal pressure is a direct consequence of a medical or surgical treatment of a primary or secondary ACS. This includes patients undergoing emergency laparotomies for diffuse peritonitis, in which the closure of the abdomen is followed by an ACS due to visceral edema, inflammation, and reduced compliance of the abdominal wall.

As underlined by Reintam et al., it is important to recognize the cause of IAH/ACS because it implies a high risk of mortality. In general, secondary IAH/ACS are more difficult to treat and present a worse prognosis, whereas in primary IAH/ACS a surgical treatment can be rapidly effective [7].

12.3 Principles of Treatment of Patients with IAH and ACS

In cases of IAH/ACS, the WSACS guidelines suggest medical and surgical interventions [4].

12.3.1 Medical Treatment

Since monitoring of the IAP represents the cornerstone for the early diagnosis of ACS, patients with IAH without organ damage must undergo medical treatment to reduce intra-abdominal pressure regardless of the primary or secondary cause (Table 12.1).

Table 12.1 Medical procedures that can counteract the pathophysiological mechanisms of IAH

To increase abdominal wall compliance	Sedation and analgesia Neuromuscular blockade Reverse Trendelenburg position
To reduce endoluminal content	Rectal and nasogastric decompression Prokinetic agents
To reduce endoabdominal fluids	Paracentesis Percutaneous drainage of abscesses
To obtain negative fluid balance	Fluid restriction Diuretics Colloids/hypertonic fluids Hemodialysis/ultrafiltration

IAH intra-abdominal hypertension

12.3.2 Surgical Treatment

The failure of a medical strategy and the appearance of organ damage demonstrate that the patient is developing a true ACS. Patients with established ACS may need either medical or surgical treatments, depending on the underlying pathology.

In primary ACS, the patient undergoes surgery with the dual purpose of decompressing the abdomen through a laparotomic approach and treating the etiological causes. At the end of the surgical procedure, the option to leave an open abdomen should be carefully considered.

In secondary ACS, the initial treatment must be based on the previously described medical strategies. In the case of failure of the medical approach, a laparotomic surgical approach must be considered to decompress the abdomen despite the absence of primary abdominal pathologies.

12.3.3 Open Abdomen

Open abdomen treatment represents a fundamental tool in the presence of secondary ACS refractory to medical approaches. It allows easier removal of primary or secondary causes of ACS up to resolution of the visceral damage.

Final closure of the abdominal wall can be delayed, but it should be done as quickly as possible to avoid the development of further severe complications (infections, enteroatmospheric fistulas, ventral hernias) [8]. When the abdomen is open, a temporary abdominal closure with a passive or active system must be considered [9]. Passive systems are cheap mechanical barriers (like the "Bogota bag" or "Wittmann patch") that protect the abdominal content; they can be used in the case of shortage of resources. An active system associates a suction device with a protective dressing for the so-called "negative pressure wound therapy" (NPWT) [10, 11] (Fig. 12.1). The negative pressure within the system contributes to remove exudates and maintain tension on the abdominal wall. Among the active systems,

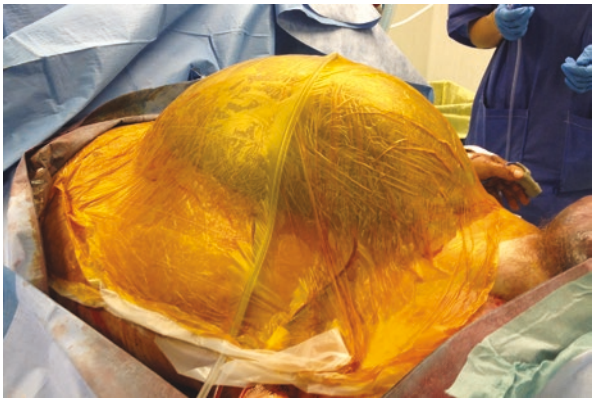

Fig. 12.1 Temporary abdominal closure with NPWT: a perforated barrier covers the viscera and a polyurethane sponge placed between the fascial edges. The wound is covered by an airtight seal and the sponge is connected to a suction drain. *NPWT* negative pressure wound therapy

further progress could be represented by the association of NPTW and a polypropylene mesh, sutured to the fascial margins, progressively cut and sutured to improve final closure of the abdomen because a prolonged delay provokes a severe fascial retraction [11].

12.4 Morbid Obesity and IAP, IAH and ACS

A direct correlation between the degree of obesity and elevated values of IAP is well known. In obese patients (BMI ≥ 30–35) baseline IAP is significantly higher than in the normal-weight population (9–12 mmHg) [11–14]. Central obesity (metabolic syndrome) with an increased sagittal abdominal diameter is associated with an increase in IAP [15]. The elevated chronic value of IAP in morbid obesity could depend on a direct mass effect from the intra-abdominal adipose tissue due to the fact that it is not a fluid and does not equalize pressure inside the abdominal cavity. The mass effect is more clearly detected when the abdominal fascia is intact [5]. In the immediate postoperative period, IAP is increased in obese patients undergoing either open or laparoscopic gastric bypass, most likely in relation to the surgical trauma and inflammatory response or a massive fluid administration [16].

The chronic elevation of IAP may be responsible for the pathogenesis of obesity-related comorbidities such as arterial hypertension, pseudotumor cerebri, pulmonary dysfunction, gastroesophageal reflux and abdominal wall hernias [5], hypertrophic cardiomyopathy, reduction of venous return, reduction of glomerular function: a clinical picture described as “chronic compartment syndrome” [17].

The question of whether the chronically elevated values of IAP observed in morbidly obese patients can promote a faster increase in IAH and a more rapid organ deterioration when ACS occurs has not been answered. The risk of developing an IAH and an ACS in obese patients is higher than in normal-weight subjects due to the fact that physiological compensation mechanisms, normally arising in the case of increased IAP, are already involved in maintaining homeostasis. Therefore, in the critically ill patient with morbid obesity, assessment of the IAP should be a part of common clinical practice in order to establish an early diagnosis of IAH and prevent ACS. Management of IAH is similar in obese and non-obese patients, but the treatment of ACS in the obese is more challenging and may be associated with higher complication rates [4].

12.5 Open Abdomen Treatment in Obese Patients for Abdominal Sepsis

Generalized peritonitis is a major cause of primary ACS especially in obese patients due to the severe peritoneal edema and the presence of intra-abdominal fluids in response to the inflammatory process or perforation of hollow organs. In these patients a surgical decompressive laparotomy represents the first-choice treatment.

The role of surgery is to control the source of the infection and to contribute to treat the cause of ACS. Open abdomen with temporary abdominal closure represents the ideal technique to cure ACS and avoid relapses while putting the surgeon in the condition to easily re-explore the abdomen and to control intra-abdominal infections.

The clinical criteria in favor of an open abdomen are not yet completely defined. Open abdomen treatment is recommended [18, 19] when:

- it is impossible to approach the fascia;
- IAP is above 15–20 mmHg after a partial closure of the abdomen with some stitches;
- after closure of the abdomen hemodynamic changes appear or high ventilation pressures are required;
- it is impossible to perform effective control of ACS with a single laparotomy.

Another indication for open abdomen is represented by obese patients when candidate to a procedure of damage control after trauma.

When an open abdomen treatment is planned in obese patients, it is desirable to use active temporary abdominal closure systems with NPWT. According to the more recent World Emergency Surgery Society guidelines [8], in the management of an acute abdomen due to sepsis, surgeons should follow these recommendations:

- multidisciplinary patient management with close cooperation among all the operators in order to agree on a common strategy; particular attention should be paid to maintain a slightly negative water balance, to correct acidosis, hypothermia and coagulopathy;
- since the patient with open abdomen is hypercatabolic, enteral or parenteral nutritional support becomes mandatory;
- re-explorations of the abdomen should be performed every 24–48 h in the case of clinical worsening of the patient.

Healing may be a very difficult target when enteroatmospheric fistulas are present in the open abdomen. Nutritional and hydroelectrolytic support associated with dynamic wound closure systems are essential. NPWT can be a valuable tool, especially when fistulas arise from the anterior aspect of the exposed viscera [20]. In these cases, through an appropriate use of drains/catheters/nipples or other specific devices, it is possible to exert a negative pressure within the abdomen after isolating the fistula properly. The delay of the final suture of the abdominal wall is mandatory.

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