Abdominal compartment syndrome and open abdomen management with negative pressure devices

Ann. Ital. Chir., 2015 86: 46-51
pii: S0003469X15023192

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BACKGROUND: *Abdominal compartment syndrome (ACS) is defined as an increase of intra-abdominal pressure (IAH) to values higher than 20 mmHg, associated with reduced perfusion and organ dysfunction.*
MATERIALS AND METHODS: *There is a classification of open abdomen which stratifies patients according to the natural history of improvement or clinical deterioration. The aim of treatment is to maintain the open abdomen at the lowest level and to prevent progression to a more complex level.*
DISCUSSION: *Surgical treatment essentially consists in abdominal decompression by leaving the abdomen open. Analysis of the literature shows that negative pressure increases the rate of primary fascial closure; entero-cutaneous fistulas are seen in a minority of cases, without seeming consequence of the application of the dressing. Open abdomen management consists of three treatment stages: acute (24-48 hours), intermediate (from 48 hours to 10 days) and late or reconstruction (from 10 days to the final closure).*
CONCLUSION: *It's important to recognize patients at risk of IAH and the first signs of ACS and intervene early with abdominal decompression if this will establish itself. Management of the open abdomen is now facilitated by negative pressure devices, which positively affect the morbidity and mortality of patients with ACS.*

KEY WORDS: Abdominal compartment syndrome, Negative pressure devices, Open abdomen management

Introduction

In literature, the first descriptions of open abdomen management go back to the 40s: Ogilvie described the use of a double sheet of light canvas or stout cotton cloth fastened with interrupted catgut sutures to muscle defects in abdominal war wounds that could not be closed primarily. Later, Ogilvie treated also cases of abdominal sepsis by placing vaselinate gauze over exposed abdominal viscera, and bringing togheter the edges of the wound with stitches or strips of Elastoplasts: the purpose of this procedure was draining the abdomen and saving abdominal wall in order to close it at a later time [1].
Subsequent studies on feasibility and indications of laparostomy appeared in the literature only forty years later. Actually, indications for temporary abdominal closure reported in literature are [2]:
– conditions under which abdominal wall can not be closed (necrotizing fasciitis, severe interstitial oedema);
– conditions in which the abdominal wall should not be closed (after aortic aneurysm surgery, need to re-exploration of abdominal cavity, for example for intra-abdominal infection in which the infectious outbreak is not properly controlled at first operation; intestinal ischemia;

Pervenuto in Redazione Luglio 2014. Accettato per la pubblicazione Ottobre 2014
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abdominal hypertension or surgeon operator suspicion of a possible development of compartmentimental syndrome, for example in conditions that require high volume of fluids infusion, such as shock).
Objective of open abdomen management is to prevent abdominal compartment syndrome and allow gradual wall closure.

ACS patophysiology

Abdominal compartment syndrome (ACS) is defined as an increase of intra-abdominal pressure (IAH) to values higher than 20 mmHg, associated with reduced perfusion and organ dysfunction [4]. Measurement of intra-abdominal pressure (IAP) is performed by a manometer connected to a Foley catheter placed into bladder, with the patient supine and relaxed at the end of expiration [5].
Abdominal perfusion pressure is represented by the difference between the mean arterial pressure and intra-abdominal pressure: it is considered normal if > than 60 mmHg.
Abdominal hypertension classification involves the distinction into four grades [6]:
– GRADE I: 12-15 mmHg
– GRADE II: 16-20 mmHg
– GRADE III: 21-25 mmHg
– GRADE IV: >25 mmHg
Causes of IAP increase are: spontaneous (peritonitis / intra-abdominal abscess, intestinal obstruction[8], rupture of an abdominal aortic aneurysm, pneumoperitoneum under tension, acute pancreatitis, mesenteric vein thrombosis, fecal bulk); post-operative (peritonitis, paralytic ileus, acute gastric dilatation, postoperative hemorrhage, visceral post-operative oedema); iatrogenic (abdominal packing, voluminous hernias reduction, abdominal forced closure, laparoscopy); burns; heart failure; space occupying lesions [9].

ACS onset [10]

In the first 12-36 hours after an increase of abdominal pressure, capillary leak of fluid into extravascular tissutal compartment determinates oedema of intestinal wall and mesentery. This determines a compensation mechanism (expanding the abdomen), which has limited efficacy, so a further seizure of liquids increases IAP exponentially. When IAP is > 12 mmHg (IAH) vascular, pulmonary, gastrointestinal, renal and nervous alterations are established: systemic vascular resistance are increased, pressure on the diaphragm makes breathing difficult, then occurres a reduction of intestinal perfusion with ischemia, a reduction of abdominal wall perfusion, a reduction of cardiac output and preload, a vena cava compression, a reduced urinary output, and a release of inflammatory cytokines. Once exceeded 15 mmHg, IAP increases exponentially. If intra-abdominal pressure (IAP) increases further, reaching values between 16 and 20 mmHg, occult organ ischemia (not clinically objectivizable) is determined, with increased intracranial pressure and decreased cerebral perfusion, further compression of the abdominal diaphragm with pulmonary dysfunction, edema and intestinal ischemia, increased central venous pressure and wedge pressure, further reducing cardiac output and increased vena cava compression, reduction of renal perfusion with oliguria, increased acidosis. Exceeded 20 mmHg, ACS occurs, with multiple organ dysfunction syndrome (MODS): onset of edema and cerebral ischemia, difficulty in ventilation and oxygenation up to ARDS (Acute Respiratory Distress Syndrome), intestinal wall ischemia and necrosis, cardiovascular instability , vena cava collapse, anuria and acute renal failure, severe acidosis. ACS mortality is 50%; its incidence among patients in RIA is 30-50% [11].

ACS treatments

ACS treatment includes medical and surgical treatment. Medical treatment involves abdominal compliance increase (sedation, analgesia, position, neuromuscular blocking drugs), the evacuation of intestinal contents (rectal and nasogastric decompression, prokinetic agents), evacuation of abdominal fluid collections (paracentesis, percutaneous drainage), correction of the partitioning of third space fluid and maintaining an adequate water balance (diuretics, colloids, hemodialysis, infection control), specific treatments (control of abdominal perfusion pressure). Surgical treatment essentially consists in abdominal decompression [12].
The optimal product for temporary abdominal closure after decompression should meet the following requirements: protect the contents of the peritoneal cavity by external agents; preserve abdominal wall integrity; ensure easy replacement and maintenance, allow to re-explore easily the abdominal cavity and have minimal adverse effects.
Over the past 30 years, several techniques have been proposed to obtain the temporary abdominal closure (bogota bag, Marlex zippers, Velcro strips, absorbable or non-absorbable mesh associated with gauze soaked in povidone-iodine) [13].
In 1995 Baker et al. [14] presented the first series of 28 patients treated with a negative pressure device, the vacuum-pack, which consisted in a fenestrated polyethylene sheet, placed between the abdominal viscera and the parietal peritoneum, above which was placed a wet laparotomic drape on which were placed two drainage at negative pressure. The entire wound and a large portion of the surrounding skin were covered with an adhesive drape. The positioning of the suction drains create the vacuum, approaching wound margins and making peritoneal

cavity drainage easier. This simple and economic technique, saves abdominal wall structures, facilitating the subsequent closure, but does not seem to be equally effective in preventing loss of domicile of abdominal viscera, with a rate of fascial closure of 52% in a Boele VanHensbroek systematic review [15].

To achieve an higher rate of fascial closure, vacuum pack has been modified as described by Garner et al. [16] and by Miller et al. [17]: vacuum-assisted fascial closure (VAFC) or vacuum-assisted wound closure (VAWC). In these early studies was used a polyurethane sponge placed on a polyethylene sheet and connected to a special suction pump. The method does not exert suction on abdominal wall and allows proximal and distal wound edges closure after each dressing change, by shaping a progressively smaller sponge. As reported in literature, this method allows a fascial closure rate in 65-100% of cases and reduces mortality rates and length of stay in intensive care units [18-24].

Stevens's review in 2009 [25] analyzes the results of a RCT, three prospective studies and some observational studies: literature shows that negative pressure increases analysis primary fascial closure rate; entero-cutaneous fistulas are rare without seeming consequence of the application of the dressing. In literature four primary mechanisms underlying treatment efficacy are described [26]:

– macrodeformation: skin and soft tissues have a natural tension under physiological conditions; when an incision is made, skin edges tend to recede. Keep wound edges closer accelerates tissue healing. The porous foam, when exposed to a -125 mmHg pressure, is reduced by 80%; it determines a wound contraction in three dimension, allowing an edges approaching;

– microdeformation: microscopic deformation of wound surface has been shown to stimulate cell proliferation and neo-angiogenesis in vivo. Indeed mechanical stress determines a physiological change in cellular function, refered to the action of mechano-transducers, a tipe of cytoskeletal molecules including integrins, that surface signals within the cell and modify genes transcription. Currently known vascular cells responses to mechanical stimuli include inhibition of apoptosis, up-regolation of signaling molecules, change of gene expression and increased proliferation;

– removal of excess fluid and oedema: negative pressure is able to absorb wound exudate. Oedema slows down the healing tissue by causing a cell compression that determinate a reduction of proliferative response. Negative pressure allows fluid evacuation from extracellular space and reduces edema;

– Control of wound environment: semi-occlusive drape has a limited permeability to gases and water vapor and is impermeable to proteins and microorganisms. Dressing with gauze, that allows fluids evaporation, determines a protein concentration on the wound bed, which can lead to deposit and fibrin, a consequent deceleration of healing process. Fluids evacuation, electrolytes and proteins exerted by negative pressure, seems to keep wound oncotic and osmotic gradient stable.

Secondary effects of these four mechanisms are formation of granulation tissue; increased cell proliferation; modulation of inflammation; neuropeptides regulation and change in bacterial levels. Abdominal negative pressure devices currently in use allow to set the pressure to applied to the abdominal cavity. Experts advise 125 mmHg for cases of peritonitis, -75 mmHg if the abdomen is decontaminated, -25/50 mmHg (never more than 75) in trauma / bleeding disorders. Open abdomen management consists of three treatment stages: acute (24-48 hours), intermediate (from 48 hours to 10 days) and late or reconstruction (from 10 days to the final closure). At each dressing change, the surgeon decides or not to close the abdominal wall according to: intra-abdominal pressure (must be <15 mmHg), tension exerted on fascia to close the wall, treatment of local infection, prevision of further surgeries.

Abdominal closure can occur gradually (to reduce the risk of fasciitis) or through the use of a thick biological prosthesis on which is placed a plastic cover and set the washing unter the skin. Abdominal wall must be closed after 24 hours . If you can not close all the layers of the abdominal wall, the attempt must regard at least the peritoneum, to reduce morbidity and mortality.

Abdominal wall closure must be made within 10-11 days: risk of fistulas increases after 8 days and immunosuppression onset after 14 days [27].

Caution is required in case of prolonged treatment (increased risk of fistulas) and in the presence of anastomoses (which must be protected from direct exposure, positioning in depth, not in contact with the intake system).

Open abdomen classification

In 2009 a classification of open abdomen was proposed: it stratifies patients according to natural history of improvement or clinical deterioration [28-30]. The aim of treatment is to maintain open abdomen at the lowest level and to prevent progression to a more complex level.

LEVEL 1a: Open abdomen clean without adhesions and / or stiffness:

This situation often occurs after decompressive laparotomy for ACS, ruptured abdominal aorta aneurysm or abdominal trauma not associated with bowel perforation. If patient has no other risk factors for an unsatisfactory outcome, the prognosis is good. Negative abdominal pressure therapy is indicate.

LEVEL 1b: Contaminated open abdomen without adhesions and / or stiffness:

The typical clinical picture includes patients with local or generalized peritonitis due to infection (diverticulitis, anastomotic dehiscence after colorectal surgery, trauma, gastrointestinal tract). The aim is to decontaminate the

abdomen and prevent stiffness or fascial lateralization.
LEVEL 2a: Clean open abdomen with adhesions and / or stiffness:
If adhesions between bowel wall have developed and / or the fascia is retracted laterally, primary closure fascial can be difficult to obtain. In this case, negative pressure therapy is indicated, by entering the protective layer of the device in the visceral parieto-colic gutter, to completely separate the viscera from contact with the abdominal wall and to ensure the drainage of exudate. The formation of adhesions can reduce the chances of fascial approximation and increase the risk of developing fistulas [31].
LEVEL 2b: Contaminated open abdomen with adhesions and / or stiffness:
In patients infective source control has not yet been obtained and where adhesions and / or stiffness may preclude a subsequent fascial closure, the main purpose is to control contamination and bring the patient to the second level without further deterioration. Is possible to apply abdominal negative pressure devices.
LEVEL 3: Open abdomen complicated by fistula. Development of fistula represents a serious deterioration, with significant impact on outcome. Control of sepsis, careful nutritional assessment and timing of surgery are important. The main purpose are fistula management and avoiding skin damage, fascia lateralization and loss of abdominal. If fistula can be controlled, the patient into stage 1 or 2 and then can be subjected to delayed fascial closure; in clinical practice, a switch from a level 3 to level 1 is rare. The prevention of adhesions formation is extremely important to avoid progression to the frozen abdomen (level 4). Only specialist units use negative pressure abdominal devices in patients with open abdomen at level 3.The purpose is to manage and isolate fistula outflow to prevent continuous abdominal contamination.
LEVEL 4: frozen open abdomen with bowel adherent / fixed, impossible to close surgically, with or without fistula. Patients should be addressed to a specialized center. In there is a fistula, the main purpose is to control it and optimize patient physiology protecting skin and fascia, as well as prevent sepsis. It may be possible a simple skin closure over the wound bed granulation / viscera or to schedule an incisional hernia. These strategies allow for the early patient discharge but are associated with occurrence of incisional hernias, which require reconstructive surgery.

Initial application and dressing changes

Before applying the medication, surgeon must identify necrosis, ischemia, infection or contamination. Application of negative pressure system and subsequent changes must be carried out under aseptic conditions. Recommended interval for dressing changing is 24-72 hours. More frequent dressing changes may be indicated in presence of infection or contamination.

Timing of final closure

Timing of closure is determined by clinical experience and several factors: hypothermia, acidosis, lactate, coagulation disorders and intra-abdominal hypertension are predictive factors of SCA development and represent a fascial closure controindication. However, these parameters are usually most crucial in the initial phase of open abdomen management.

Conclusion

IAH is a factor to be taken into account in chronic abdominal emergency. The problem may be obvious, clinically silent or have a trend, but contributes ACS and SIRS on set with organ dysfunction and death. It's very important, therefore, to recognize patients with an increased risk at of IAH to operate an early abdominal decompression [32]. Management of open abdomen is now facilitated by negative pressure devices, which positively affect the morbidity and mortality of patients with ACS.

Riassunto

Per sindrome compartimentale addominale (SCA) si intende un incremento della pressione intra-addominale (PIA) a valori superiori a 20 mmHg, associato ad una ridotta perfusione e disfunzione d'organo. La mortalità della SCA è del 50%; la sua incidenza tra i pazienti in RIA è del 30-50%. Il trattamento chirurgico consiste sostanzialmente nella decompressione addominale con apertura dell'addome; attualmente la metodica più utilizzata per la gestione della SCA è la terapia a pressione negativa. Nel 2009 è stata realizzata una classificazione dell'addome aperto, che stratifica i pazienti in base alla cronologia naturale del miglioramento o del deterioramento clinico dei pazienti con addome aperto. Lo scopo del trattamento è mantenere l'addome aperto del paziente al livello più basso ed evitare la progressione ad un livello più complesso. Secondo quanto riportato in letteratura questa metodica consente un tasso di chiusura fasciale tra i 65 e il 100% dei casi, riduce i tassi di mortalità e i tempi di degenza nei reparti di terapia intensiva. La chiusura dell'addome può avvenire progressivamente (per ridurre il rischio di fascite) oppure mediante l'utilizzo di una protesi biologia spessa su cui viene posizionata una copertura in plastica e impostato il lavaggio nel sottocute: la parete addominale deve essere chiusa dopo 24 ore. Se non fosse possibile chiudere tutti gli strati della parete addominale, il tentativo deve riguardare almeno il peritoneo, per ridurre morbilità e mortalità.

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