

Review

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ABDOMINAL COMPARTMENT SYNDROME IN BURN PATIENTS

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Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are consistently associated with morbidity and mortality among the critically ill or injured. Thus, avoiding or potentially treating these conditions may improve patient outcomes. Despite a large number of special publications devoted to this problem, very little attention is paid to the ACS in patients with severe burn injuries.

Severe burns have been shown to be a risk factor for developing IAH. Fluid resuscitation practices used in burns management further predispose patients to increase intra-abdominal pressure. The incidence of intra-abdominal hypertension in patients with severe thermal injury is, according to different authors, 57.8–82.6 %. The mortality associated with IAH in severe burns is very high once organ dysfunction occurs.

The purpose of this work is to collect and analyze the problem of abdominal hypertension in burn patients, as well as to draw conclusions on the prevention of this condition and improve the results of treatment of patients with severe burn injury.

KEY WORDS: Abdominal compartment syndrome, Intra-abdominal hypertension, Burns, Fluid resuscitation

АБДОМІНАЛЬНИЙ КОМПАРТМЕНТ СИНДРОМ У ОПІКОВИХ ХВОРИХ

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Внутрішньочеревна гіпертензія і абдомінальний компартмент синдром тісно пов'язані з захворюваністю і смертністю серед критично хворих і уражених. Уникаючи або проводячи адекватне лікування цих потенційно небезпечних для життя станів можна поліпшити результати лікування пацієнтів.

Незважаючи на досить велику кількість спеціальних публікацій, присвячених даній проблемі, дуже мало уваги приділяється абдомінальному компартмент синдрому у хворих з важкою термічною травмою.

У ряді досліджень показано, що важкі опіки є фактором ризику розвитку внутрішньочеревної гіпертензії. Великі обсяги інфузійної терапії, що використовуються при лікуванні важкої опікової травми, додатково привертають пацієнтів до збільшення внутрішньочеревного тиску. Частота розвитку інтраабдомінальної гіпертензії у хворих з тяжкою термічною травмою становить, за даними різних авторів 57,8–82,6 %. Летальність, пов'язана з внутрішньочеревної гіпертензією при великих опіках досить висока після виникнення поліорганної дисфункції.

Мета даної роботи – зібрати і проаналізувати проблему абдомінальної гіпертензії у опікових хворих, а також зробити висновки щодо профілактики даного стану і поліпшенню результатів лікування постраждалих з важкою термічною травмою.

КЛЮЧОВІ СЛОВА: абдомінальний компартмент синдром, інтраабдомінальна гіпертензія, опіки, інфузійна терапія

АБДОМИНАЛЬНЫЙ КОМПАРТМЕНТ СИНДРОМ У ОЖОГОВЫХ БОЛЬНЫХ

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Внутрибрюшная гипертензия и абдоминальный компартмент синдром тесно связаны с заболеваемостью и смертностью среди критически больных и пораженных. Избегая или проводя адекватное лечение этих потенциально опасных для жизни состояний можно улучшить результаты лечения пациентов.

Несмотря на достаточно большое количество специальных публикаций, посвященных данной проблеме, очень мало внимания уделяется абдоминальному компартмент синдрому у больных с тяжелой термической травмой.

В ряде исследований показано, что тяжелые ожоги являются фактором риска развития внутрибрюшной гипертензии. Большие объемы инфузионной терапии, используемые при лечении тяжелой ожоговой травмы, дополнительно предрасполагают пациентов к увеличению внутрибрюшного давления. Частота развития интраабдоминальной гипертензии у больных с тяжелой термической травмой составляет, по данным разных авторов 57,8–82,6 %. Летальность, связанная с внутрибрюшной гипертензией при обширных ожогах очень высокая после возникновения полиорганной дисфункции.

Цель данной работы – собрать и проанализировать проблему абдоминальной гипертензии у ожоговых больных, а также сделать выводы по профилактике данного состояния и улучшению результатов лечения пострадавших с тяжелой термической травмой.

КЛЮЧЕВЫЕ СЛОВА: абдоминальный компартмент синдром, интраабдоминальная гипертензия, ожоги, инфузионная терапия

Abdominal compartment syndrome (ACS) is a pathological condition in which organ dysfunction is the result of intra-abdominal hypertension (IAH). It is determined by a steady or repeated increase of intra-abdominal pressure (IAP) over 20 mm Hg. and/or abdominal perfusion pressure (APP) less than 60 mm Hg in combination with newly discovered dysfunction of one system or multiple organ failure [1].

Much good evidence now supports the concept that elevated IAP may impair physiology and organ function by producing the ACS. Complex, adverse physiological consequences of increased IAP develop as the pressure is transmitted to adjacent spaces and cavities, decreasing cardiac output, restricting pulmonary ventilation, diminishing renal function and visceral perfusion, and increasing cerebrospinal pressure [2].

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are associated with increased morbidity and mortality among multiple types of patient populations [3].

The World Society of the Abdominal Compartment Syndrome (WSACS) has published definitions and guidelines for the

diagnosis and management of patients with IAH and ACS [4].

Final 2013 consensus definitions of the World Society of the Abdominal Compartment Syndrome:

1. IAP is the steady-state pressure concealed within the abdominal cavity.
2. The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25 mL of sterile saline.
3. IAP should be expressed in mmHg and measured at end-expiration in the supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line.
4. IAP is approximately 5–7 mm Hg in critically ill adults.
5. IAH is defined by a sustained or repeated pathological elevation in IAP \geq 12 mm Hg.
6. ACS is defined as a sustained IAP $>$ 20 mm Hg (with or without an abdominal perfusion pressure (APP) $<$ 60 mm Hg) that is associated with new organ dysfunction/failure.
7. IAH is graded as follows:
Grade I – IAP 12–15 mm Hg;
Grade II – IAP 16–20 mm Hg;
Grade III – IAP 21–25 mm Hg;

Grade IV – IAP > 25 mm Hg.

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 (MAP – mean arterial pressure).

12. A polycompartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures.

13. 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.

14. The open abdomen is one that requires a temporary abdominal closure due to the skin and fascia not being closed after laparotomy.

15. 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 [4].

There are a lot of risk factors for intra-abdominal hypertension and abdominal compartment syndrome. Major burns are one of these factors [4].

It should be noted that an increase in IAP is not always accompanied by the occurrence of ACS. The regularity is known: the higher the IAP and the more factors leading to its increase, the more likely is the development of the ACS [1].

High abdominal pressures lead to several systemic impairments: cephalad movement of the diaphragm leads to cardiac and lung compression, reduced venous return and, subsequently, contributes to hypoxemia, hypercapnia, atelectasis and ventilation-perfusion mismatch. ACS will also compress renal vessels, activating sympathetic drive and the renin-angiotensin system; these effects contribute to a decrease in urine output. Primarily, renal vasoconstriction leads to a

significant decrease in urine output, and is typically the first indicator of the onset of ACS – oliguria is noted at IAPs > 15 mm Hg and anuria at IAPs of 30 mm Hg. Reports document a decrease in mesenteric blood flow at 10 mmHg IAP; intestinal mucosa perfusion decreases at 20 mm Hg IAP, and celiac and superior mesenteric artery flow is compromised at IAPs > 40 mm Hg. To further exacerbate the effects on gastric circulation, the increased pressure may compress mesenteric veins, impairing drainage and exacerbating ACS, ultimately leading to further gut hypoperfusion, ischemic bowel, decreased intramural pH and worsening lactic acidosis [5–8]. In the context of tissue injury consistent with severe burn trauma, inflammatory responses can also exacerbate an ischemic bowel. The inflammatory cytokines released will increase capillary permeability, leading to more edema and higher IAP [9]. This is a vicious cycle in which edema results in injury, which in turn worsens edema.

The generalized increase in capillary permeability that occurs in severe burn patients contributes to extensive edema formation and intra-peritoneal accumulation of «third-space» fluid [10].

Capillary leak and third spacing are universal in major burns. In patients with burns of more than 60 % of their body surface area and without abdominal pathology, the pathogenesis for increased IAP is most likely due to massive fluid resuscitation with third spacing and secondary extrinsic compression by burn eschars. «Capillary leak» following shock, with ischemia-reperfusion injury and the release of vasoactive substances and oxygen-derived free radicals increases extracellular volume. Especially when it occurs with associated inhalational injury, delayed resuscitation, and abdominal wall injuries [11–12].

Bowel edema and fluid translocation is further worsened by venous hypertension caused by elevated IAP [13]. This increasing volume in the abdominal cavity, however, is reduced after capillary permeability improves. Therefore, secondary IAH in burn patients generally occurs within 48 hours after injury, during the initial resuscitation period, while ACS usually occurs after the acute phase, during subsequent septic episodes [14–15]. Burn patients are also at risk of tertiary or recurrent ACS any time they require aggressive

resuscitation as, for instance, after any overly aggressive burn excision [10, 13].

IAH/ACS should be suspected in all patients with severe burns. The incidence of IAH in major burn patients is variable in the literature and is associated with the burn area. Patients with > 20 % TBSA burned presented a very high prevalence of IAH. Development of organ failure occurred even at moderately increased values of IAP. In this scenario, monitoring of IAP is the first step for establishing the importance of IAH/ACS in this patient population [16–17]. IAP measurement should therefore be performed every 2 to 4 hours throughout the resuscitation period in burn patients with more than 20 % TBSA [18].

The use of mechanical ventilation is also associated with an increased incidence of IAH and to a worse prognosis in untreated cases [19]. This risk factor is proportional to the severity of respiratory symptoms and the mechanical ventilation requirement.

Malbrain ML at al. [18] believes that IAH will develop in most (if not all) severely burned patients, and may contribute to early mortality [18]. A recent systematic review showed that the prevalence of ACS and IAH in severely burned patients is 4.1–16.6 % and 64.7–74.5 %, respectively [20]. The risk of ACS is higher in burned patients with a higher percentage of total body surface area (TBSA) burned; however, patients with a lower burned TBSA may develop IAH/ACS as well [15]. ACS typically occurs when resuscitation volumes are greater than 275 mL/kg during the first 24 hours or TBSA burned is larger than 60% [21–22]. Patients with severe burn injuries greater than 60 % of TBSA, associated inhalational injuries, delayed resuscitation, and intra-abdominal injuries are at the highest risk of developing IAH and ACS [23]. The mortality rate of patients developing ACS is 50–84 %, even when treated [18, 24].

The effects of IAH/ACS in patients with severe burns are multifactorial. Raised IAP can lead to organ dysfunction and can affect all organ systems. The use of excessive fluid resuscitation in combination with increased capillary permeability as a result of the systemic inflammatory response to burn injury makes these patients particularly vulnerable to the development of IAH and ACS and cardiovascular, respiratory, and renal system dysfunction [15]. In severe burn patients, the

kidneys are especially vulnerable to elevated IAP-related injury [25].

Talizin TB at al. evaluated the frequency of intra-abdominal hypertension in major burn patients and its association with the occurrence of acute kidney injury (AKI) [25]. A total of 46 patients were analyzed. Of these, 38 patients developed IAH (82.6 %), thirty-two patients (69.9 %) developed acute kidney injury. The median time to development of acute kidney injury was 3 days. The individual analysis of risk factors for acute kidney injury indicated an association with intra-abdominal hypertension, use of glycopeptides, use of vasopressors [25].

The use of nephrotoxic drugs, such as glycopeptides, is associated with direct kidney injury and the consequent dysfunction of this organ. Changing organic perfusion in the case of circulatory instability, as evidenced in the literature, is a risk factor for kidney injury [26]. The IAH patient also presents hemodynamic changes with impaired renal perfusion [20]. An association between AKI and higher 30-day mortality in intensive care patients has been found [25].

Since an elevated IAP affects renal blood flow, urinary output is an unreliable index of the preload and intravascular volume resulting in the loss of an important physiologic parameter.

Moreover, ACS as well as abdominal decompression for ACS increases susceptibility to multiple organ dysfunction syndrome (MODS) for severe burn patients and may also induce acute lung injury [18].

One should pay attention to the fact that IAH/ACS might occur in patients without circumferential 3rd degree burns of their trunk. Burn patients with smoke inhalation may also be at risk of fluid sequestration [21].

It is fundamental to: 1) recognize IAP and ACS; 2) resuscitate effectively; and 3) prevent the development IAP-induced end-organ dysfunction and failure [27].

The WSACS medical management algorithm for IAH/ACS is based on five treatment options: 1) evacuation of intraluminal contents; 2) evacuation of intra-abdominal space occupying lesions; 3) improvement of abdominal wall compliance; 4) optimization of fluid administration; 5) optimisation of systemic and regional perfusion [4].

According to WSACS recommendations if patient has IAP \geq 12 mm Hg medical

management to reduce IAP should be started. If IAP > 20 mm Hg and new organ dysfunction/failure is presented, patient's IAH/ACS is refractory to medical management. Strongly consider surgical abdominal decompression (GRADE 1D) [4].

But management of ACS with decompressive laparotomies is associated with significant morbidity and mortality ranging from 50 % to 100 % [28].

Thus, the main thing is the prevention of ACS. Key to the prevention of ACS is the early recognition and treatment of IAH [29–30].

Many burn physicians lack awareness of the deleterious effects of raised IAP and do not regularly measure it [29].

Resuscitation in the very first hours after a burn is a key point in the treatment of severe burn shock [31]. Judicious use of fluids and avoidance of fluid over-resuscitation is the key element in the prevention of secondary ACS. Moreover, the choice of resuscitation fluid among critically ill patients with burns may have a clinical importance [22, 32].

There is no perfect resuscitation protocol and studies have demonstrated that patients frequently receive larger amounts of fluids than required a patient. This condition recently recognized as «fluid creep», a phenomenon which may also be attributed to «opioid creep».

Fluid creep is an iatrogenic phenomenon resulting from misuse of the originally described approaches to crystalloid resuscitation. It is associated with massive edema and compartment syndromes (orbital, abdominal, and extremity compartment syndrome) [18, 33–35].

It is currently unknown whether the syndrome is an iatrogenic consequence of excessive fluid resuscitation or an unavoidable sequelae of the primary injury. A recent systematic review of severely burned patients concluded that the fluid resuscitation volume was directly responsible for the development of ACS. It exacerbates splanchnic edema leading to an increase in gut permeability, bacterial translocation, and increased intra-abdominal pressure. Resuscitation-related ACS is associated with a mortality of 97 % when burn size is greater than 60 % TBSA [12, 36–37].

Groups of burn patients that have been identified in whom resuscitation requirements are usually greater than the parkland Formula predictions include patients with inhalation injuries, electrical burns, those with additional

injuries, patients with high alcohol or drug intake, and those in whom resuscitation was delayed. To avoid «fluid creep», the resuscitation formulas have to be used only as indicators for the initial fluid resuscitation rate. This rate must be adjusted according to several parameters, the most important and most frequently used being urine output. According to a survey of the American Burn Association and the International Society of Burn Injuries, 94.9 % of respondents use urine output as the main indicator of successful infusion therapy [38]. This parameter should not be allowed to exceed the recommended hourly urine output range of 0.5 to 1 ml/kg/h [27]. But in overhydrated severely burned patients, a decreased urine output may reflect overresuscitation and the onset of abdominal compartment syndrome [35].

For patients with severe burn injury, it is necessary to strive to restore microcirculation in the shortest possible time, using the minimum amount of fluid necessary to maintain the physiological functions of the body. Both insufficient and excessive amount of injected fluid leads to the dysfunction of organs and tissues, the development of multiple organ failure (MOF).

Ivy has identified 250 ml/kg of volume administration within the first 24 hours as a risk factor for ACS [21]. Regular calculation of the Ivy index will identify patients at risk of developing ACS. However, in the eventuality of a thick abdominal eschar, abdominal distention is restricted, thus the critical point of increased IAP is reached with lesser increase in intra-abdominal volume and IAH and ACS may occur with lesser fluid resuscitation volumes [10, 39].

Now novel resuscitation strategies in burn patients to avoid IAH/ACS are evolving. Recent evidence supports the use of hypertonic sodium chloride solution and colloids enabling less overall fluid volume resuscitation. Despite efforts to minimize fluid administration many patients end up grossly fluid overloaded leading to IAH and ACS [22, 40].

Randomized studies have shown that hypertonic lactated saline (HLS) or plasma-based resuscitation requires less fluid and is associated with a lower risk of IAH and ACS. On the other hand, isotonic resuscitation was associated with a 3.5-fold increased risk for developing IAH. [22].

Treatment of burns with a hypertonic solution reduced the secretion of cytokines by cardiomyocytes, decreased their sensitivity to the action of lipopolysaccharides against cytokine secretion, and improved pumping function [41].

Some authors add colloids to their resuscitation regimen within the first 24h to reduce the total resuscitation volumes. However, this remains a debatable issue even though there is growing evidence of its usefulness. Despite some reservation concerning the use of albumin in the early phases of burn resuscitation, recent work demonstrated a decreased mortality rate.

The use of only salt solutions can be limited in cases where dehydration does not reach the stage of reducing the volume of circulating blood.

If dehydration progresses to the stage of intravascular space reduction, then early administration of colloids is necessary. And later, saline solutions can be assigned to rehydrate the interstitial space. It should be noted that dehydration of the vascular space occurs after interstitial dehydration, and the injected salt solutions will immediately move to the interstitial space before filling the vascular sector.

Low molecular weight dextrans (dextran 40), native plasma, hydroxyethyl starch 130/0.4 are recommended as colloids. Also, glucose and fructose solutions are included in the burn shock infusion therapy.

The ratio of colloids, crystalloids, salt-free drugs in patients with severe and extremely severe thermal injury is an average of 1: 1: 1, but is corrected according to the state of the particular patient. The order of their administration depends on the hemodynamic parameters, especially the central venous pressure [42].

There is also growing evidence that vitamin C supplementation, in the early post-burn period, seems to decrease the needed fluid volumes.

A pronounced inflammatory response in severe burn injury contributes to the release of free oxygen radicals, which further impair the microcirculation and contribute to the development of interstitial edema [43].

Oxidative tissue damage as assessed by increased myeloperoxidase (MPO) activity, lipid peroxidation, and decreased levels of glutathione levels in intestinal and hepatic

tissue plays an important role in progression from IAH to ACS. However, reperfusion of decompressed tissue induces a more prominent injury compared to ischaemia itself. Reperfusion promotes generation of various reactive oxygen metabolites via activated neutrophils that cause increased microvascular permeability, interstitial oedema, impaired vasoregulation, inflammatory cell infiltration, and parenchymal cell dysfunction and necrosis [44].

Therefore, antioxidants, prescribed in burn shock, binding free radicals, reduce vascular permeability, improve the course of burn disease, prevent the development of complications, reduce damage to internal organs [45].

Tanaka et al. found that adjuvant high dose ascorbic acid (66 mg/kg/h for 24h), administered during the first 24h after thermal injury, significantly decreased the amount of fluid given compared to the control (patients who received vitamin C required infusions of 3 ml/% of burn /kg, while patients who received one Ringer's solution lactate, required 5.5 ml/% of burn/kg of solutions per day [46].

High-dose vitamin C treatment (bolus 66 mg/kg and maintenance dose 33 mg/kg/hr) reduces endothelial damage to sham burn levels, whereas half the dose is inefficient. High-dose vitamin C should be considered for parenteral treatment in every burn patient [12].

Octreotide, a synthetic somatostatin analogue, has been shown to improve the reperfusion-induced oxidative damage in rats with ACS by reducing levels of MPO activity and malondialdehyde and increasing levels of glutathione when given before decompression. Therefore, octreotide might ultimately be shown to have a therapeutic role as a reperfusion injury-limiting agent among patients with IAH and ACS [47].

So, resuscitation of patients with severe burn injuries should be aimed at the early restoration of the circulating blood volume and microcirculation using a minimum number of solutions. This helps to prevent IAH, the development of the ACS, MOF in patients with severe burn injury.

Non-operative and percutaneous interventions may be applied before surgical decompression is considered. Nasogastric decompression, the use of neuromuscular blocking agents, prokinetic agents, enemas, or colonic decompression, the removal of excess

fluid by ultrasound-guided percutaneous drainage, or by a combination of continuous veno-venous hemofiltration (CVVH) with ultrafiltration and/or diuretics, are simple and possibly effective tools to reduce IAP [3, 44].

Cheatham et al. [48] showed in 62 patients with IAH/ACS treated with percutaneous catheter decompression (PCD) versus traditional open abdominal decompression (OAD) that both techniques were equally effective. Successful PCD therapy was associated with either fluid drainage above 1,000 mL or a decrease in IAP of > 9 mm Hg in the first four hours post decompression. PCD appears to be most effective in patients with secondary ACS due to massive fluid resuscitation in burns. Latenser et al. [49] showed that PCD reduced IAP and prevented ACS in 55 % of burned patients. PCD is a relatively simple technique, cost effective and less invasive than OAD. Bedside ultrasonography to identify intraperitoneal fluid or blood is necessary [44].

Circumferential abdominal burn eschars might also lead to ACS by producing a tourniquet effect. At bedside, urgent decompressive escharotomy of the abdominal wall is a safe surgical procedure that provides rapid relief of intra-abdominal pressure. It improves ventilation, hemodynamic parameters, and oxygen metabolism and can decrease morbidity and mortality [27]. An escharotomy of the trunk to improve abdominal wall compliance should be performed early, especially in the presence of 3rd degree burns [18].

The open abdomen in trauma and non-trauma patients has been proposed to be effective in preventing or treating deranged physiology in patients with severe injuries or

critical illness when no other perceived options exist. Its use, however, remains controversial as it is resource consuming and represents a non-anatomic situation with the potential for severe adverse effects [50].

Although a midline laparotomy may make wound management more difficult in abdominal burn patients, it remains very effective in reducing IAP.

Regardless of surgical decompression, it is important to continue to measure IAP postoperatively in order to recognize recurrent IAH and/or ACS. The presence of abdominal burns may pose specific challenges to the management of the open abdomen with regard to infectious complications. The presence of significant protein loss via an open abdomen needs to be considered [51]. Early enteral and/or parenteral nutrition is of the utmost importance in these hypercatabolic patients, although recent literature results may advocate the opposite in ICU patients with ACS [52–53]. However, strong emphasis needs to be placed on the tremendous morbidity and high mortality of an open abdomen in patients with burns [18]. Its use, therefore, should only be considered in patients who would most benefit from it [50].

CONCLUSIONS

Intra-abdominal hypertension is a frequent complication in severe burn patients requiring massive fluid resuscitation. Development of ACS in burn patients is associated with high mortality. Prevention, early detection and proper management may avoid this usually fatal complication. Fluid resuscitation volume is directly responsible for the development of ACS in severe burned patients. Thus, optimal fluid resuscitation can be the best prevention of IAH and ACS.

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