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Review Article

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Post Traumatic Abdominal Compartment Syndrome: Clinical Presentation, Complications, Risk Factors, Management and Prevention

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Abstract

Abdominal compartment syndrome (ACS) is a lethal clinical entity occurring in severely injured patients and resulting in multiple organ failure and death .As a consequence of anincreased intra-abdominal pressure (IAP), this syndrome was first described early in 1890, and well characterised in the late 20th century. Continued researches in trauma care has ledtoidentify risk factors and determine prediction models resulting in a great help for clinicians in early detecting and preventing post-traumatic ACS. Damage control surgery with tightly abdomen closure, large crystalloid volume resuscitation and delayed bleeding control were identified as modifiable ACS predictors. Untreated or delayed decompression, post-injury ACS leads to multiple organ failure including mainly respiratory, renal and cardiac systems. Progress made in trauma care has led to the development of new concepts for trauma management including damage control resuscitation, damage control surgery and open abdomen strategy. The implementations of these new strategies resulted in increased improvement of survival and almost complete elimination of post-traumatic ACS with very early recognition and prevention. These new concepts form the modern trauma care for the management of severely injured patients.

Keywords: Decompression; Damage control resuscitation; Intra-abdominal hypertension; Organ failure

Introduction

The intra-abdominal hypertension (AIH) leads to the development of abdominal compartment syndrome (ACS) in severely injured patients. First described in 1980 [1], this clinical entity was clearly recognized in pediatric surgery in the late 20th century [2]. Started in the 19th century[1] and continued during the 20th century [3], clinical research on IA pathophysiology resulted in more characterizing the abdominal compartment syndrome with highlighting the relationship between the massive abdominal distension not due to bleeding and high volume resuscitation[4]. Damage control surgery (DCS) with abdomen closure has increased the prevalence and clinical relevance of ACS in injured patients [3,5]. Performing definitive surgery in critically injured patients was reputed to be detrimental for outcomes with increased on-table mortality. Introduced in 1983, staged surgery approach which aimed to restore normal patient physiology and delay definitive surgery, has substantially improved survival [6].Moreover, staged surgery associated with open abdomen strategy has resulted in substantial reduction of ACS development. Very large volume resuscitation in severely injured patients with traumatic shock has led to increased intra-abdominal pressure (IAP) resulting in ACS occurrence with multiple organ failure [7-9].The continued research in trauma care has led to the development of a new concept 'damage control resuscitation (DCR) [9,10,11,12,13]. Subsequently, DCR associated to DCS has increasingly reduced the ACS rate. Therefore, DCR and DCS form the modern trauma care continuum in critically injured patients.

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History of ACS

Acute augmentation of intra-abdominal pressure in injured patients leads to the development of abdominal compartment syndrome (ACS). This lethal complication of intra-abdominal hypertension (IAH) was described early in 1890 [1], but it was well recognized in the late 20th century by the pediatric surgeons in patients who underwent omphalocele closure [2]. These patients developed pulmonary compromise and impaired renal function secondary to increased intra-abdominal pressure [2]. Clinical research on pathophysiology of IAH begun in the 19th century by highlighting the catastrophic effects of shock and IAH on body functions in animals [1]. The research on IAH was continued in the 20th century, and in 1984, Kron reported the first case series [3]. Five years later, Fietsam gave the name 'Abdominal Compartment Syndrome (ACS)' to this syndrome that it was described as "increased ventilator pressure, increased central venous pressure, and decreased urinary output associated with massive abdominal distension not due to bleeding [4]. The same author published [4] a case series (four patients) who developed ACS within 24 h after surgery for the ruptured abdominal aorta, and all patients experienced this syndrome have received a high resuscitation volume (> 25 L). Continued research in trauma care has led to more characterize the ACS and identifying the risk factors and clinical prediction models. Created in 2004, the World Society of ACS published an updated consensus definitions and clinical practice guidelines of the Abdominal Compartment Syndrome and the IAP [14].

Clinical presentation

The ACS has been classified as primary and secondary ACS. Primary ACS often occurs in the context of severe injuries with abdominal or retroperitoneal lesions requiring emergency surgery [14,15]. The classic pattern of primary ACS is a severely injured patient with substantial intra-abdominal bleeding and traumatic shock who under-

went damage control surgery with abdominal wall closure [6,14,16 and 17]. Also, the ACS can be occurred in blunt trauma with solid organ injuries necessitating surgical management and treated conservatively [18,19]. First described in 1995 [18], secondary ACS often occurred in injured patients presented with only major extra-abdominal injuries [14,15], resulting in hemorrhagic shock and need of substantial transfusion [14,20-29], Major injuries can include pelvic fractures, penetrating chest or cardiac injuries or vascular limb trauma.

Since the introduction of the IAP measurement method using the urinary catheter, the secondary ACS recognition was increased particularly in patients with uncontrolled bleeding receiving large volumes blood products and crystalloid [22,23]. The ACS occurs rapidly and evolution can be fatal within 24 h from injury, so early prediction and prevention are the best way to avoid this lethal syndrome [26]. Because of its clinical significance and lethality, continued research in trauma care has investigated this clinical entity (ACS) to more characterize the syndrome, identify the risk factors and develop methods for early detection and prevention. Refinement of the surgical technique and innovation in resuscitation methods made in the beginning of the 21st century, have led to the development of damage control surgery and damage control resuscitation strategies resulting in almost complete eradication of the ACS in trauma patients.

3.3. Pathophysiologic bases and organ system effects of ACS

Formed in 2004, the World Society of ACS published based -evidence guidelines on definition and management of the Abdominal Compartment Syndrome [14].The urinary bladder is used to measure the IAP, and the IAH was defined as IAP superior to 12 mm Hg without physiology derangements and was graded from I-V on the basis of IAP increments [14]. The ACS is defined as an intra-abdominal pressure (IAH) of more than 20 mmHg with new organ dysfunction or failure, that improved after eliminating increased IAP. The ACS is presently understood as a clinical entity frequently signaling uncontrolled resuscitation. High volume resuscitation in majorly injured patients with traumatic shock resulted in intestinal oedema and increased intra-abdominal pressure in tightly packed or closed abdomen leading to the development of ACS [30]. Sometimes delayed bleeding control associated with substantial crystalloid overload results in ischemia-reperfusion injuries, intestinal oedema and coagulopathy that are described as the salty water vicious cycle leading to ACS development with multiple organ failure and death [15]. Detrimental effect of large crystalloid volume resuscitation has been confirmed [15], and additional crystalloid administration worsens the intestinal oedema, aggravates the IAP and propagates the cycle [15,28,29]. The increase of hydrostatic pressure and decrease of oncotic pressure lead to fluid flows into the interstitium. The lymphatic system designed to remove excess flow is impaired, and similarly, the increased venous outflow obstruction is leads to increasing fluid movement into the interstitium and bowel lumen [31].

Pathophysiologically, post injury ACS is considered as the second hit mechanism of multiple organ failure [26,32,33]. Severe trauma results in systemic inflammatory response and ischemia–reperfusion injury that can lead to multiple organ failure development, if this primary insult is severe enough. However, further stimulation of immune response can be provoked by a second additional hit (second hit mechanism) leading to exacerbation of patient condition and development of multiple organ failure when the initial insult is not severe enough. Therefore, this second hit phenomenon results in exaggerated immune response after second stimulation of activated neutrophils [34] and it has been shown to occur as early as 3-6 h after injury and continue for further 24 h. Also, decompression causes a second hit through an ischemia- reperfusion mechanism which was characterized by a large decrease in the gastric mucosal carbon dioxide (GAPCO20) [34]. The decrease in carbon dioxide has been shown to be a strong predictor of ACS [35]. Reperfusion injury after decompression was the major cause of sudden death on the operating table [36]. The ACS effects on organ systems include cellular swelling, hypoxia and dysfunction leading to multiple organ failures. As reported by prospective and retrospective studies [25,26,36,37], the ACS associated multiple organ failure rates varied from 11% to 71%.

Cardio-circulatory and pulmonary effects

The AIH raises the diaphragm resulting in reducing thoracic cavity volume and cardiac return through inferior vena cava compression. So, blood preload is further decreased by pooling blood in the pelvis and lower limbs [38]. The direct cardiac compression results in reducing contractibility, increasing load on the right ventricle, augmenting systemic vascular resistance and pulmonary pressure [39]. Decrease in thoracic cavity volume reduces thoracic compliance and increases ventilation pressure leading to diminishing the functional residual capacity [9,40].So ACS is associated with highly risk of developing acute respiratory distress syndrome [41].

Renal effects

The IAH reduces the renal artery blood flow by direct compression, resulting in reduced glomerular filtration rate [42,43]. Renal dysfunction clinically presents as oliguria progressing to anuria. In addition, the renal hemodynamic alteration activates the renin–angiotensin– aldosterone system which increases renal vascular resistance to compensate reduced blood flow by retaining salt and water [42,44,45].

Gut effects

The IAH impairs splanchnic perfusion leading to intestinal ischemia and infarction [46,47]. As suggested, intestinal effects of ACS are some of the first to occur [31].

Effects on the abdominal wall and liver

Decreased abdominal wall perfusion results in ischemia and reperfusion leading to oedema with reducing compliance and exacerbating IAH [48]. The liver function can be impaired during IAP increase by reduction of hepatic arterial and microcirculatory blood flow, and portal vein compromise [49].

ACS risk factors and ACS as predictor of second hit mechanism

The abdominal wall and abdominal content volume are the key factors determining the IAP, and any change of these variables leads to exacerbate or alleviate the future ACS. The abdominal wall compliance is affected by constriction including tight dressings, tension closure, burns, eschar and interstitial abdominal wall oedema.

Instead, the intra-abdominal content can be increased by packing, bleeding, bowel obstruction, hemopneumoperitoneum and intestinal oedema. The effects of damage control surgery, large blood loss and large crystalloid resuscitation on the IAP increase and ACS development have clearly been demonstrated [50]. Despite the similar presentation of both primary and secondary ACS, the causes, the injury patterns and the resuscitation differ as previously shown. Factors indicative of damage control management are mostly the predictors of primary ACS, whereas, secondary ACS has the features of the large resuscitation volume [15]. Some risk factors for ACS development have been established including severe trauma, substantial fluid re-

suscitation, massive transfusion, hemorrhagic shock, damage control surgery, penetrating abdominal trauma and highly positive 24h fluid balance [5,17,29]. As known, hemorrhagic shock with no response to aggressive resuscitation and severe tissue trauma are the risk factors for development of multiple organ failure [51].

As previously reported, both primary and secondary ACS occurred early, often within 3-6 h from injury [31,52]. Indeed, Parameters within the first 6H from admission have been used to develop an early predictive model for both primary and secondary ACS [15]. So, two clinical relevant time points have been determined, emergency department discharge (< 3h) and admission in ICU (< 6h) (Table 1). Also, the gastric mucosal acidosis measurement has been described as an another early predictor of ACS [15]. The gastric mucosal acidosis is a sensitive and independent predictor of both primary and secondary ACS before the pathological increase of IAP, but this monitoring method did not have a widespread acceptance in clinical practice [15].

Time	Primary ACS	Secondary ACS
At the time of ED discharge	urgent laparotomy without imaging >3 L crystalloid infusion	>3 L crystalloid infusion >3 units of blood transfusion No urgent bleeding control procedure
At the time of ICU admission	Temperature <34°C Haemoglobin < 8g/l Base deficit worse than 8 mmol/L	>7•5 L of crystalloids < 150 mL urine during the first hour on ICU

Table 1: Predictors of post-traumatic ACS.

Also, ACS was identified as a strong independent predictor factor for multiple organ failure development [15,31,53], and is currently considered asa second hit mechanism of organ failure [26,33, 34,54,55]. So, the development of prediction models in high -risk patients with severe trauma resulted in large-scale elimination of ACS.

ACS Management

The standard treatment of post injury ACS is the surgical decompression with increasing abdominal cavity volume, and decreasing abdominal contents by removing fluid, blood, or packs from the abdomen. Surgical decompression frequently resulted in temporary renal function improvement, increased cardiac output, and improvement of lung function. Despite slight physiological improvement following decompression, the outcomes were still poor. Occurring very early after injury (within 6 H) and if untreated, the ACS is followed by multiple organ failure. Surgical decompression was often performed too late or resulted in extensive ischemia-reperfusion injuries [56]. The time to decompression has been shown to be crucial factor for patient survival in the early 21st century [24,25,30]. Indeed, the reperfusion injury following late decompression of full ACS was major cause of sudden death in the operating room [36,56]. The reported mortality associated with reperfusion injury related to late or delayed decompression was up to 75% [24,25,30]. Importantly, early recognition of ACS and early decompression within 24 h has increased survival rates compared to delayed decompression after 48 h with a mortality rate of 38% and up to 80% respectively [20,21,36]. So, early recognition and surgical decompression are important but the primary prevention remains the best strategy to eradicate the ACS and its associated complications (multiple organ failure).

Instead of non-traumatic ACS, Less invasive or conservative management (drugs and supportive care in ICU) is not a treatment option for post injury ACS because of the rapid progression to organ failure. Additionally, the World Society of Abdominal Compartment Syndrome has summarized the non-operative strategies applicable principally in less severe and non-traumatic ACS [14,57]. Percutaneous decompression for secondary and post burn ACS has a promising evidence [58,59]. Also, the use of laparoscopic and percutaneous linea alba fasciotomies in post pancreatitis ACS is well documented [60]. Although surgical abdominal decompression remains the life-saving procedure, it results in open abdomen. The open abdomen (OA) is a morbid condition including peritoneal infection, atmospheric fistulas (EAF), abdominal wall hernias, increased health care utilization and poor life quality [61-64]. A variety of techniques has been described for temporary closure of the abdominal wall (towel-clip closure, Bogota bag, mesh), but recurrent ACS can occur with an open abdomen, so, sustained surveillance of IAP is highly recommended [31,24,65]. OA is associated with an over workload in ICU and operating room and prolonged hospital stay The negative pressure wound therapy (NPWT) is presently the recommended therapy for OA and results in enhancing fascial closure rates and preventing viscero-abdominal wall dherence [66,67]. Recurrent ACS and EAF can develop with NPWT [15,31], and late surgical reconstruction is needed to treat resultant hernia [68,69,70] . As previously reported, high crystalloid volume resuscitation is detrimental for outcomes leading to primary and secondary ACS development [65,71]. Continued research on trauma care and ACS has led to developing a new approach so called "damage control resuscitation". This concept includes fluid restriction, early and massive transfusion of blood products, and hemostatic resuscitation by early administrating clotting factors in severely injured patients [11,12]. The damage control resuscitation (DCR) has increasingly reduced the severe traumatic shock rate in resuscitated patients to only sporadic cases [50].

ACS status and prevention

Blunt or penetrating abdominal trauma with severe shock, physiological derangements (hypothermia, acidosis, coagulopathy) and need to damage control surgery approach to achieve bleeding controand abdominal contamination, are the predictive models of post injury ACS. Therefore, primary ACS prevention can be achieved with rapid control of bleeding (implementing DCS), damage control resuscitation with fluid restriction and open abdomen approach [72]. AS the Clinical pattern of secondary ACS is an injured patient with extra-abdominal bleeding and delayed definitive care (bleeding control), prevention of secondary ACS is based on identifying and managing the shock causes with controlled crystalloid-based resuscitation. he bleeding control is an essential step in preventing and limiting the intra-abdominal hypertension and its timeliness should be independent from the type of method to use (surgical or trans catheter) [73,74].

The modern trauma care based on Damage control resuscitation, damage control surgery and open abdomen strategy and the introduction of predictive models, damage control resuscitation and damage control surgery have led to almost eradication of both post injury ACS and secondary ACS [50] resulting in increasingly reduced incidence [11,12,54,62,72] .However, the prevention remains the best way, and identifying the risk factors previously described, meticulous surveillance of high-risk patients based on established prediction models and high adherence to modern care principles are the keys of successful early detection and prevention of the post injury ACS.

ACS and future perspectives

The great documented achievement of the modern trauma care is the complete eradication and successful prevention of post injury ACS. This important achievement resulted in increased reduction of mortality in high-risk patients with severe traumatic shock (about 5 %with very low margins for potentially avoidable deaths). The damage control resuscitation strategy (DCR) is the important element of the modern trauma care. This new approach is rested on restriction of crystalloid administration, haemostatic resuscitation, early blood product transfusion and permissive hypotension [9-13]. So, the DCR implementation has increasingly reduced the adverse effects of the high volume fluid resuscitation. When correctly implemented, DCR has improved patient survival, decreased hospital stay length and increased definitive surgery with reducing need to DCS strategy [12,75,76]. More recently, the DCR strategy has reduced the need for surgical haemostasis in severe liver trauma by directly addressing the trauma-induced coagulopathy [77]. Moreover, this new approach of resuscitation can be implemented in the management of any situation of severe bleeding irrespective of the bleeding cause.

Lessons learned from excellent results of trauma care research and improvements of post injury ACS are highly valuable for the surgical and critical trauma care community and should be translated to the benefit of broad populations of patients. In fact, the incidence of 0-2% of post injury ACS is valid only for the highest performing trauma centers and outcomes can be much poorer in non-specialized centers. Therefore, continued education about risk factors, preventive strategies, IAP monitoring and ACS surveillance are so essential and important in major abdominal or pelvic trauma and in patients with trauma shock undergoing resuscitation. In addition, most severely injured patients with trauma shock develop some transient sub-ACS frequently in the ICU which remains clinically insignificant. Therefore, establishing the relevance of transient and persistent IAH in high-risk injured patients may avoid potential poor out comes.

Declaration of Interests

None Declared

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