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Postinjury Abdominal Compartment Syndrome: Are We Winning the Battle?

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Abstract Postinjury (primary) abdominal compartment syndrome (ACS) was described more than 15 years ago as severe abdominal distension with high peak airway pressures, CO_2 retention, and oliguria, which led to unplanned re-exploration after damage-control laparotomy. Later, a more elusive type of ACS was recognized, which develops without abdominal injuries (secondary ACS). Both syndromes were recently characterized, their independent predictors were identified, and preventive strategies were developed to reduce their incidence. Once viewed as a syndrome with almost uniform mortality, systematic preventative strategies and therapeutic efforts have reduced the prevalence, morbidity, and mortality of the syndrome. This review was designed to summarize the recent advances in the management of ACS, to classify the currently available evidence, and to identify future directions of research and clinical care.

Introduction

Postinjury abdominal compartment syndrome (ACS) was described more than 15 years ago as severe abdominal distension with raised peak airway pressures, CO_2 retention, and oliguria, which led to unplanned re-exploration after damage-control laparotomy [1]. This severe life-threatening complication of abdominal packing often was associated with lethal reperfusion syndrome at the time of decompression. The incidence of ACS was 15% amongst packed patients with a mortality of 62.5%. Systematic clinical and basic science research has helped us to better characterize, monitor, prevent, and treat this deadly syndrome. This review was designed to summarize the recent advances in the management of ACS, classify the currently available evidence, and identify future directions. These goals would serve clinicians who believe that they have potentially not recognized the syndrome so far, as well as those who are contributors to our currently improved understanding of ACS, and those who are eager to find answers to some of the persisting unsolved questions.

Definitions

In the literature before 2000, many different definitions of ACS and intra-abdominal hypertension (IAH) were used; these were generally based on abdominal distension, ventilatory pressures, and oliguria [1, 2]. Intra-abdominal pressure (IAP) measurement was not routinely performed and often was reported in different units, such as cmH_2O or mmHg [3, 4]. Later, IAP was routinely measured and some leading research groups defined ACS as IAP $>$ 25 mmHg with organ dysfunction, which improved after surgical decompression [5–9]. The lack of consensus in definitions made it difficult to compare the results of published data, where both the numerator and the denominator differed (Table 1). At the World Congress of ACS in 2004, the World Society of Abdominal Compartment Syndrome established consensus definitions [10].

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Table 1 Postinjury abdominal compartment syndrome studies

Study	Population	Demographics	ACS definition	Incidence	Mortality (%)	MOF (%)
Morris et al. [1]	107	ISS = 32, age = 32	Tense abdomen, PAP	16 (15%)	63	N/A
Hirshberg et al. [2]	124	ISS = 46, age = 22	Tense abdomen, PAP	4 (3%)	100	N/A
Meldrum et al. [3]	145	ISS = 26, age = 39	IAP > 20 mmHg with OD	21 (14%)	29	43
Ivatury et al. [4]	70	ISS = 22, age = 28	IAP > 25 cmH_2O	23 (32%)	44	N/A
Chang et al. [5]	11	ISS = 27, age = 37	IAP > 25 mmHg, OD	N/A	63	N/A
Ciresi et al. [34]	9	ISS = 25, age = 37	IAP > 20 mmHg with OD	9 (7%)	22	11%
Maxwell et al. [32]	46	ISS = 25, age = 36	Decompression	6 (13%)	67	0
Ertel et al. [6]	311	ISS = 30, age = 38	IAP > 25 mmHg with OD	17 (5.5%)	35	N/A
Chen et al. [7]	25	ISS = 20, age = 30	IAP > 25 mmHg	5 (20%)	N/A	N/A
Offner et al. [25]	52	ISS = 28, age = 33	IAP > 20 cmH_2O with OD	17 (33%)	35	71
Raeburn et al. [63]	77	ISS = 29, age = 35	IAP > 20 mmHg with OD	28 (36%)	43	34
Tremblay et al. [64]	131	ISS = 24, age = 36	Tense abdomen	12 (9%)	58	N/A
Balogh et al. [29]	128	ISS = 28, age = 41	Decompressed abdomen	11 (9%)	55	55
Hong et al. [65]	706	ISS = 18, age = 42	IAP > 20 mmHg with OD	6 (1%)	50	50
Gracias et al. [26]	30	ISS = 33, age = 35	IAP > 25 mmHg with OD	5 (17%)	60	N/A
Balogh et al. [8]	188	ISS = 28, age = 39	IAP > 25 mmHg with OD	26 (14%)	58	54
Mayberry et al. [66]	9	ISS = 24, age = 47	IAP > 25 mmHg with OD	N/A	22	N/A
Cothren et al. [9]	2,762	ISS = 33, age = 36	IAP > 25 mmHg	37 (1.3%)	37	24
Howdieshell et al. [67]	88	ISS = 28, age = 32	IAP > 30 mmHg with OD	10 (11%)	N/A	23
Kozar et al. [28]	337	ISS = 24, age = 31	Decompression	3 (0.9%)	0	0
Miller et al. [68]	344	ISS = 35, age = 36	IAP > 20 mmHg with OD	115 (33%)	20	N/A
Cothren et al. [37]	N/A	ISS = 27, age = 40	IAP > 20 mmHg with OD	N/A	54	62
Scalea et al. [69]	102	ISS = 29,age = 34,	IAP > 20 mmHg with OD	24%	42	N/A
Madigan et al. [70]	48	ISS = 25, age = 41	IAP > 25 mmHg with OD	1.3%	60	N/A

ACS abdominal compartment syndrome, *Incidence* incidence of ACS, *mortality* mortality of ACS, *MOF* incidence of multiple organ failure among patients with ACS, *N/A* not available, *ISS* injury severity score, *IAP* intra-abdominal pressure, *OD* organ dysfunction, *age* in years

- Intra-abdominal hypertension is defined as IAP > 12 mmHg without pathophysiology of ACS. IAH is graded from I–IV based on the IAP value: Grade I: 12–15 mmHg; Grade II: 16–20 mmHg; Grade III: 21–25 mmHg; Grade IV: >25 mmHg.
- Abdominal compartment syndrome is defined as sustained IAP > 20 mmHg that is associated with new organ dysfunction/failure defined.
- Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention.
- Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region.

Clinical presentation/epidemiology

Postinjury ACS is an early complication of severe shock and resuscitation, which presents with (relative) oliguria, renal dysfunction, increased airway pressures, decreased pulmonary compliance, increased intracranial pressure, decreased cardiac output, artificially high filling pressures, increased vascular resistance, and poor intestinal perfusion [4, 11–13]. Many of these signs are common with severe shock, acute lung injury, and acute renal failure. If these acute organ dysfunctions are caused by increased IAP, more aggressive ventilation strategies and preload-driven resuscitation are unlikely to improve the patient condition. Further resuscitation attempts without addressing the IAH can lead to further physiological derangement and full-blown ACS. Ongoing bleeding and consequent uncontrolled resuscitation can present as severe hypovolemia compounded by ACS. Distinguishing between ACS and nonresponding shock and/or early pulmonary failure is driven by the measurement of the IAP, exclusion of continued bleeding, and the critical preload assessment. Clinical examination has repeatedly been shown to be inaccurate to estimate IAP [14, 15]. For the definition and the diagnosis of ACS, the monitoring of IAP is essential [16]. Several techniques have been introduced since Kron's original description but the intravesical ("urinary bladder pressure") seems to be the most feasible technique in high-

risk polytrauma patients [17, 18]. Postinjury ACS usually develops within 12 h of intensive care unit (ICU) admission, therefore, immediate regular (2 hourly) or continuous IAP monitoring is recommended in traumatic shock resuscitation patients [8, 19].

Primary ACS

Damage-control surgery has made it possible to salvage patients with catastrophic abdominal injuries and severe hemorrhagic shock [8, 20–22]. The classical presentation of primary patients with ACS was severe abdominal trauma undergoing damage-control laparotomy with packing and abdominal closure [1, 2, 23, 24]. Previously, attempts to perform primary definitive surgery led to death on the operating table, but with the introduction of the damage-control concept the abbreviated laparotomy helped to get patients to the ICU to restore their physiology. ACS arose as a potentially lethal new complication and one of the frequent causes of unplanned return to the operating room (OR) [1, 4]. Total body shock and subsequent reperfusion with intestinal edema and a tightly packed and closed abdomen created the scenario for increased abdominal pressure. Later, liberal application of open abdomen management with temporary abdominal closure was adopted for prevention [25]. Today, cold, acidotic, coagulopathic patients are transferred from the OR to ICU with an open abdomen. As a result, the incidence of postinjury primary ACS is decreasing in centers where this practice is followed [25]. IAP monitoring also is important in this group, because ACS can still develop in open-abdomen patients (recurrent ACS) [8, 26, 27]. Another presentation of postinjury primary ACS is during the nonoperative management of abdominal solid organ injuries [7, 28]. In the current era of modern trauma care, most blunt solid organ injuries are successfully managed nonoperatively. The development of ACS in this patient group is viewed as a complication or failure of nonoperative management. In this setting ACS could present early with massive intra-abdominal bleeding, which necessitates laparotomy to achieve hemostasis or later when the symptoms of increased IAP are more pronounced than those of the acute blood loss.

Secondary ACS

Without the presence of intra-abdominal injuries, the presentation of ACS is very elusive [8, 29–31]. The term was coined by Maxwell et al. [32], but the first case was described earlier by Burrows et al. [33]. The common characteristics of these patients are hemorrhagic shock requiring massive resuscitation without abdominal injuries [29, 34–36]. The typical injury patterns are penetrating heart, major vessel, or extremity vascular trauma associated with profound shock and subsequent massive (crystalloid) resuscitation resulting in whole-body ischemia/reperfusion injury [29, 34–38]. Secondary ACS may occur in scenarios with multiple long bone fractures, where the acuity of bleeding is less, but definitive hemorrhage control is more difficult to achieve. These patients are at high-risk to undergo cyclic uncontrolled resuscitation and coagulopathy-related rebleeding from fractures and soft-tissue injuries. “The futile crystalloid loading” to maintain target resuscitation end points (blood pressure, oxygen delivery index) leads to generalized edema, manifesting as intestinal swelling, ascites and decreased abdominal wall compliance resulting in secondary ACS [39].

The epidemiology of both primary and secondary ACS is continuously evolving. During the early 1990s, primary ACS was epidemic due to damage control-related improved survival and tight abdominal closure after packing [3, 23]. Secondary ACS at that time was infrequently recognized because of its elusive nature and the lack of monitoring [32, 35]. The incidence of the two syndromes leveled at the turn of the millennium, as the importance of open abdomen management in this critically ill group was recognized, reflected in decreased incidence of primary ACS [8]. At the same time, secondary ACS was increasingly recognized with bladder pressure monitoring in patients who had uncontrolled bleeding and underwent massive crystalloid based resuscitation [37–39]. In 2003, we reported an ACS incidence of 14% (6% primary and 8% secondary) among a high-risk group of shocked polytrauma patients undergoing standardized computerized shock resuscitation [8]. Both types of clinical presentation of ACS occurred within 24 h of ICU admission, with secondary ACS being a slightly earlier phenomenon than primary ACS. Later, the liberal use of open abdomen and the introduction of hemostatic resuscitation further decreased the incidence of primary ACS. We identified the independent predictors for both forms of ACS and recognized the detrimental effects of supranormal resuscitation with excessive use of crystalloids [39]. These efforts started to decrease the incidence of postinjury secondary ACS [40].

We believe that today in centers with an appropriate level of awareness, primary ACS should be extremely rare, occurring mainly in the nonoperatively managed abdominal solid organ injured group, and that secondary ACS must disappear. According to our view, in the future both syndromes might be regarded as indicators of suboptimal care (delayed presentation, inadequate/delayed hemorrhage control, and poor-quality resuscitation).

Independent predictors and recommended treatment

The seminal case reports, retrospective series, and expert opinions identified a long list of risk factors (shock, severe trauma, damage control laparotomy, massive transfusion, massive crystalloid resuscitation, penetrating abdominal trauma, highly positive 24-h fluid balance etc.) [1–4, 24]. Based on a uniformly resuscitated large prospective cohort, both primary and secondary ACS can be predicted as early as emergency department discharge but at the latest by ICU admission [8]. These findings made it possible to identify patients who were later likely to develop the lethal syndrome even before IAP measurement started on ICU. Considering the early presentation of ACS during the ICU course of these critical patients, any strategy after calculating the highly positive 24-h fluid balance seemed futile [41]. Although patients with primary and secondary ACS had similar demographics, injury severity (obviously different injury pattern), and shock severity, their independent predictors were surprisingly distinct. The independent predictors of primary ACS are the indicators of the damage control physiology (transfer to the operating room without further imaging, temperature <34°C, hemoglobin <8 g/dl, base deficit >8 mmol/l), whereas the secondary ACS predictors are markers of uncontrolled resuscitation (>7.5 l of crystalloids before ICU admission, no indication for life-saving surgical intervention, relatively low urine output ($\leq$150 ml/h) on ICU admission considering the massive resuscitation) [8]. Poor intestinal perfusion measured by gastric tonometry was an independent predictor for both primary and secondary ACS. The usefulness of gastric tonometry also was shown by other groups [4, 13]. Our findings showed that gastric tonometry can indicate impending ACS well before the clinically apparent IAH [42].

Identification of these independent predictors urged us to strive for earlier hemorrhage control in orthopedic trauma, abandoning crystalloid-based supranormal resuscitation goals, introducing hemostatic resuscitation, and the extension of the ICU resuscitation protocol to the emergency department [43]. The fact that shock resuscitation is currently going through a fundamental change and the radically decreasing incidence of ACS, make it futile to validate the previously developed independent predictors. Most likely new predictors are required and the old predictors might be still valid for postinjury IAH but not for ACS (Balogh ZJ, van Wessem K, Yoshino O, Moore FA, unpublished data).

The current treatment of full-blown postinjury ACS is surgical decompression. Usually the acuity of the syndrome and the potential for abdominal rebleeding does not allow conservative measures, which could be useful in IAH or less rapidly developing nontraumatic ACS cases [44–46]. There is evidence that percutaneous decompression may be efficacious in secondary ACS trauma cases and post-burn ACS scenarios [47, 48]. Furthermore, subcutaneous decompression is recommended in acute pancreatitis-related ACS [49]. During the first 24 h after injury, ACS presents as an immediately life-threatening condition with profound organ dysfunctions. In these situations, the risk of a potentially inadequate decompression cannot be afforded, therefore, our recommendation is immediate surgical decompression/re-exploration. Abdominal decompression may be performed in the ICU, especially in secondary ACS cases, whereas in primary ACS repeated hemorrhage control is usually required, preferably undertaken in the OR.

After decompression, open abdomen management starts with the application of temporary abdominal closure followed by timely restoration of the abdominal wall as soon as the IAH is resolved [50]. Our results indicate that the previously recommended hypervolemic resuscitation with crystalloid boluses in IAH and impending ACS is detrimental and leads to a "vicious cycle of crystalloid loading" with worsening IAH leading to full-blown ACS [42, 51].

Outcomes

Untreated postinjury ACS is uniformly lethal. The mortality and incidence of multiple organ failure (MOF) among patients with ACS is depicted in Table 1. Clinical and basic science research has revealed that ACS is an independent predictor of MOF and can be considered as a modifiable second hit in our current concept of the pathomechanism of postinjury MOF [8, 43, 52, 53]. Historically, ACS described by landmark reports was decompressed too late, leading to fatal reperfusion syndrome. The authors recommended preventive strategies to overcome the effects of reperfusion at the time of decompression [1]. Timely decompression could result in improved outcomes when secondary ACS case series were compared (before and after 24 h) [32, 36]. Among patients who develop ACS and are decompressed within the first 24 h, earlier decompression does not lead to improved outcomes [29, 36]. This finding, and the potentially morbid nature of open abdomen management prompted us to target prevention rather than earlier decompression [54]. The response to decompression with improved urine output and cardiac index are indicators of potentially improved outcome, whereas other postdecompression parameters had no association with survival [8]. Although a life-saving strategy, open abdomen is potentially a morbid condition with increased risk for abdominal abscess, fistula, and major abdominal wall hernia formation [50]. Patients with planned ventral hernia after open abdomen have a poor quality of life, especially in the short-term, and require major reconstructive

procedures several months after their initial injury [55]. Despite this knowledge, the latest prospective data show that the long-term physical and emotional outcomes for patients after open abdomen ventral hernia and fascial closure are similar [56].

Available level of evidence

There are 148 peer-reviewed publications in the literature that use the search terms "intra-abdominal pressure"; "intra-abdominal hypertension"; "abdominal compartment syndrome"; and "trauma" as of November 30, 2008. The time distribution and the type of studies are depicted in Fig. 1. Similar to other recently recognized syndromes, case reports are published first, followed by retrospective series, and later by prospectively designed studies together with basic science work to answer specific questions. Later, randomized trials and meta-analyses could supply the highest level of evidence. Based on postinjury ACS literature during the past 15 years, the authors believe that there are three phases of the development of our understanding of the syndrome. The first 5 years was the "recognition" phase with seminal case and retrospective reports, followed by the next 5-year period of boom or "momentum." Most of our current best evidence is from this era, when postinjury primary and secondary ACS were characterized, including epidemiology, risk factors, independent predictors, and outcomes. Supported by basic science and prospective clinical studies, recommendations were published on prevention and treatment of ACS. The most recent 5-year era of "plateau" is characterized by continued linear growth of case reports, retrospective series, and review articles but no further improvement of our higher level knowledge. There are two possible reasons for the plateau period of postinjury ACS-related evidence. The continued linear growth of lower level of evidence is not surprising because it was similar during the previous phases; this is due to the widespread recognition of the syndrome in different parts of the world. We believe that the major achievements in the understanding of causative factors of ACS have decreased the incidence of the syndrome in centers where most of the original prospective data was taken. The decreasing incidence of postinjury ACS will make it more difficult to answer more focused clinical questions, because research will take longer and will probably require multicenter efforts to produce the necessary higher level of scientific evidence. The discussed phases also are well represented if the literature is classified according to the Oxford recommendations on levels of evidence in science (Fig. 2) [57]. The current best level of evidence (1b) is related to IAP measurement in trauma patients, where it is relatively simple to run prospective studies on patients who do not necessarily have ACS [15, 19]. The 2b and 2c studies are currently our best evidence applicable to the prevention and treatment of postinjury ACS. These are related to the effects of IAP on intracranial pressure, the harmful effects of crystalloids during supranormal resuscitation, the reduction of planned ventral hernia formation with utilization of vacuum-assisted closure, the influence of body position on IAP, and the long-term effects of open abdomen management [39, 55, 56, 58–60].

Conclusions and future directions

Postinjury primary and secondary ACS became evident with the ability to keep critically shocked trauma patients alive until ICU admission. The trauma surgical and critical

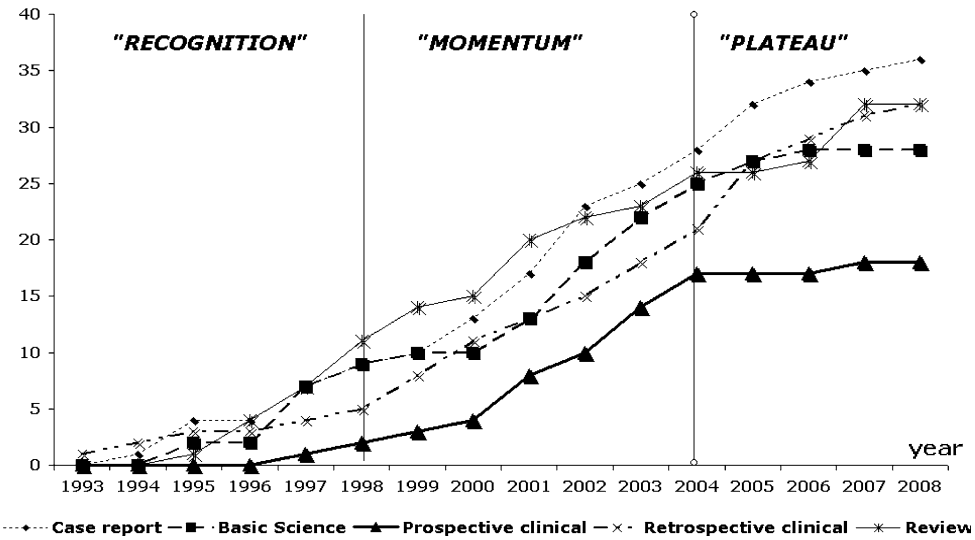

Fig. 1 Cumulative number and type of published studies on postinjury ACS

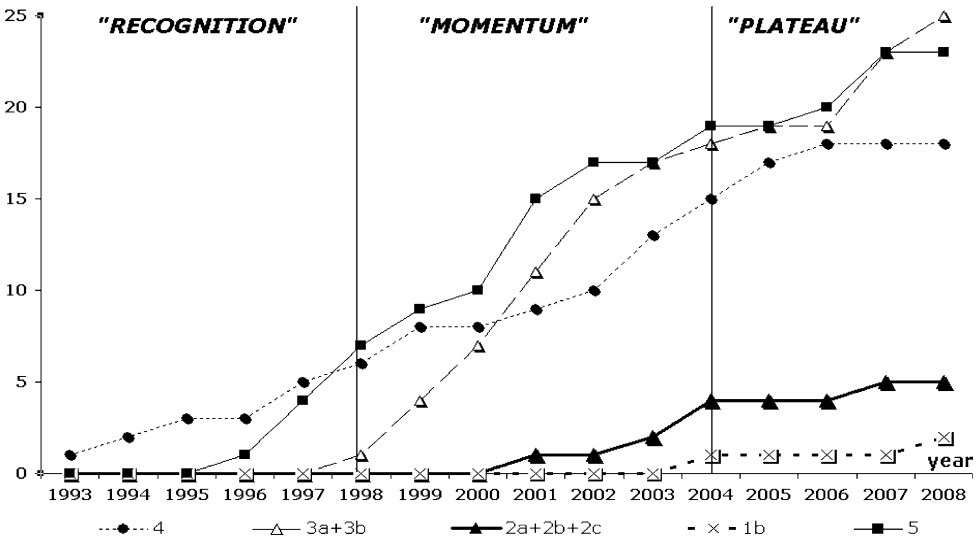

Fig. 2 Levels of evidence in postinjury ACS publications based on the Oxford classification system [57]

care community learned to refine damage control to enable earlier hemorrhage control, combined with open abdomen management, therefore minimizing the chances of developing primary ACS. The understanding of the pathophysiology and predictors of secondary ACS fundamentally changed our approach to traumatic shock resuscitation. Uncontrolled crystalloid resuscitation aiming for supranormal oxygen delivery goals was the standard of practice for a long time, especially in North America; however, this has become obsolete due to deleterious outcomes. Although it was driven by ACS research, this change in resuscitation strategy has had a major impact not just on the elimination of secondary ACS, but on overall trauma care as well.

The next challenges of postinjury ACS research are in four major areas. First, parallel to the development of traumatic shock resuscitation: to find convincing evidence on the superiority of hemostatic resuscitation and to define the optimal ratios of blood component therapy and the roles of alternative fluid resuscitation strategies, such as blood substitutes, hypertonic saline, and colloids. Alternative resuscitation strategies may have a major role in reversing the cascade of pathophysiological events in impending postinjury (mainly secondary) ACS without decompression [61, 62]. Second, the identified independent predictors and subsequent prevention strategies must be implemented on a larger scale and their impact needs to be monitored. In this strategy, the role of the World Society of Abdominal Compartment Syndrome (WSACS) is fundamental. Third, although the incidence of full-blown postinjury ACS seems to be decreasing, most of shock resuscitation patients develop grade II-III IAH. The clinical significance of this transient phenomenon is poorly understood. Multicenter epidemiological studies orchestrated by the Clinical Trial Working Group of the WSACS are attempting to answer this question. Fourth, the prevention of lethal consequences of ACS has created the new clinical challenge of open abdomen-related short- and long-term morbidity. The optimal management of the open abdomen is one of our major surgical challenges.

Conclusions

Systematic work during the last 15 years has made an impact on the incidence and outcome of postinjury ACS. We believe that our research and clinical community are progressing toward the elimination of this deadly syndrome. To achieve this, a higher level of scientific evidence is needed to overcome the current "plateau" phase by focusing internationally on the four key areas that we identified.

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