Postinjury Primary Abdominal Compartment Syndrome

Zsolt Balogh[1], Cino Bendinelli[1], Timothy Pollitt[1], Rosemary A. Kozar[2], Frederick A. Moore[3]

Abstract

Postinjury abdominal compartment syndrome (ACS) has evolved during the 1980s together with the introduction of damage control surgery (DCS) principles. DCS made it possible to salvage severely injured trauma patients who previously would have exsanguinated due to uncontrollable coagulopathic bleeding. These patients had severe hemorrhagic shock; their abdomens were tightly packed and had ongoing massive resuscitation. ACS is a lethal complication of the damage control patients. For today the pathophysiological characteristics of ACS are described, the intra-abdominal pressure is measured on many intensive care units. Postinjury ACS (primary and secondary) is one of the better characterized etiological types of ACS: risk factors, diagnostic criteria, independent predictors and preventive strategies are all well documented. Since the mortality of full-blown postinjury ACS is still unacceptably high and does not seem to improve with earlier decompression, prevention is the recommended strategy to decrease the morbidity and mortality. Open abdomen is one of the important preventive strategies but it is not free from morbidity and mortality. With aggressive open abdomen management in postinjury ACS these complications can be minimized. More importantly, timely hemorrhage control and hemostatic resuscitation are the likely solutions for more efficient prevention of the postinjury ACS.

Key Words
Abdominal compartment syndrome (ACS) · Damage control surgery (DCS) · Abdominal

Eur J Trauma Emerg Surg 2008;34:369–77

DOI 10.1007/s00068-008-8106-9

[1] Division of Surgery, Department of Traumatology, John Hunter Hospital and University of Newcastle, Newcastle, Australia,
[2] Department of Surgery, Memorial Hermann Hospital, UT-Houston Medical School, Texas Medical Center, Houston, TX, USA,
[3] Department of Surgery, Methodist Hospital, Texas Medical Center, Houston, TX, USA.

Received: June 20, 2008; revision accepted: July 2, 2008;
Published Online: July 30, 2008

Introduction

Postinjury abdominal compartment syndrome (ACS) is a serious complication of abdominal solid organ injuries causing significant morbidity and mortality [1–7]. The pathophysiological changes with increased intra-abdominal pressure (IAP) were recognized as early as the ninteenth century and basic science data are available from the early twentieth century [8]. ACS was neglected for a long time in surgical practice; the mandatory closure of the abdomen was an accepted practice after trauma laparotomy, even when the closure was difficult. Pediatric surgeons recognized the problem during the closure of large omphaloceles. To prevent the catastrophic respiratory consequences of tight abdominal closure, the application of a silo with gradual decrease of its volume was recommended [9]. The term ACS was coined by Fietsam in 1989 after describing four postoperative abdominal aortic aneurysm patients with tense abdomen, oliguria, hypoxia, hypercarbia and high peak inspiratory pressures [10]. Later, the IAP measurement became available and high IAP was a part of the definition of ACS [11, 12]. Clinical studies showed that physical examination is inadequate to estimate IAP, and measurement primarily via the urinary bladder became a standard practice on most of the trauma intensive care units (ICP) [13, 14]. Postinjury ACS presented as an epidemic with the widespread application of damage control surgery. Patients with critical physiology and catastrophic abdominal injuries underwent abbreviated surgery, involving rapid hemorrhage and contamination control followed by packing (Figure 1). The fact that these previously unsalvageable patients survived after whole body ischemia-reperfusion injury, massive fluid resuscitation and tightly packed abdomens made ACS a frequently described new syndrome [15–17].

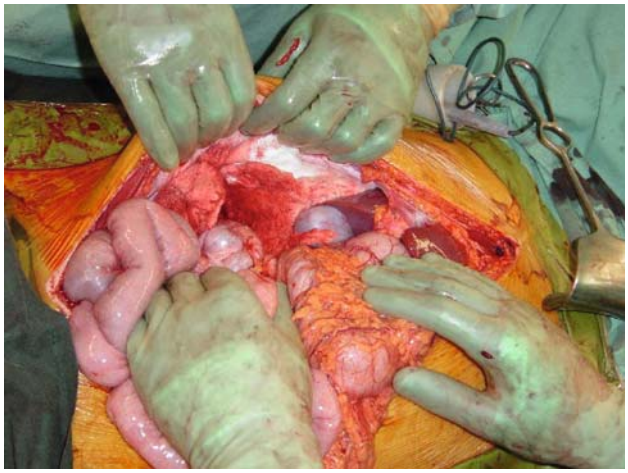


Figure 1. Peritoneal packing during damage control laparotomy.

Definitions

These are based on the World Society of Abdominal Compartment Syndrome (WSACS) consensus definitions [12, 18].

Intra-abdominal pressure (IAP) is the steady-state pressure concealed within the abdominal cavity. IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line. The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 ml sterile saline. The normal IAP is approximately 5–7 mmHg in critically ill patients.

Intra-abdominal hypertension (IAH) is defined by a sustained or repeated pathological elevation in IAP $\geq$ 12 mmHg. IAH is graded as Grade I: 12–15 mmHg, Grade II: 16–20 mmHg, Grade III: IAP 21–25 mmHg, Grade IV: IAP $>$ 25 mmHg

Abdominal compartment syndrome (ACS) is defined as a sustained IAP $>$ 20 mmHg that is associated with new organ dysfunction/failure. ACS is an all or nothing diagnosis and should not be graded like IAH.

Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention.

Secondary ACS refers to conditions that do not originate from the abdominopelvic region. Most often it is a consequence of whole body ischemia and consequent massive resuscitation.

Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.

The Pathophysiology of ACS

IAH can affect virtually all organ functions of the body, when impending ACS emerges the dysfunctional/failing organs became obvious. Before the central role of the increased IAP in the complex pathophysiology was recognized, the support of individual failing organs was futile and frequently made the situation worse (crystalloid loading, increasing positive and expiratory pressure, diuretics, etc).

Cerebral Perfusion

The altered cerebral perfusion was classically described in morbidly obese patients with chronic IAH. Increased IAP forces the diaphragm upward thus decreasing the size of the thoracic cavity and causing intra-thoracic pressure (ITP) to increase. High ITP causes increased jugular venous pressure and impairs venous return from the brain, which can increase intracranial pressure and consequently decrease cerebral blood flow [19–21]. These changes do occur in the acute setting and IAH/ACS can make the intracranial pressure and cerebral perfusion worse in polytrauma patients with combined head and abdominal injuries [22, 23].

Cardiac Function

Increased IAP impairs venous return causing a ''pooling'' of fluid in the lower extremities. High ITP artificially increases central venous and pulmonary wedge pressure measurements. Simultaneously, left ventricular afterload increases due to increased systemic vascular resistance. Increased ITP can increase right ventricular afterload, which, when extremely high, causes right ventricular failure and dilation with consequent leftward displacement of the ventricular septum and impairment of left ventricular filling [24–28]. Clinically the patient has poor cardiac output with high filling pressures and high systemic vascular resistance.

Respiratory Function

Increased IAP pushes the diaphragms into the thoracic cavity. Thoracic compliance decreases and increased pressure is required for mechanical ventilation. Additionally, functional residual capacity is decreased and ventilation/perfusion mismatch is increased, leading to impaired oxygenation [27, 28]. Clinically the patient is ''difficult to ventilate and oxygenate''.

Renal Function

Oliguria or anuria despite aggressive fluid resuscitation is a typical sign of ACS. Mechanisms responsible for the decreased renal function include direct compression of the renal parenchyma, decreased perfusion of

the kidneys due to decreased cardiac output, and the increased water and sodium retention due to activation of the renin–angiotensin system [28–31]. It is important to interpret the urinary output in the context of the magnitude of fluid resuscitation instead of relying on relatively normal absolute numbers.

Gut Function

Increased IAP impairs splanchnic perfusion by decreasing cardiac output and increasing splanchnic vascular resistance. When severe, tissue ischemia can result [32–34]. Clinically this can be monitored with gastric tonometry by recording the gastric mucosal pH, P_{CO_2} and GAP_{CO_2}.

Extremity Perfusion

Increased IAP increases femoral venous pressures, increases peripheral vascular resistance, and reduces femoral artery blood flow by as much as 65% [35]. Extremity compartment syndrome due to resuscitation and reperfusion injury shares some of the common predictors with ACS [36].

Epidemiology and Typical Patient Characteristics

Postinjury primary ACS in the context of solid organ injuries most likely occurs in the following typical scenarios:

(1) Damage control laparotomy, traumatic shock with massive resuscitation, tight abdominal packing and fascial closure or inadequate temporary abdominal closure performed.
(2) As above, but with functional temporary abdominal closure. Later, in the ICU the patient's intestinal reperfusion injury worsens, more edema develops, or the patient continues to bleed. This is defined as a postinjury recurrent ACS.
(3) Cases with attempted nonoperative management of solid organ injury with massive hemoperitoneum can develop primary ACS without previous laparotomy.
(4) Late development of primary ACS (after 24 h) may occur due to complications of solid organ injuries (operative or nonoperative management) such as abdominal sepsis, bile or pancreatic leak.

Many factors make it difficult to describe the incidence of postinjury ACS. Most of the studies in the literature are case series, reporting on mixed (trauma and nontrauma) populations and do not separate the incidence of primary and secondary ACS. The reported incidence of ACS also depends on how the syndrome is defined (numerator) and on the study population (denominator). Since most of the trauma studies were reported before the consensus definitions, it is not surprising that numerators are hardly comparable (Table 1). For the purpose of this review (ACS with abdominal solid organ injuries) the incidence of the postinjury primary ACS is the targeted outcome.

We evaluated our prospective traumatic shock resuscitation database to describe the epidemiology of ACS and aimed to compare primary and secondary ACS and develop a prediction model [1]. Over a 44-month period 188 major torso trauma patients were resuscitated by standard computer-assisted protocol; 26 developed ACS, 162 did not. There were 11 (6%) primary ACS patients. Basic epidemiologic comparisons are summarized in Table 2. The demographics, Injury Severity Score, injury patterns, Glasgow Coma Scale, and initial base deficit were not different among the non-ACS, and primary ACS groups. In the emergency department (ED), the ACS patients had lower systolic blood pressure, which prompted more aggressive fluid and blood resus-

Table 1. Postinjury abdominal compartment syndrome studies.

Author	Study population	ISS and age	ACS definition	Inc (%)	Mort (%)
Morris [2]	Retrospective, damage control, n = 107	ISS = 32, age = 32	Tense abdomen, ↑PAP	15	63
Hirshberg [6]	Retrospective, damage control, n = 124	ISS = 46, age = 22	Tense abdomen, ↑PAP	3	100
Meldrum [3]	Prospective, ISS > 15, trauma laparotomy, n = 140	ISS = 26, age = 39	IAP > 20 mmHg with OD	14	29
Ivatury [4]	Retrospective, "severe abdominal trauma", n = 70	ISS = 22, age = 28	IAH: IAP > 25 cmH_2O	32	44
Ertel [17]	Retro- and prospective, damage control, n = 311	ISS = 30, age = 38	IAP > 25 mmHg with OD	5.5	35
Offner [5]	Retrospective, damage control, n = 52	ISS = 28, age = 33	IAP > 20 cmH_2O with OD	33	35
Raeburn [16]	Retrospective, damage control, n = 77	ISS = 29, age = 35	IAP > 20 mmHg with OD	36	43
Hong [7]	Prospective, ICU admissions, n = 706	ISS = 18, age = 42	IAO > 20 mmHg with OD	1	50
Balogh [1]	Prospective, ISS > 15, BD > 6, > 6 U PRBC/12 h, n = 188	ISS = 28, age = 39	IAP > 25 mmHg with OD	14	58

ACS: Abdominal compartment syndrome; inc: the incidence of ACS; mort: the mortality of ACS; ISS: injury severity score; IAP: intra-abdominal pressure; IAH: intra-abdominal hypertension; ICU: intensive care unit; BD: base deficit in mEq/l; PRBC: packed red blood cells in units; OD: organ dysfunction; PAP: peak airway pressure

Table 2. Demographics, injury severity, and outcomes.

	Non-ACS n = 162	1° ACS n = 11
Demographics		
Age (years)	39 ± 1	36 ± 5
Male gender (%)	76	73
Blunt mechanism (%)	85	82
Injury severity		
ISS	27 ± 1	29 ± 2
GCS	13 ± 1	13 ± 1
Severity of shock		
ED BD (mEq/l)	9 ± 1	11 ± 1
ED SBP (mmHg)	93 ± 2	79 ± 3*
ED PRBCs (U/h)	1.4 ± 0.2	3.1 ± 1*
ED crystalloids (l/h)	1.8 ± 0.1	1.6 ± 0.1
Hospital times (hours from admission)		
ED discharge	2 ± 0.1	0.9 ± 0.1*
ICU admission	7 ± 0.3	3.7 ± 0.5
Decompressive lap	–	14 ± 1
ICU admission data		
BD (mEq/l)	4 ± 0.3	9.5 ± 1*
Lactate (mmol/l)	4.9 ± 0.2	7.7 ± 1*
Crystalloids (l)	6.7 ± 0.3	8.5 ± 0.8
PRBCs (units)	6.3 ± 0.3	8.9 ± 2*
Hb (mg/dl)	11.4 ± 0.2	7.9 ± 1*
Outcomes		
Ventilator (days)	8 ± 2	13 ± 3*
ICU (days)	12 ± 2	14 ± 5
MOF (%)	12	55*
Mortality (%)	17	64*

* $p < 0.05$ between non-ACS and primary ACS

ACS: Abdominal compartment syndrome; 1° ACS: primary abdominal compartment syndrome; ISS: injury severity score; GCS: Glasgow coma scale; ED: Emergency Department; BD: base deficit; SBP: systolic blood pressure; PRBC: packed red blood cells; OR: operating room; IR: interventional radiology; lap: laparotomy; Hb: hemoglobin concentration; ICU: intensive care unit; MOF: multiple organ failure

citation. Primary ACS patients typically spent less time in ED; as soon as their catastrophic abdominal injuries were recognized, they were triaged to the operating room for emergency laparotomy. On arrival to the ICU the primary ACS group had worse metabolic acidosis, lower hemoglobin and required more PRBC transfusion. Primary ACS patients required decompression in an average of 10 h after ICU admission and 14 h after hospital admission. Despite initially adequate physiologic responses to decompression, the outcomes (ventilator days, multiple organ failure (MOF), mortality) of primary ACS were uniformly poor compared to the non-ACS group. In summary, primary ACS is an early complication in about 6% of the shocked trauma patients and tends to occur among those who are hypotensive on presentation, require emergency laparotomy, and do not resolve their acidosis by the end of the surgery, necessitating significant transfusion on ICU admission.

Risk Factors, Independent Predictors, Clinical Indicators

These three terms were often used interchangeably especially in the early literature describing postinjury ACS as a new clinical problem.

Risk factors are based on expert opinion and not necessarily on sound statistical analysis. For postinjury primary ACS these could be severe trauma, major abdominal injuries, hemorrhagic shock, damage control laparotomy, tight fascial closure after damage control laparotomy and low intestinal pH [2–7, 16, 17]. The international experts of the WSACS listed 29 risk factors that may lead to the development of IAH (Table 3); of these 14 risk factors could potentially

Table 3. Risk factors for IAH/ACS.

Postinjury primary ACS
Abdominal surgery, especially with tight fascial closures
Acidosis (pH < 7.2)
Coagulopathy
Damage control laparotomy
Gastroparesis/gastric distention/ileus
Hemoperitoneum/pneumoperitoneum
Hypothermia (core temperature < 33 °C)
Intra-abdominal infection/abscess
Major trauma
Massive fluid resuscitation (> 5 l colloid or crystalloid/24 h)
Mechanical ventilation
Peritonitis
Polytransfusion (> 10 U packed red blood/24 h)
Use of positive end expiratory pressure (PEEP) or the presence of auto-PEEP
Other from of ACS
Acute pancreatitis
Bacteraemia
Distended abdomen
High body mass index (> 30)
Intra-abdominal or retroperitoneal tumors
Laparoscopy with excessive inflation pressures
Liver dysfunction/cirrhosis with ascites
Major burns
Massive incisional hernia repair
Peritoneal dialysis
Pneumonia
Prone positioning
Sepsis (American–European consensus conference definitions)
Volvulus

have a role in the development of postinjury primary ACS [12]. Since clinical examination has been proven to be inaccurate to estimate IAP [13, 14] and postinjury ACS develops during the first 12 h after ICU admission [1], the WSACS recommends that patients with any of these risk factors should be screened for IAH/ACS by IAP measurement upon ICU admission and in the presence of new or progressive organ failure [12, 18].

To find the statistically sound independent predictors of primary ACS, we performed a multiple logistic regression analysis on our data set. Given the early occurrence of postinjury ACS, we focused our prediction models on the first 6 h after hospital admission [1]. We developed two prediction models to address two major clinical decision-making timeframes: ED model (0–3 h, i.e., all patients have initial diagnostic work-up, clinical laboratory results and discharge from the ED completed) and ICU model (0–6 h, i.e., all patients have been admitted to ICU and their first physiological and clinical laboratory measurements on the resuscitation protocol are available). As ACS patients' outcomes were far worse than their non-ACS counterparts with similar initial physiology and injury severity, our goals were to identify the independent risk factors that may be causative and to build prediction models that will identify high-risk patients early in resuscitation so that standard care can be modified to prevent or improve the outcome of patients who show early signs and symptoms of ACS. Since postinjury ACS is not a homogenous group, separate models were built for primary and secondary ACS patients. The ED model for primary ACS has identified the need for rapid transfer of the patient to the operating room, and more than 3 l of crystalloids during this short timeframe as independent predictors (Table 4). This model is highly sensitive; applying this to strategies for prevention of postinjury primary ACS will ensure that minimal patients will be missed. Primary ACS predictors upon ICU admission (low temperature, low hemoglobin concentration, increased gastric regional P_{CO_2} and high base deficit) are the reasons that damage control surgery is elected. The ICU model is very specific, which could accurately identify the patients who already have impending primary ACS. It is important to clarify that IAP (a variable used in the definition of ACS) was not used in the prediction model. The receiver operator characteristic analysis showed that ACS can be predicted with 0.88 accuracy by the ED model and with 0.99 accuracy by the ICU model.

The clinical indicators for postinjury primary ACS are the actual criteria, which define the syndrome: the presence of IAH and newly developing organ dysfunction(s).

Prevention

Despite timely decompression (within 10 h of ICU admission) the outcome of the primary ACS patients is very poor (Table 2) [1]. ACS patients in our resuscitation protocol were decompressed as soon as the symptoms developed, frequently even at the bedside to save time and restore the physiology. In this scenario earlier decompression is very unlikely to be a feasible aim to improve outcomes. Based on the identified independent predictors, the timely hemorrhage control and the prevention of the ''bloody vicious cycle'' (coagulopathy, acidosis and hypothermia) are paramount [37]. This review focuses on the intra-abdominal sources of bleeding, but in polytraumatized patients a more holistic approach to control all extraperitoneal sources is essential. Our extensive clinical research has helped to identify that the preventative interventions must aim at the very early hospital course, where after the identification of these patients an alternative

Table 4. Independent predictors of primary ACS in ED and ICU models.

Independent predictors	Odds ratio	95% confidence interval	Sens	Spec	PPV	NPV
ED model 1° ACS						
To OR < 75 min	102.7	9.65–> 999.9	91	67	16	99
Crystalloid > 3 l	69.8	10.21–477.7	82	76	18	98
ICU model 1° ACS						
Temp < 34 °C	22.9	1.39–378.25	55	94	43	96
GAP_{CO_2} > 16	54.3	2.15–> 999.9	91	76	22	99
Hb < 8 g/dl	206.1	7.41–> 999.9	73	92	40	98
BD > 12 mEq/l	3.5	1.37–839.50	46	98	56	96

1° ACS: Primary abdominal compartment syndrome; To OR: time to the operating room; Sens: sensitivity; Spec: specificity; PPV: positive predictive value; NPV: negative predictive value; GAP_{CO_2} : CO_2 gap; UO: urinary output; Hb: hemoglobin concentration; Temp: temperature; BD: base deficit; ED: emergency department

resuscitation strategy could be used. The role of crystalloid loading is more evident in secondary ACS but it is also a strong independent ED predictor of primary ACS [1, 38]. Vital sign driven uncontrolled crystalloid resuscitation should be avoided from the very beginning of the resuscitation. Because of these the UT-Houston shock resuscitation protocol was extended to the ED and more recently started to use 1:1 ratio of fresh frozen plasma and packed red blood cells [39].

In the operating room, timely hemorrhage control and minimization of heat loss together with hemostatic resuscitation and active/passive rewarming are the main principles of management. After damage control surgery in the abdomen, it is a standard practice today to leave the abdomen open (Figure 2). This can minimize but not completely eliminate the risk of ACS. There are many techniques available for temporary abdominal closure, from the classic Bogota-bag, through the improvised vacuum seal techniques to the proprietary devices, such as vacuum-assisted closure (VAC) [40]. Open abdomen management in this critical patient population was probably the single most efficient measure to decrease the incidence of ACS; however, the concept of open abdomen should not be over utilized. Most of the abdominal trauma patients are still best managed with primary fascial closure at the time of the first surgery if no damage control physiology is present and/or no re-exploration is necessary. On ICU admission the previously recommended resuscitation to a supra-normal oxygen delivery goal (DO2I > 600) is shown to have detrimental effects in causing more IAH, ACS, MOF and in increasing mortality compared to the normal goals (DO2I > 500) [41]. Some of the initial expert opinions in this field recommended hypervolemic resuscitation (to increase the preload) in impending ACS to overcome the early signs of organ dysfunctions [42]. Our results have shown that this strategy is not successful and will cause full-blown ACS [43]. The evaluation of more recent changes in the shock trauma resuscitation protocol (hemostatic resuscitation, extension of the protocol to the ED, central venous pressure driven resuscitation instead of pulmonary artery catheter resuscitation) is ongoing and the results are likely to offer more efficient prevention.

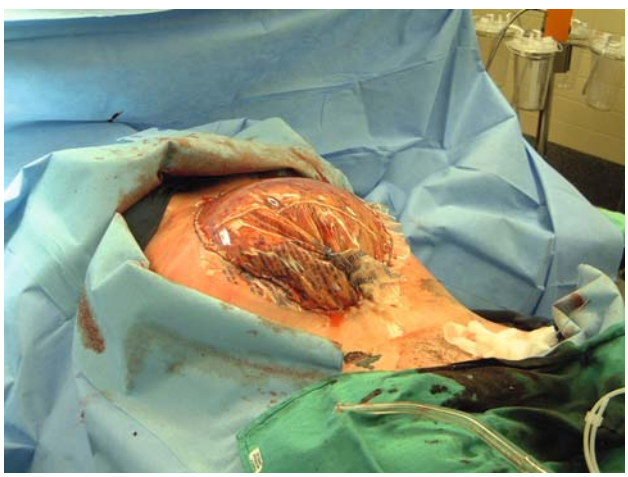


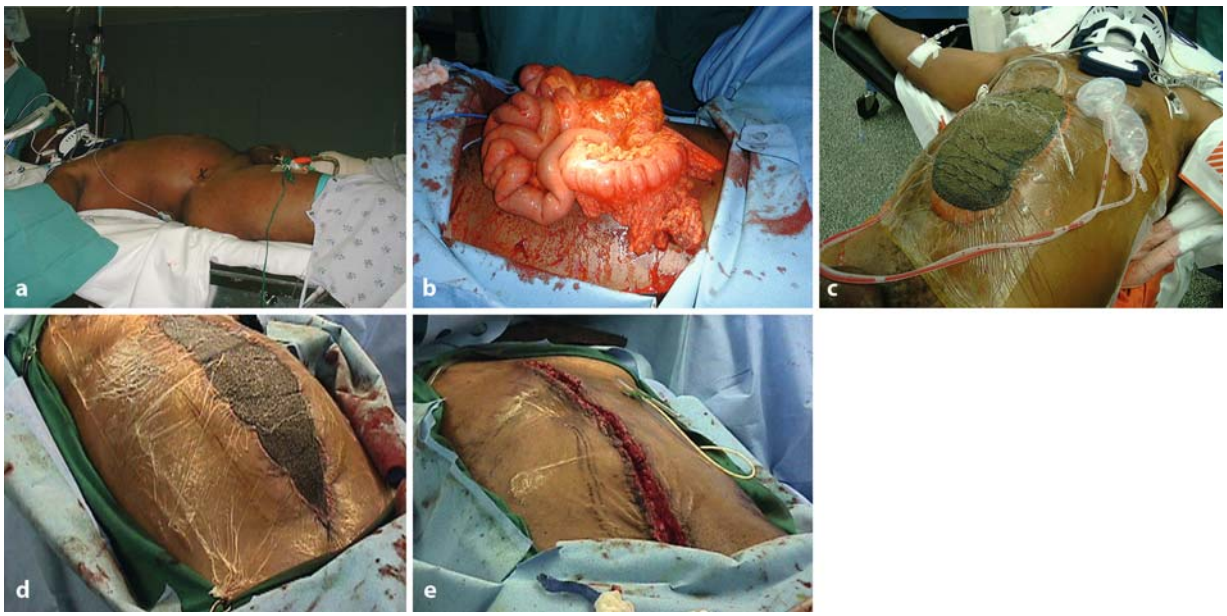
Figure 2. Temporary abdominal closure with Bogota-bag (sterile infusion bag stitched to the skin).

Treatment

Once the diagnosis of primary ACS is established, the gold standard is prompt surgical decompression via a midline xipho-pubic laparotomy. Retained blood clots and excess packs are evacuated, and the abdomen is left open, utilizing one of the temporary abdominal closure techniques. The requirements of any temporary abdominal closure technique are to provide adequate decompression of the fascia, containment for the peritoneal contents, and a proper seal to control peritoneal fluid. The optimal temporary abdominal closure should not harm the fascia or the skin and should facilitate the gradual approximation of the fascial/skin edges. Detailed discussion of the open abdomen management is beyond the scope of this review. Open abdomen is an important strategy to prevent primary ACS but it has significant morbidity and mortality. Temporary abdominal closure must be changed regularly with the aim of earliest possible closure (Figures 3a–e). Recent publications on aggressive open abdomen management report over 80% primary fascial closure rate with a very low complication (fistula, infection, and abscess) rate [40, 44].

Percutaneous drainage of the peritoneal fluid is an attractive option and well documented in the burn and pediatric literature [44–46]. First, this method is very unlikely to be efficient in acute damage control patients where ACS is caused by intestinal edema, space-occupying packs and clotted blood, rather than drainable free peritoneal fluid. Second, in a damage control patient with multiple intra-abdominal injuries, primary or recurrent ACS on day 1 means rebleeding until proven otherwise. Rebleeding obviously necessitates reassessment of the abdominal hemostasis with decompression and relook laparotomy. Percutaneous peritoneal drainage can be a valuable tool for a selected patient group, where primary ACS develops during the nonoperative management of abdominal solid organ injuries (liver or spleen).

There are elegant case series published on the use of minimally invasive subcutaneous midline fascioto-



Figures 3a to 3e. Stages of open abdomen management. (a) Distended abdomen with intra-abdominal pressure of 36 mmHg acutely developing renal, cardiac and respiratory dysfunction. The patient is on the operating table before decompression. (b) Edematous small and large bowel after decompression. (c) Temporary abdominal closure with vacuum-assisted closure. (d) Gradual fascial approximation during abdominal vacuum-assisted closure changes. (e) Fascial closure achieved on day 7 postinjury.

mies in severe acute pancreatitis patients [47]; however, no results are available on trauma patients. This technique is unlikely to play a major role in damage control patients with significant solid organ injuries.

The WSACS summarizes the available nonoperative medical management strategies, which are increasingly recognized as playing potentially important roles in both the prevention and treatment of IAH [12, 18]. By reducing thoracoabdominal muscle tone, sedation, paralysis, and analgesia may potentially decrease IAP to less detrimental levels and therefore could have a role in IAH management. There is a lack of prospective trials evaluating benefits and risks of sedation and analgesia in IAH/ACS. These nonoperative methods are potential adjuncts in IAH, but in the presence of full-blown ACS with organ dysfunction(s), surgical decompression is currently the accepted treatment of choice. Nasogastric and rectal drainage, enemas, and endoscopic decompression are other simple and minimal invasive methods to reduce IAP and treat mild to moderate IAH in nonacute, non-life-threatening scenario(s). Motility agents such as erythromycin, metoclopromide, or neostigmine may have an unproven role in evacuating intraluminal contents and decreasing the size of the viscera. The role of these adjuncts role is potentially limited during the first 24 h when postinjury primary ACS develops.

Outcome

The outcome of ACS patients measured by ventilator days, the incidence of MOF and mortality was significantly worse than that of the non-ACS patients' with similar demographics, shock and injury severity (Table 1 mortality, and Table 2 outcomes). Several papers suggested the possible connection between ACS and poor outcome intuitively [2–7, 16, 17]. Later, it was proven statistically that ACS is a predictor for both MOF and mortality based on logistic regression analysis [1].

Abdominal decompression, unless it is done very late, results in uniform improvement in physiology of the ACS patients regardless of their outcome [1, 3, 38]. The IAP, systemic vascular resistance, gastric regional CO_2, base deficit and peak airway pressure decrease while the urinary output, mean arterial pressure, cardiac index, mixed venous oxygen saturation, arterial pH, pulmonary compliance and the PaO_2/FiO_2 ratio increase [1]. We found only two variables that were different after decompression between survivors and nonsurvivors. Survivors had a better urinary output response than nonsurvivors. The cardiac index was the only variable that improved only among survivors, but not among those who died [1].

Studies in the last 15 years, including our biggest cohort have failed to document convincing improvement in the outcome of ACS. Despite the use of earlier decompression and the liberal use of the temporary

open abdomen techniques, the outcome of ACS remains very poor [48]. This suggests that the prevention of the syndrome should be more fruitful than early recognition and decompression. Our more recent unpublished data in a smaller cohort shows promising results that postinjury ACS could be almost completely eliminated with prospective awareness.

Future Directions

Abdominal solid organ injury-related primary postinjury ACS is still not uniformly recognized everywhere [49]. Since clinical examination is unreliable in the detection of ACS and IAH, it is essential that every ICU dealing with trauma patients should measure IAP through the urinary catheter. The high-risk groups such as damage control patients, abdominal trauma patients and shocked patients requiring massive resuscitation could benefit from continuous IAP monitoring during the resuscitation phase [50].

The optimal resuscitation fluid, which carries oxygen and offers prevention from reperfusion injury and interstitial edema is still to be described. Efficient hemorrhage control techniques without significant space-occupying packs are required during the solid organ injury management. With the liberal use of the open abdomen management in high-risk patients the incidence of classic primary ACS developing during the first 24 h of hospital admission should diminish. Sporadic cases, especially during the nonoperative management of abdominal solid organ injuries and late septic complications of abdominal organ injuries are still likely to occur.

After eliminating the lethal syndrome of primary ACS, the challenge of the future will be to define the significance of IAH without organ dysfunction and to manage the open abdomens in a safe, timely and cost efficient manner.

Conclusion

Postinjury primary ACS has emerged as a clinical entity due to our increased ability to keep the most severely injured patients alive by applying the principles of damage control. During the management of abdominal solid organ injuries, ACS could develop among both operated and nonoperatively managed patients. The high-risk group with obvious shock on arrival requiring damage control laparotomy, could be identified early based on the available independent predictors, and preventive measures should be undertaken (open abdomen, fine-tuned resuscitation). Adequate monitoring is essential for the timely recognition of postinjury ACS, which based on the current evidence, should be treated with urgent surgical decompression. Prevention of the syndrome is the key strategy, given that once ACS develops, the prognosis is uniformly poor. The increased use of preventive open abdomen will decrease the incidence of ACS, but trauma surgeons must be prepared to deal efficiently with the challenge of open abdomen management.

References

1. Balogh Z, McKinley BA, Holcomb JB, et al. Both primary and secondary abdominal compartment syndrome (ACS) can be predicted early and are harbingers of multiple organ failure. J Trauma 2003;54:848–59 (discussion 859–61).
2. Morris JA, Eddy VA, Blinman TA, et al. The staged celiotomy for trauma: issues in unpacking and reconstruction. Ann Surg 1993;217:576–86.
3. Meldrum DR, Moore FA, Moore EE, et al. Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg 1997;174:667–72.
4. Ivatury RR, Porter JM, Simon RJ, et al. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance of gastric mucosal pH and abdominal compartment syndrome. J Trauma 1998;44:1016–23.
5. Offner PJ, de Souza AL, Moore EE, et al. Avoidance of abdominal compartment syndrome in damage-control laparotomy after trauma. Arch Surg 2001;136:676–81.
6. Hirshberg A, Wall MJ Jr, Mattox KL. Planned reoperation for trauma: a two year experience with 124 consecutive patients. J Trauma 1994;37:365–9.
7. Hong JJ, Cohn SM, Perez JM, et al. Prospective study of the incidence and outcome of the abdominal compartment syndrome. Br J Surg 2002;89:591–6.
8. Coombs HC. The mechanism of the regulation of intra-abdominal pressure. Am J Physiol 1920;61:159–63.
9. Lacey SR, Bruce J, Brooks SP. The relative merits of various methods of indirect measurements of intra-abdominal pressure as a guide to closure abdominal wall defects. J Pediatr Surg 1987;22:1207–11.
10. Fietsam R Jr, Villalba M, Glover JL, Clark K. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. Am Surg 1989;55:396–402.
11. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for exploration. Ann Surg 1984;199:28–30.
12. Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. Part I Definitions. Intensive Care Med 2006;32:1722–32.
13. Kirkpatrick AW, Brenneman FD, McLean RF, et al. Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients?. Can J Surg 2000;43:207–11.
14. Sugrue M, Bauman A, Jones F, et al. Clinical examination is an inaccurate predictor of intraabdominal pressure. World J Surg 2002;26:428–1431.
15. Rotondo MF, Schwab CW, McGonigal MD, et al. 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma 1993;35:375–82 (discussion 382–3).

16. Raeburn CD, Moore EE, Biffl WL, et al. The abdominal compartment syndrome is a morbid complication of postinjury damage control surgery. Am J Surg 2001;182:542–6.
17. Ertel W, Oberholzer A, Platz A, et al. Incidence and clinical pattern of the abdominal compartment syndrome after "damage control" laparotomy in 311 patients with severe abdominal and/or pelvic trauma. Crit Care Med 2000;28:1747–53.
18. Cheatham ML, Malbrain ML, Kirkpatrick A, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. Part II Recommendations. Intensive Care Med 2007;33:951–62.
19. Bloomfield GL, Ridings PC, Blocher CR, et al. A proposed relationship between increased intraabdominal, intrathoracic and intracranial pressure. Crit Care Med 1997;25:496–503.
20. Josephs L, McDonald J, Birkett D, et al. Diagnostic laparoscopy increases intracranial pressure. J Trauma 1994;36:815–9.
21. Bloomfield GL, Ridings PC, Blocher CR, et al. Effects of increased intra-abdominal pressure upon intracranial and cerebral perfusion pressure before and after volume expansion. J Trauma 1996;40:936–40.
22. Scalea TM, Bochicchio GV, Habashi N, et al. Increased intra-abdominal, intrathoracic, and intracranial pressure after severe brain injury: multiple compartment syndrome. J Trauma 2007;62:647–56(discussion 656).
23. Joseph DK, Dutton RP, Aarabi B, Scalea TM. Decompressive laparotomy to treat intractable intracranial hypertension after traumatic brain injury. J Trauma 2004;57:687–93 (discussion 693–5).
24. Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 1976;20:401–4.
25. Kasthan J, Green JF, Parsons EQ, et al. Hemodynamic effects of increased abdominal pressure. J Surg Res 1981;30:249–55.
26. Robotham JL, Wise RA, Bomberger-Barnea B. Effects of changes in abdominal pressure on left ventricular performance and regional blood flow. Crit Care Med 1985;12:803–8.
27. Ridings PC, Blocher CR, Sugerman HJ. Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. J Trauma 1995;39:1071–5.
28. Cullen DJ, Coyle JP, Teplick R, Long MC. Cardiovascular, pulmonary and renal effects of massively increased intra-abdominal pressure in critically ill patients. Crit Care Med 1989;17:118–21.
29. Richards WO, Scovill W, Shin B, et al. Acute renal failure associated with increased intra-abdominal pressure. Ann Surg 1983;197:183–7.
30. Harmann PK, Kron IL, McLachlan HD, et al. Elevated intra-abdominal pressure and renal function. Ann Surg 1982;196:594–7.
31. Sugrue M, Buist MD, Hourihan F, et al. Prospective study of intra-abdominal hypertension and renal function after laparotomy. Br J Surg 1995;82:235–8.
32. Diebel LN, Dulchavsky SA, Wilson RF. Effects of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. J Trauma 1992;33:45–9.
33. Rasmussen IB, Berggren U, Arvidsson D, et al. Effects of pneumoperitoneum on splanchnic hemodynamics: an experimental study in pigs. Eur J Surg 1995;161:819–24.
34. Sugrue M, Jones F, Lee A, et al. Intraabdominal pressure and gastric intramucosal pH: Is there an association? World J Surg 1996;20:988–91.
35. Biffl WL, Moore EE, Burch JM. Femoral arterial graft failure caused by the secondary abdominal compartment syndrome. J Trauma 2001;50:740–2.
36. Kosir R, Moore FA, Selby JH, et al. Acute lower extremity compartment syndrome (ALECS) screening protocol in critically ill trauma patients. J Trauma 2007;63:268–75.
37. Moore EE, Thomas G. Orr memorial lecture: staged laparotomy for the hypothermia, acidosis and coagulopathy syndrome. Am J Surg 1996;172:405–10.
38. Balogh Z, McKinley BA, Cocanour CS, et al. Secondary abdominal compartment syndrome: an elusive complication of traumatic shock resuscitation. Am J Surg 2002;184:538–43.
39. Gonzalez EA, Moore FA, Holcomb JB, et al. Fresh frozen plasma should be given earlier to patients requiring massive transfusion. J Trauma 2007;62:112–9.
40. Suliburk JW, Ware DN, Balogh Z, et al. Vacuum-assisted wound closure achieves early fascial closure of open abdomens after severe trauma. J Trauma 2003;55:1155–60 (discussion 1160–1).
41. Balogh Z, McKinley BA, Cocanour CS, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. Arch Surg 2003;138:637–42 (discussion 642–3).
42. Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. Surg Clin North Am 1996;76:833–42.
43. Balogh Z, McKinley BA, Cocanour CS, et al. Patients with impending abdominal compartment syndrome do not respond to early volume loading. Am J Surg 2003;186:602–7discussion 607–8.
44. Corcos AC, Sherman HF. Percutaneous treatment of secondary abdominal compartment syndrome. J Trauma 2001;51:1062–4.
45. Jensen AR, Hughes WB, Grewal H. Secondary abdominal compartment syndrome in children with burns and trauma: a potentially lethal complication. J Burn Care Res 2006;27:242–6.
46. Tuggle D, Skinner S, Garza J, et al. The abdominal compartment syndrome in patients with burn injury. Acta Clin Belg Suppl 2007;1:136–40.
47. Leppäniemi AK, Hienonen PA, Siren JE, et al. Treatment of abdominal compartment syndrome with subcutaneous anterior abdominal fasciotomy in severe acute pancreatitis. World J Surg 2006;30:1922–4.
48. De Waele JJ, Hoste EA, Malbrain ML. Decompressive laparotomy for abdominal compartment syndrome – a critical analysis. Crit Care 2006;10:R51.
49. Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Abdominal compartment syndrome: it's time to pay attention. Intensive Care Med 2006;32:1912–4.
50. Balogh Z, Jones F, D'Amours S, et al. Continuous intra-abdominal pressure measurement technique. Am J Surg 2004;188:679–84.

Address for Correspondence
Prof. Zsolt Balogh MD, PhD, FRACS
Division of Surgery
Department of Traumatology
John Hunter Hospital and University of Newcastle
Locked Bag 1
Hunter Region Mail Center
Newcastle
NSW 2310
Australia
Phone (+61/2) -49214259, Fax -49214274
e-mail: zsolt.balogh@hnehealth.nsw.gov.au