Chapter 23
The Abdominal Compartment Syndrome

Dieter G. Weber and Zsolt J. Balogh

23.1 Introduction

The abdominal compartment syndrome (ACS) is a challenging clinical condition that may develop in a wide variety of clinical settings and which is associated with significant morbidity and mortality. The clinical syndrome was researched and described over one century ago [1], though practical management strategies in surgical specialties appeared only recently: Paediatric surgeons recognised problems with intra-abdominal pressure

D.G. Weber, MBBS (Hons.), FRACS
Departments of Trauma and General Surgery, Royal Perth Hospital, The University of Western Australia and The University of Newcastle, Newcastle, Australia
e-mail: dieter.weber@health.wa.gov.au

Z.J. Balogh, MD, PhD, FRACS, FACS (✉)
Department of Traumatology, Division of Surgery, John Hunter Hospital, The University of Newcastle, Lookout Rd,
New Lambton Heights, NSW 2305, Australia
e-mail: zsolt.balogh@hnehealth.nsw.gov.au

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after the forced closure of omphalocoeles [2, 3], vascular surgeons recognised this entity following emergency aortic aneurysm repairs [4] and trauma surgeons experienced a high incidence during the initial experiences with damage control laparotomies [5–8]. More recently, these experiences have been applied into the field of emergency general surgery [9, 10].

Consensus definitions of the ACS and associated entities were published in 2006, and revised in 2013, by the World Society of the Abdominal Compartment Syndrome [9]. The ACS is defined as a sustained intra-abdominal pressure >20 mmHg and which is associated with new organ dysfunction/failure. Sustained intra-abdominal pressures of ≥12 mmHg are considered to represent intra-abdominal hypertension, and graded (I: 12–15 mmHg, II: 16–20 mmHg, III: 21–25 mmHg, and IV: >25 mmHg) [9]. No organ dysfunction is present in the case of pure intra-abdominal hypertension [9].

For both research and applied clinical practice, measurement of intra-abdominal pressure has been standardised by manometry of the intravesical, bladder pressure. This is referenced at the level of the mid-axillary line, following maximal instillation of 25 mL of sterile saline, with the patient in the supine position, at end expiration, and in the absence of abdominal muscle contraction [9].

23.2 Aetiology and Risk Factors

An ACS may be of a primary or secondary cause: Primary ACS refers to the syndrome arising in the presence of an inciting condition associated with injury or disease in the abdomino-pelvic region, while secondary ACS occurs in conditions not originating in the abdomino-pelvic region. In secondary ACS, the pathophysiological processes in the abdominal cavity appear to be the consequence and result of events elsewhere [9, 11].

Patients affected by ACS are typically critically ill and admitted in an intensive care environment. While all such patients are potentially at risk of the syndrome, patients usually exhibit one or more of the following risk factors: (1) reduced abdominal compliance (e.g. major burns, trauma, intra-abdominal inflammation), (2) increased intraluminal contents (e.g. intestinal ileus), (3) increased intra-abdominal contents (e.g. intra-abdominal sepsis, ascites, etc.), (4) local or generalised oedema due to permeability changes secondary to inflammation and infection and (5) some potential iatrogenic effects from resuscitation, intensive therapy or surgical interventions [2]. In addition, age, obesity, abdominal surgery, certain patient positions, mechanical ventilation and other manoeuvres resulting in increased intra-thoracic pressure, bacteriaemia, sepsis, metabolic derangement and shock have all been linked with the development of ACS [9, 12]. A recent meta-analysis identified large volume crystalloid resuscitation, the respiratory status of the patient and shock as common risk factors for ACS among a heterogeneous group of inciting pathologies. In this review, obesity, sepsis, abdominal surgery, and large volume fluid resuscitation were risk factors achieving statistical significance.

23.3 Pathophysiology

Fundamental in the development of an ACS, both primary and secondary, is a complex cellular and tissue oedema and hypoxia [11]. In turn, organ dysfunction ensues. With the deteriorating organ function, a viscous cycle develops, further adding to the cellular injury and inflammatory response. Excessive and injudicious crystalloid resuscitation has been particularly implicated in further compounding this cycle [13, 14].

The effects of the ACS on the body are extensive and widespread [11]. In the brain, increased venous pressure is measured.

In the heart, reduced cardiac return due to *vena cava* compression in association with peripheral venous pooling in the lower extremities and torso is seen. Cardiac contractility is reduced by direct compression, and increased cardiac effort is required due to changes in afterload. The thoracic volume is reduced and pulmonary ventilation impaired. The precarious ventilator situation is further complicated by the high risk of an acute lung injury/adult respiratory distress syndrome. Hepatic dysfunction and renal impairment are the result of direct vascular and tissue compression, as well as the inflammatory organ crosstalk. Altered and dysfunctional neuro-hormonal axes are also implicated. In the kidney, the post-renal urinary obstruction does not completely account for the organ's dysfunction; ureteric stenting and renal pelvis decompression does not lead to major clinical improvement. In the intestine, numerous changes in blood flow, mucosal integrity and bacterial translocation are seen. All these changes by the ACS effectively perpetuate the viscous cycle responsible for the secondary ACS.

Most non-traumatic, emergency general surgical conditions leading to an ACS are of a primary nature (i.e. abdomino-pelvic pathological origin) [15]. The location of the primary stimulus necessitates effective source control and potential removal of pathological volume as well as the management of the ACS within the same anatomical region. Rapid attention to both these endeavours is critical to facilitate interruption of the futile cycle. Secondary ACS could present in acute general surgical cases when extra-abdominal bleeding or sepsis requires massive resuscitation.

23.4 Epidemiology

In initial studies, the incidence of ACS in major trauma patients was higher than one-third [16–18], though more

recent experience suggests this has much reduced during the last two decades [11]. The combination of modern resuscitation strategies, management of coagulopathy, damage control surgical strategies and modern anaesthesia and intensive care has been credited with this reduction in incidence. However, while the incidence has reduced, if untreated, the condition remains highly lethal, with mortalities in excess of 50 % [8]. Reported reductions in the mortality rate of ACS reflect the more timely diagnosis and treatment.

Early studies during the 2000s, into the prevalence of ACS among non-traumatic emergencies (both abdominal and elsewhere), suggested the presence of intra-abdominal hypertension in greater than 50 % of intensive care patients, and ACS around 2–5 % of patients. Similar to trauma patients, a high mortality rate was reported [19, 20]. Approximately two-thirds of patients experienced a primary ACS [15]. While the benefits of modern critical care and resuscitative strategies may predict a reducing incidence in recent years, alarming reports of continued high prevalence may be found. A recent series from the Netherlands on pancreatitis reports all patients at their institution experiencing intra-abdominal hypertension, and almost half fulfilling the diagnostic criteria for an ACS [21], and almost equally high incidences were reported in a Belgian series on severe burns patients [22].

23.5 Clinical Presentation

As most patients developing an ACS are unable to communicate symptoms due to their critical illness, or sedation for ventilation, symptoms are rarely reported. On examination, most patients will have a distended abdomen, though the sensitivity and specificity of this sign is poor [23–25]. Signs of organ dysfunction, particularly of renal and pulmonary, are common. Imaging is

largely unhelpful (though there may be evidence of the secondary effects of the increased intra-abdominal pressures: *vena cava* compression, renal compression, bowel oedema, abdominal wall herniation, lung atelectasis and reduced volume, etc.) [26].

An ACS may rapidly develop (over hours), and a high clinical level of suspicion is required for its timely diagnosis. Ideally, continuous screening on all patients at risk could be obtained; unless active screening for ACS is undertaken, the diagnosis will likely be missed early in its clinical course. Due to practical and operational constraints, most centres will rely on intermittent measurement, using the standardised technique of urinary bladder pressure, on patients clinically thought to be at risk. Abnormal measurement results must then trigger a timely clinical response. Regretfully, delayed decision-making and tardy decompression are common, and associated with poor outcomes [8].

The precise clinical course will vary depending on the underlying cause of the ACS. For example, the clinical course of an ACS that develops in the setting of severe acute pancreatitis is different to that seen if an ACS develops in the setting of a small bowel obstruction. In acute pancreatitis, the ACS may develop as a primary phenomenon due to the pancreatic phlegmon and oedema in the retroperitoneal tissues. It may also occur as a secondary ACS, due to the systemic inflammatory response that develops during the first day or so of severe acute pancreatitis, in combination with the chosen fluid resuscitation strategy. In an adhesive small bowel obstruction, an ACS may develop from critical distension, and provide an absolute surgical indication, even though the adhesive small bowel obstruction may have otherwise settled. As another example, a septic phlegmon from acute sigmoid diverticulitis may cause a primary ACS both from the inflammatory phlegmon, associated retroperitoneal oedema, potential space occupied by abscess formation, and from the possible large bowel obstruction, or due to secondary events due to injudicious fluid resuscitation, or an associated severe systemic inflammatory phenomenon. Each of these clinical

cases is fundamentally different, and the early presentation will be dominated in the underlying pathology. However, in all cases, an acute deterioration occurs with the onset of physiologic compromise associated with the ACS. In all cases, an acute surgical indication to deal with the ACS develops, though in addition to the abdominal decompression, the patient will need a tailored strategy to address the underlying cause. In most cases, the patient's acute physiologic compromise will dictate a damage control operative strategy [10]. The type of abdominal closure will also depend on the clinical situation, and is influenced by the primary pathology and its treatment strategy, the severity (and success of treatment) of the ACS, as well as patient and situational factors.

23.6 Treatment

Surgical decompression of the abdomen is the ultimate definitive manoeuvre in the treatment of an ACS [9]. An immediate decrease in the abdominal pressures is realised, and the decompression correlates well with returned organ function [27–29]. However, the invasive procedure is associated with significant morbidity and mortality [9, 11, 28].

The surgical decompression of the peritoneal cavity is usually performed through a midline laparotomy. This incision may also serve treatment of the underlying cause of the compartment syndrome, such as control of a septic source control, evacuation of ascites or control of haemorrhage, depending on the situation. However, other incisions, such as subcostal and transverse, are also described. Furthermore, endoscopic release of the linea alba, with maintenance of skin integrity, has also been successfully described [29]. The intact skin may assist reducing septic complications from a formal laparostomy; this is of particular relevance in acute pancreatitis with otherwise exposed necrotic tissues.

In conjunction with surgical decompression, various supportive adjuncts deserve consideration [9]. These less invasive therapies may avoid the need for decompression in cases of mild intra-abdominal hypertension, and where the primary cause for the potential ACS can be rectified early in the clinical course. These therapies include: sedation and analgesia, neuromuscular blockade, body positioning, nasogastric and colonic decompression, evacuation of ascites or other intra-peritoneal fluids, promotility agents, enhanced ratios of plasma/packed cells during the resuscitation of major haemorrhage, and a fluid balance protocol to attempt to avoid positive cumulative balance. Each of these strategies has been suggested by the recent consensus guidelines [9]. Diuretics, renal replacement therapies and albumin administration are not sufficiently studied for a consensus recommendation [9]. Judicious and considered use of traditional intensive care interventions used to support organ dysfunction may minimise potentially deleterious effects of these therapies on the pathophysiological processes causing the compartment syndrome in the first place [30, 31].

For trauma, prophylactic use of a temporary laparostomy in conjunction with a damage control surgical strategy is relatively accepted [32, 33]. However, for non-traumatic, emergency general surgical laparotomies, consensus does not support such prophylactic application in the absence of specific concerns [9]. This approach reflects modern, tailored surgical strategy, balancing the aggressiveness of the index operation, the need for a subsequent surgical procedures and the strength of the indication for a damage control strategy, in light of the quality of the resuscitation and intensive medical care received, and the unique patient and situational factors that define the individual case.

The management of the open abdomen is challenging, complex, highly individualised and is beyond the scope of this chapter. Central in this management is the ability to achieve primary fascial closure; early closure may minimise many of

the long-term complications seen with this technique. Negative pressure wound management systems, both proprietary and home-made, are associated with improved primary fascial closure rates, and appear well tolerated by both the patient and staff [34–36]. The negative pressure may facilitate removal of pro-inflammatory cytokines and fluids from the peritoneal space [34–37]. Unfortunately, some concerns regarding enteric fistulisation rates remain.

23.7 Conclusion

ACS is a highly lethal condition, typically encountered in critically ill surgical patients. The epidemiology of the disease continues to change, particularly regarding a reduced incidence attributable to the evolution of modern resuscitation practices, damage control surgical strategies and an increasing understanding of the entity. A high index of suspicion and associated accurate and timely diagnosis is critical in the management of the ACS. The lessons learnt from the clinical experiences in trauma surgery have extended into emergency general surgery, and are applicable to both primary and secondary ACS. In the situation of non-traumatic abdominal emergencies, the ACS is commonly primary in nature, and separate attention to address this pathology will be required; source control of sepsis, and/or haemorrhage control, is essential, and requires careful incorporation into the overall clinical strategy. Surgical decompression remains the ultimate therapeutic intervention.

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References

1. Schein M. Historical background. In: Ivatury R, Cheatham M, Malbrain M, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Biosciences; 2006. p. 1–07.
2. Wesley JR, Drongowski R, Coran AG. Intragastric pressure measurement: a guide for reduction and closure of the silastic chimney in omphalocele and gastroschisis. J Pediatr Surg. 1981;16:264–70.
3. Hershenson MB, Brouillette RT, Klemka L, Raffensperger JD, Poznanski AK, Hunt CE. Respiratory insufficiency in newborns with abdominal wall defects. J Pediatr Surg. 1985;20:348–53.
4. Fietsam R, 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.
5. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Ann Surg. 1984;199:28–30.
6. Burch JM, Ortiz VB, Richardson RJ, Martin RR, Mattox KL, Jordan GL. Abbreviated laparotomy and planned reoperation for critically injured patients. Ann Surg. 1992;215:476–83.
7. Stone HH, Strom PR, Mullins RJ. Management of the major coagulopathy with onset during laparotomy. Ann Surg. 1983;197:532–5.
8. Morris JA, Eddy VA, Blinman TA, Rutherford EJ, Sharp KW. The staged celiotomy for trauma. Issues in unpacking and reconstruction. Ann Surg. 1993;217:576–84.
9. Kirkpatrick AW, Roberts DJ, de Waele J, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines form the World Society of the Abdominal Compartment Syndrome. Intensive Care Med. 2013;39:1190.
10. Weber DG, Bendinelli C, Balogh ZJ. Damage control surgery for abdominal emergencies. Br J Surg. 2014;101:e109.
11. Balogh ZJ, Lumsdaine W, Moore EE, Moore FA. Post injury abdominal compartment syndrome: from recognition to prevention. Lancet. 2014;384:1466.
12. Holodinsky JK, Roberts DJ, Ball CG, et al. Risk factors for intra-abdominal hypertension and abdominal compartment syndrome among adult intensive care unit patients: a systematic review and meta-analysis. Crit Care. 2013;17:R249.
13. Balogh Z, McKinley BA, Cocanour CS, et al. Secondary abdominal compartment syndrome is an elusive early complication of traumatic shock resuscitation. Am J Surg. 2002;184:538.

14. Balogh Z, McKinley BA, Holcomb JB, et al. Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. J Trauma. 2003;54:848.
15. Reintam A, Parm P, Kitus R, et al. Primary and secondary intra-abdominal hypertension – different impact on ICU outcome. Intensive Care Med. 2008;34:1624.
16. Ivatury RR, Porter JM, Simon RJ, Islam S, John R, Stahl WM. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma. 1998;44:1016–21.
17. 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.
18. Raeburn CD, Moore EE, Biffl WL, et al. The abdominal compartment syndrome is a morbid complication of post injury damage control surgery. Am J Surg. 2001;182:542–6.
19. Malbrain ML, Chiumello D, Pelosi P, et al. Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple centre epidemiological study. Crit Care Med. 2005;33:315.
20. Malbrain ML, Chiumello D, Pelosi P, et al. Prevalence of intra-abdominal hypertension in critically ill patients: a multicenter epidemiological study. Intensive Care Med. 2004;30:822.
21. Smit M, Buddingh KT, Bosma B, et al. Abdominal compartment syndrome and intra-abdominal ischemia in patients with severe acute pancreatitis. World J Surg. 2016;40:1454–61.
22. Wise R, Jacobs J, Pilate S, et al. Incidence and prognosis of intra-abdominal hypertension and abdominal compartment syndrome in severely burned patients: pilot study and review of the literature. Anaesthesiol Intensive Ther. 2015;48:95–109.
23. Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. Intensive Care Med. 2006;32:1722.
24. 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.
25. Sugrue M, Bauman A, Jones F, et al. Clinical examination is an inaccurate predictor of intraabdominal pressure. World J Surg. 2002;26:1428.
26. Pickhardt PJ, Shimony JS, Heiken JP, et al. The abdominal compartment syndrome: CT findings. AJR Am J Roentgenol. 1999;173:575.
27. De Waele J, Desender L, De Laet I, et al. Abdominal decompression for abdominal compartment syndrome in critically ill patients: a retrospective study. Acta Clin Belg. 2010;65:399.

28. De Waele JJ, Hoste EA, Malbrain ML. Decompressive laparotomy for abdominal compartment syndrome – a critical analysis. Crit Care. 2006;10:R51.
29. De Waele JJ, Kimball E, Malbrain M, et al. Decompressive laparotomy for abdominal compartment syndrome. Br J Surg. 2016;103:709.
30. Sugrue M, D'Amours S. The problems with positive end expiratory pressure (PEEP) in association with abdominal compartment syndrome (ACS). J Trauma. 2001;51:419.
31. Burch JM, Moore EE, Moore FAC, Franciose R. The abdominal compartment syndrome. Surg Clin North Am. 1996;76:833.
32. Kirkpatrick AW, Ball CG, D'Amours SK, Zygun D. Acute resuscitation of the unstable adult trauma patient: bedside diagnosis and therapy. Can J Surg. 2008;51:57.
33. Sugrue M, D'Amours SK, Joshipura M. Damage control surgery and the abdomen. Injury. 2004;35:642.
34. Dalfino L, Tullo L, Donadio I, et al. Intra-abdominal hypertension and acute renal failure in critically ill patients. Intensive Care Med. 2008;34:707.
35. Miller RP, Thompson JT, Faler BJ, et al. Late facial closure in lieu of ventral hernia; the next step in open abdomen management. J Trauma. 2002;53:843.
36. Miller RP, Meredith JW, Johnson JC, et al. Prospective evaluation of vacuum assisted fascial closure after open abdomen: planned ventral hernia rate is substantially reduced. Ann Surg. 2004;239:608.
37. Batacchi S, Matano S, Nella A, et al. Vacuum assisted closure device enhances recovery of critically ill patients following emergency surgical procedures. Crit Care. 2009;13:R194.