The secondary abdominal compartment syndrome: not just another post-traumatic complication

Chad G. Ball, MD, MSc;* Andrew W. Kirkpatrick, MD;†‡ Paul McBeth, MD‡

The secondary abdominal compartment syndrome (ACS) is defined as the presence of organ dysfunction with concurrent intra-abdominal hypertension (IAH) in a scenario lacking primary intraperitoneal injury or intervention. This state appears to be related to visceral, abdominal wall and retroperitoneal edema and ascites induced by resuscitation. Despite a diverse range of associated causes such as pancreatitis, intra-abdominal sepsis, cardiac arrest, thermal injury and extraperitoneal trauma, this class of ACS is characterized by the presence of shock requiring aggressive fluid resuscitation. Secondary ACS is an extreme condition along a continuum of raised intra-abdominal pressure (IAP) that is pathoneumonic when associated with new overt organ failure. When IAP is above normal but is not associated with organ failure, IAH is diagnosed. Because these conditions are common among critically ill patients, the measurement of IAP is crucial. It is unclear whether preventing IAH reduces progression to ACS or influences outcomes. When overt ACS is confirmed, immediate surgical decompression of the patient's abdomen via a standard laparotomy is usually required. Because many disease processes resulting in critical illness require aggressive fluid resuscitation as a primary therapy, it is likely that secondary ACS is much more common than previously believed. Further study is needed.

Le syndrome du compartiment abdominal (SCA) secondaire s'entend de la présence d'un dysfonctionnement organique conjugué à une hypertension intra-abdominale (HIA) dans un scénario où il n'y a pas d'intervention ou de traumatisme intrapéritonéal primaire. Cet état semble relié à la paroi abdominale des viscères, ainsi qu'à l'œdème rétropéritonéal et à l'ascite provoqués par la réanimation. En dépit d'un éventail diversifié de causes connexes comme la pancréatite, un sepsis intra-abdominal, l'arrêt cardiaque, le traumatisme thermique et le traumatisme extrapéritonéal, cette catégorie de SCA est caractérisée par la présence d'un choc qui nécessite une réanimation liquidienne agressive. Le SCA secondaire est un problème extrême dans un continuum d'élévation de la pression intra-abdominale (PIA) qui est pathopneumonique lorsqu'il est associé à une nouvelle défaillance organique évidente. Lorsque la PIA dépasse la normale mais n'est pas associée à une défaillance organique, on diagnostique une HIA. Comme ces problèmes sont courants chez les patients en phase critique, il est crucial de mesurer la PIA. On ne sait pas clairement si la prévention de l'hypertension intra-abdominale atténue l'évolution en SCA ou a un effet sur les résultats. Lorsque l'on confirme un SCA évident, il faut habituellement procéder sur-le-champ à la décompression chirurgicale de l'abdomen du patient par laparotomie ordinaire. Comme beaucoup de processus morbides qui aboutissent à une maladie critique nécessitent une réanimation liquidienne agressive comme thérapie de première intention, il est probable que le SCA secondaire est beaucoup plus courant qu'on le croyait auparavant. Une étude plus poussée s'impose.

A compartment syndrome is a condition of increased pressure in a confined anatomic space that adversely affects the circulation and threatens the function and viability of the tissues therein. This may arise in any closed compartment within the body. Secondary abdominal compartment syndrome (ACS) is characterized by the presence of shock requiring aggressive fluid resuscitation. Because many disease

From the *Departments of Trauma, Surgery and Critical Care, Grady Memorial Hospital, Emory University, Atlanta, Ga., and the †Department of Critical Care Medicine and the Regional Trauma Program, and the ‡Department of Surgery, Foothills Medical Centre, University of Calgary, Calgary, Alta.

Correspondence to: Dr. A.W. Kirkpatrick, Department of Surgery, Foothills Medical Centre, 1403-29th Street NW, Calgary AB T2N 2T9; fax 403 944-1277; andrew.kirkpatrick@calgaryhealthregion.ca

processes resulting in critical illness require aggressive fluid resuscitation as a primary therapy, it is likely that this condition is much more common than previously believed.

Background

The World Society of the Abdominal Compartment Syndrome (WSACS) defines ACS as a sustained increase in intra-abdominal pressure (IAP) greater than 20 mm Hg concurrent with a new onset of organ dysfunction.[1] This state of organ failure frequently affects the cardiovascular, respiratory and renal systems.[2–8] The condition is uniformly fatal if left untreated.[2–8]

It is widely understood that ACS represents the end stage of a patho-physiologic spectrum that begins with normal IAP, proceeds to intra-abdominal hypertension (IAH) and ends with overt ACS. To standardize definitions and facilitate communication and research, the WSACS recently outlined working definitions of the conditions.[1,9] They also published evidence-based guidelines for the diagnosis, measurement, management and prevention of IAH and ACS. These documents will be revised regularly and are intended to provide guidance to clinicians.

Historically, ACS was often diagnosed when the effects of IAH had become overtly obvious. The signs included severe respiratory distress, elevated peak airway pressures, hypotension, diminished cardiac output and oliguria.[2,3,6] Diagnosing IAH and ACS at this point is clearly too late, as evidenced by an increasingly poor prognosis, infectious complications and death.[1–16] Because many of the effects of ACS are clinically indistinguishable from those of other common syndromes related to critical illness, it is probable that the influence of abnormal IAP is not infrequently missed in a critically ill patient with multifactorial complications. As a result, clinicians must possess a high index of suspicion and monitor IAP aggressively.

Definitions

The WSACS defines IAP as the pressure within the abdominal cavity, measured at end-expiration in a relaxed, supine patient.[1] It defines IAH as a sustained elevation of IAP greater than 12 mm Hg.[1] It further classifies IAH into grades I–IV (Box 1). The WSACS defines ACS as a sustained increase of IAP greater than 20 mm Hg that is associated with the onset of organ dysfunction.[1] Because abdominal perfusion pressure (APP) is potentially a more accurate predictor of visceral perfusion, and a theoretically superior outcome of resuscitation, it is often included in the definition of ACS (APP < 60 mm Hg).[1,17] This index is derived by subtracting the IAP from the mean arterial pressure, and it should be considered in relation to the overall physiologic status of the patient.

The ACS can also be subcategorized based on its causes. Primary or "surgical" ACS is associated with an injury or disease in the abdomino-pelvic region that requires surgical or angiographic intervention.[1] This is also considered to be "classic" ACS. Patients with primary ACS typically have intraperitoneal or retroperitoneal bleeding, solid organ injury, damage control surgery (e.g., packing of liver hemorrhage) or transplantation. Primary ACS also includes bleeding pelvic fractures.[1]

Secondary or "medical" ACS, the focus of our review, is a fundamentally unique entity because it occurs in patients without a primary intraperitoneal injury or intervention.[1] This terminology represents a significant departure from the same descriptors employed in the lexicon of peritonitis.[10]

Finally, tertiary ACS happens when ACS recurs despite attempts at prophylactic or therapeutic treatment of either primary or secondary IAH or ACS.[1,11] Examples may include persistent ACS despite surgical decompression or an entirely new episode of ACS after the fascia has been reapproximated following temporary abdominal closure.

Incidence and cause

IAH and ACS are diseases related to critical illness. Their incidence reflects both the acuity of the population and a clinician's diligence in measuring IAP. The reported incidence of IAH and ACS is about 32.1% and 4.2%, respectively, in the mixed intensive care unit (ICU) population.[12] Rates of IAH have also been reported for patients with severe burns (36.7%–70%)[4,7,13] and traumatic injuries (2%–50%)[6,14–16] and for patients who had major abdominal procedures (31.5%–40.7%).[14–16,18–21] Overt ACS occurs in 1%–31% of those with burns[4,7,13,22] and in 0.5%–36% of those with injuries.[15,16,23–29] An institution with both an aggressive fluid resuscitation protocol and diligent IAP monitoring reported an incidence of 8% for secondary ACS and an incidence of 6% for primary ACS in severely injured trauma patients who presented with shock.[18] When compared with patients with primary ACS, secondary IAH has also been shown to occur later in a patient's hospital admission and to be associated with more prolonged and severe elevations of IAP and with higher mortality.[30]

Box 1. World Society of the Abdominal Compartment Syndrome definitions of intra-abdominal hypertension and abdominal compartment syndrome	
Condition	Measured intra-abdominal pressure
Normal	< 12 mm Hg
IAH	
Grade I	12–15 mm Hg
Grade II	16–20 mm Hg
Grade III	21–25 mm Hg
Grade IV	> 25 mm Hg
ACS	> 20 mm Hg + new organ failure
	or
	APP* < 60 mm Hg + new organ failure

ACS = abdominal compartment syndrome;
APP = abdominal perfusion pressure;
IAH = intra-abdominal hypertension.
*Mean arterial pressure minus intra-abdominal pressure.

The occurrence of secondary ACS appears to be directly related to visceral, abdominal wall and retroperitoneal edema and ascites induced by resuscitation.[1,2,4,7–9,12,13,16,17,28,31–43] Despite a diverse range of associated conditions, secondary ACS is characterized by the presence of shock requiring aggressive fluid resuscitation (Box 2). This situation is compounded by the swelling of the intestinal wall and luminal distension that accompanies

Box 2. Risk factors for IAH/ACS
• Acidosis (pH < 7.2)
• Hypothermia (core temperature < 33°C)
• Polytransfusion (> 10 U packed red blood/24 h)
• Coagulopathy (platelets < 55 000/mm³, or activated partial thromboplastin time twice the normal level or higher, or prothrombin time > 15 s, or international standardized ratio > 1.5)
• Sepsis (American-European Consensus Conference definitions)
• Bacteremia
• Intra-abdominal infection/abscess
• Peritonitis
• Liver dysfunction/cirrhosis with ascites
• Mechanical ventilation
• Use of positive end expiratory pressure (PEEP) or the presence of auto-PEEP
• Pneumonia
• Abdominal surgery, especially with tight fascial closures
• Massive fluid resuscitation (> 5l colloid or crystalloid/24 h)
• Gastroparesis/gastric distention/ileus
• Volvulus
• Hemoperitoneum/ pneumoperitoneum
• Major burns
• Major trauma
• High body mass index (> 30 kg/m²)
• Intra-abdominal or retroperitoneal tumours
• Prone positioning
• Massive incisional hernia repair
• Acute pancreatitis
• Distended abdomen
• Damage control laparotomy
• Laparoscopy with excessive inflation pressures
• Peritoneal dialysis
IAH = intra-abdominal hypertension; ACS = abdominal compartment syndrome. Adapted from Malbrain et al.[1]

shock/resuscitation. The recognition that aggressive crystalloid fluid resuscitation in shock necessitates an obligatory loss of fluid to the intracellular or "third" space has nearly eliminated renal failure as a complication of burns.[44,45] Unfortunately this benefit must be balanced against the possibility that excess crystalloid resuscitation may contribute to an increased occurrence of IAH and, therefore, secondary ACS. The phenomenon of "fluid creep" is an important problem in modern burn care, and it likely applies to other areas of critical care.[46,47] This concept was most recently supported in a large pan-European ICU study of sepsis that showed a positive fluid balance as the strongest prognostic factor for death.[48]

Pathophysiology

The pathophysiology of secondary ACS affects the entire body and is identical to primary ACS. Cardiac output is reduced owing to decreased preload and right heart volumes. Although increased systemic vascular resistance initially maintains apparent blood pressure, decreases in preload from the pooling of blood in splanchnic and lower extremity vascular beds eventually lead to reduced central venous return.[3,35,49–53] Cardiac underfilling also occurs despite apparently increased central hemodynamic measurements (central venous pressure [CVP] and pulmonary artery occlusion pressure). As respiratory compliance decreases, mechanical ventilation with increased ventilatory pressures and decreased volumes becomes difficult.[54,55] The partial pressures of oxygen will decrease, and carbon dioxide will increase.[56,57] Even modest IAH appears to exacerbate acute lung injury and the acute respiratory distress syndrome (ARDS). When IAP levels greater than 20 mm Hg are applied to critically ill animals, a dramatic exacerbation of ARDS-associated pulmonary edema is evident.[58] Furthermore, elevated IAP results in a stiffer chest wall with much lower transpulmonary pres-

sures, and therefore less susceptibility to ventilator-induced lung injury.[59,60] Oliguria is a common manifestation of the ACS because renal failure and IAH are dose-dependant.[3,19,20] These effects are exaggerated by hypovolemia and positive end-expiratory pressure.[56,61]

Beyond the heart, lungs and kidneys, almost every other organ system is altered by IAH, even if the effects are not clinically overt. Also, IAH appears to contribute to increased intracerebral pressure (ICP) via transmitted intrathoracic pressure[62–65] to the extent that laparotomies have been reported to reduce ICP in patients with secondary ACS.[66] Patients in shock are at a particularly high risk for splanchnic malperfusion because even modest elevations in IAP greatly reduce hepatic and splanchnic perfusion.[67–70] This effect is exacerbated by prior hemorrhage[71] and is observed at much lower IAPs than required to induce other clinical features of ACS. As a result, subtle organ failure, concurrent to multisystem disease compatible with other causes, may be difficult to ascertain.

Clinical settings

Secondary ACS or IAH are typically described in patients with either physical or thermal injuries. Patients with burns are at a particularly high risk for secondary ACS when the burns cover 70% or more of the body surface area (BSA). Patients with smaller burns and concomitant inhalation injuries are also at risk for secondary ACS.[7,13,22,33] It should be noted that circumferential abdominal eschar is not a prerequisite for the condition.[7,22,33] As stated previously, patients with an intraperitoneal injury who develop IAH or ACS are classified as having primary IAH or ACS. However, if no intraperitoneal injury is present, IAH or ACS may still develop if massive fluid resuscitation is required to treat hemodynamic instability resulting from injuries that are anatomically distant from the peritoneal cavity.[49] Secondary visceral swelling from massive resuscitation is

especially common in patients with displaced pelvic fractures. This scenario is classified as secondary ACS if the pelvic hematoma is not the primary cause of IAH or ACS. These patients typically have hypothermia, coagulopathy and acidosis, and they require ongoing resuscitation even after admission to the ICU.[32] Those with secondary ACS were reported to have longer stays in the emergency department, receive substantially more crystalloid fluid and experience prolonged delays before undergoing therapeutic angiography.[28] These findings reinforce the need for early hemorrhage control rather than ongoing fluid resuscitation.

In addition, IAH and ACS likely influence the clinical course of many critically ill patients with sepsis. This is a result of both primary intraperitoneal disease and the massive fluid resuscitation that is often required to stabilize hemodynamics in patients with severe sepsis or septic shock. Severe sepsis is a leading cause of death in ICUs throughout the world, with mortality approaching 30%[72,73] and an incidence that has nearly doubled over the last 11 years.[74] Recently updated international guidelines continue to prioritize fluid resuscitation to obtain a CVP between 8 and 12 mm Hg.[75] This typically requires aggressive fluid administration during the first 24 hours of management.[76] If IAH is recognized, even higher targets are required. Whether this increased emphasis on early (the period of maximal risk for secondary ACS) aggressive fluid resuscitation in the case of sepsis will lead to an increased incidence of IAH, and hence secondary ACS, remains unknown. In our own ICU, we detected IAH (IAP > 12 mm Hg) in 87% of study participants with sepsis.[77] Furthermore, elevated IAP correlated with organ dysfunction, the need for continuous renal replacement therapy and mortality.[77] This is supported by similar observations,[12,78] including a recent report that noted sepsis to be the leading cause (39%) of secondary IAH.[30]

Current indications and techniques to measure intra-abdominal pressure

Clinical examination has repeatedly been shown to be inaccurate in detecting elevated IAP.[6,79] Although numerous modalities are available, the current gold standard technique for noninvasive measurements uses the patient's urinary bladder for pressure transduction.[19,80–83] It is recommended that the patient be supine and that the transducer be zeroed at the iliac crest in the midaxillary line.[1] We believe a given clinician can select any technique, as long as it is familiar to ICU nursing staff and easily repeated in the daily care of a patient.

Indications for measuring IAP should include all risk factors for the development of IAH or ACS. These indications are numerous and have recently been summarized by the WSACS.[1] We feel it is prudent to screen all critically ill patients upon admission to an ICU, or as a minimum, all those at risk for IAH and ACS. In the case of secondary ACS, patients at high risk include but are not limited to those with abdominal infection (e.g., pancreatitis, peritonitis), thermal injury and intraperitoneal or retroperitoneal hemorrhage, as well as any patient who has received large volumes of fluid resuscitation.[1] Although authors have become increasingly conservative with the threshold volumes of crystalloid considered to be significant,[7,12,22,29,34,35] there does not appear to be a single predictive factor that is reliable and accurate in predicting secondary IAH or ACS other than critical illness. Studies performed at the University of Colorado have noted that the time required to diagnose secondary ACS in nontrauma patients was twice that required in patients with injuries, likely owing to a decreased awareness of this phenomenon outside of trauma.[35,49] Given the evidence that IAH is an independent predictor of mortality, these concepts can no longer be ignored in critically

ill patients.[4,7,12,22,25,29,32,33,50] Infrequent monitoring of IAP among patients in the ICU is in many ways synonymous to interpreting hemodynamic indices without considering thoracic pressures (i.e., peak and plateau airway and positive end expiratory pressures) and must be avoided.[84,85]

Management

The WSACS has outlined a suggested management strategy for IAH/ACS that is largely derived from consensus and retrospective data.[9] The association currently recommends surgical decompression for secondary ACS when it is refractory to medical treatment options. It remains highly controversial among clinicians as to whether a particular level of IAH should automatically necessitate surgical decompression in the absence of associated organ failure. Most surgeons are reluctant to perform laparotomies in patients who do not meet the complete definition of secondary ACS.[1,9,86,87] Although the current gold standard for overt ACS is surgical decompression of the abdomen via a laparotomy, alternative medical strategies have been described in the treatment of IAH. Although there are no randomized prospective data, authors have suggested using hypertonic crystalloid and colloid-based resuscitation,[1,88,89] neuromuscular blockade, body positioning, percutaneous catheter decompression, sedation, analgesia, nasogastric and colonic decompression, escharotomy or tangential burn wound excision, externally applied continuous negative abdominal pressure devices, and the careful use of diuretics and continuous renal replacement therapies once the initial resuscitation is complete.[1,4,7,22,32,38,65,90] We recommend routine bowel decompression via nasogastric tubes, selective rectal decompression when colonic distension is documented and percutaneous removal of intra-abdominal fluid when demonstrated in patients with secondary ACS. When this fails and/or

ACS develops, we proceed with an emergent decompressive laparotomy.

Although laparotomy often resolves the ACS, a minority of patients (8%–16%) will not obtain a subsequent primary fascial closure.[91–94] In those who survive, the open abdomen is fraught with inherent complications that include fistulae, surgical site infections, sepsis, fluid and electrolyte imbalances from exposed bowel, prolonged ventilatory requirements and massive ventral hernia.[2,39,95] Because the management options are numerous, the appropriate therapy of this condition is becoming a distinct emerging discipline. It is therefore an oversimplification to state that these patients may require absorbable mesh interposition, skin grafting, nonabsorbable mesh and/or component separation repairs.

Summary

Secondary ACS is defined as the onset of organ failure with concurrent IAH in a patient who has not experienced an injury or intervention. This condition is directly related to visceral, abdominal wall and retroperitoneal edema and ascites induced by resuscitation. As a result, secondary ACS is typically characterized by the presence of shock requiring aggressive fluid resuscitation, and therefore includes patients with almost any form of critical illness. With an incidence approaching 30%, ACS can no longer be ignored. The measurement of IAP must occur more often than it is currently among critically ill patients. Furthermore, when overt ACS is confirmed, patients typically require immediate surgical decompression via a standard laparotomy. In conclusion,
- IAH and ACS are common;
- secondary ACS is defined as the onset of organ failure in a patient with IAH > 20 mm Hg;
- secondary ACS occurs in patients without abdominal trauma or intraperitoneal surgery;
- shock and aggressive fluid resuscitation are common among patients with secondary ACS;
- the most common treatment for secondary ACS is an emergent decompressive laparotomy; and
- monitoring IAP to identify ACS is crucial in all critically ill patients.

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