Abdominal Compartment Syndrome: A Decade of Progress

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Unlike many disease processes that surgeons treat in which progress is slow, the knowledge, definition, diagnosis, research, and treatment of abdominal compartment syndrome (ACS) have undergone dramatic improvements during the past decade. Considering that ACS was only well characterized and physiologically defined in the laboratory in 1985, and better clinically defined in 1989, rapid progress has occurred in both the diagnosis and treatment of this relatively young entity.[1,2] In fact, the mortality rate has dropped from 60% to between 34% and 37% in just the last 10 years.[3-5]

Abdominal compartment syndrome is defined as a sustained intra-abdominal pressure (IAP) >20 mmHg that is associated with new onset of organ dysfunction or failure.[6] Although often mistakenly used interchangeably, ACS is a separate and distinct entity from intra-abdominal hypertension (IAH), which is defined as a sustained or repeated pathologic elevation of the IAP ≥12 mmHg.[6] Intra-abdominal hypertension does not cause organ dysfunction, and that is the key difference between the 2 disease processes. Normal or mean IAP within the nondiseased abdominal cavity is between 2 and 5 mmHg, depending on overall body mass index (BMI), but can run as high as 12 mmHg in the obese adult[7] (Table 1). This chronic IAH does not produce organ injury and is purely a result of visceral obesity. It is also important to realize that normal IAP in the critically ill, who are almost always fluid overloaded and edematous, runs slightly higher, at 5 to 7 mmHg.[6]

Determining intra-abdominal pressure

The standardized method of determining IAP is by measuring the bladder pressure transmitted through a Foley catheter. This is correctly performed by clamping the catheter tubing beyond the rubber or plastic diaphragm that allows needle puncture access into the lumen of the catheter, and then instilling a maximum volume of 25 mL saline into the bladder.[6] Overdistending the bladder with excessive volumes of fluid will increase the bladder pressure and not reflect the true IAP.[8] The IAP should be measured at end expiration with the patient supine and relaxed or sedated, with the transducer zeroed at the mid-axillary line.[6] Having the patient slightly upright at >30 degrees has been clearly shown to produce erroneous results because IAP will increase as the head of the bed is elevated from 10 to 30 to 45 degrees.[9]

Once the IAP is measured, the abdominal perfusion pressure (APP) can be calculated. The APP is simply the mean arterial pressure minus the IAP. To maintain adequate perfusion to the viscera, an APP of at least 60 mmHg is desired.[6] For example, a critically ill patient with a mean arterial blood pressure of 80 mmHg and an IAP of 22 mmHg, has an APP of 58 mmHg, which is just below the critical level. This is when end-organ dysfunction can begin to occur.

Primary, secondary, and tertiary abdominal compartment syndrome

There are 3 different types of ACS: primary, secondary, and tertiary, which is also known as recurrent ACS. Primary ACS refers to ACS that occurs due to a primary intra-abdominal (or intrapelvic) cause, such as a ruptured abdominal aortic aneurysm, abdominal trauma, or retroperitoneal hemorrhage[10]; these are the most common causes. However, other rare conditions, such as malignant ascites, a giant ovarian tumor, a rectus sheath hematoma, and ACS after Roux-en-Y gastric bypass, have also been reported.[11-13] Almost any pathology that creates a space-occupying or expanding lesion within the abdominal or pelvic cavity can cause ACS.

Secondary ACS (also known as extra-abdominal compartment syndrome) refers to ACS that occurs as a result of massive bowel edema secondary to sepsis, capillary leak, conditions requiring massive fluid resuscitation, or burns.[14-16] Secondary ACS occurs most commonly after hemorrhagic shock requiring massive fluid resuscitation or severe burn injuries requiring massive fluid infusion. Although these situations result in total body anasarca, it is the swelling of the bowel that limits renal

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Abbreviations and Acronyms

ACS = abdominal compartment syndrome
APP = abdominal perfusion pressure
BMI = body mass index
IAH = intra-abdominal hypertension
IAP = intra-abdominal pressure
ICP = intracranial pressure
RVEDVI = right ventricular end diastolic volume index

perfusion and ventilation due to increased abdominal pressure. There is also an entity known as recurrent ACS (or tertiary ACS) in which ACS recurs after resolution of an earlier episode of either primary or secondary ACS. Recurrent ACS is relatively uncommon and tends to be the result of an overly aggressive attempt at abdominal closure in an edematous patient with an open abdomen.

Prevalence and pathophysiology

The prevalence of ACS is difficult to quantify and varies with the patient population that is studied and the acuity of those patients. In the general medical or surgical population, the incidence has been reported to be around 0.5% to 8%, but increases to 6% to 14% in trauma patients, depending on the classification of primary, secondary, or both.[14,17,18] The incidence is even higher in burn patients, occurring in 1% to 20%, depending on severity and percentage burn.[18]

The various cascades of molecular and cellular events that ultimately lead to ACS are multifactorial, with extensive interplay between the capillary membrane and interstitial interface, combined with neutrophil activation and hemodilution. I will now examine some of these models at the microvascular level. Regardless of which model is used as the example, the inciting event, whether it be trauma with hemorrhagic shock, sepsis, or burns, all lead to a capillary leak syndrome resulting in the extravasation of fluid into the interstitium and massive bowel wall edema. The bowel wall has an enormous capacity to hold fluid, and a patient can easily sequester several liters of fluid within the bowel wall and mesentery. This edema leads to increased pressure within the abdominal cavity, which ultimately leads to decreased renal perfusion, decreased bowel perfusion, and loss of ventilatory capacity.

Table 1. Grading System for Intra-Abdominal Hypertension

Grade	Internal pressure, mmHg	Description
Normal	<12	This accounts for the obese
1	12–15	
2	16–20	Unusual to have organ dysfunction in this range
3	21–25	Not equivalent to ACS unless organ dysfunction occurs
4	>25	With organ dysfunction the terminology changes to ACS

ACS, abdominal compartment syndrome.

A common cause of IAH and ACS is sepsis requiring massive fluid resuscitation. Clinical and basic science research has shown that interleukin-6, interleukin-8, and tumor necrosis factor–α all act directly on the endothelial cell of the capillary beds within the bowel wall to contribute to the capillary leak syndrome and bowel wall edema.[19,20] In addition, when the septic source is located within the abdomen, such as with appendicitis, the peritoneal fluid itself has been shown to contain elevated levels of interleukin-6 and tumor necrosis factor–α, which in turn prime neutrophils and fuel the inflammatory cascade and capillary leak.[21,22] *Neutrophil priming* refers to an enhanced and overly exaggerated secondary response to injury that occurs after an initial injury or stimulus.[23] Priming results in the neutrophil expressing certain adhesion molecules on its surface, specifically CD11b and CD18, which allows the neutrophil to adhere to the endothelial cell of the capillary and extravasate into the interstitium.[24,25]

Central vasodilation and shunting of blood from the periphery during sepsis result in hypotension, which is usually managed with a combination of fluid resuscitation and vasopressors. Vigorous fluid resuscitation during a phase of capillary leak alters the normal hydrostatic and oncotic pressures, resulting in the formation of intestinal edema because of a net efflux of fluid into the interstitium.[26] As the bowel edema worsens and IAP increases, upward pressure on the diaphragm increases central venous pressure, creating a pressure gradient against venous outflow and causing abdominal venous hypertension. The increased IAP also hinders lymphatic efflux of fluid from the abdomen, which only worsens the process.[26] All of these factors combining at once contribute to the growing edema within the bowel wall and mesentery, and IAP continues to increase. The septic patient who has been aggressively fluid resuscitated is at obvious risk for ACS.

Another common cause of IAH and ACS is abdominal trauma, leading to hemorrhagic shock requiring massive fluid resuscitation. In this model, the initial traumatic event leads to direct blood loss from vascular damage within the abdominal cavity. Whether the patient requires surgery to control the blood loss or is managed nonoperatively, the patient has lost a considerable amount of blood and requires either blood transfusion