Abdominal compartment syndrome

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Acute renal failure frequently occurs in the intensive care unit as a primary or secondary event in association with trauma, surgery, or comorbid medical disease. An increasingly common thread linking surgical and medical disease management is the abdominal compartment syndrome. In particular, the rise of early goal-directed therapy for the initial resuscitation and management of severe sepsis and septic shock is associated with an increased frequency of secondary abdominal compartment syndrome. This paper will explore the pathophysiology underpinning the abdominal compartment syndrome and its contribution to acute kidney injury and acute renal failure with regard to intra-abdominal pressure dynamics, preload limitation, and afterload augmentation. Diagnostic modalities and therapeutic interventions will be addressed as a means of reducing the frequency of acute kidney injury and acute renal failure in the critically ill. (Crit Care Med 2008; 36[Suppl.]:S212–S215)

KEY WORDS: abdominal compartment syndrome; intra-abdominal pressure; intra-abdominal hypertension; abdominal perfusion pressure; metabolic acidosis; hypovolemia; bladder pressure; decompressive laparotomy

Recognition of the abdominal compartment syndrome and its genesis, complications, and therapy has it roots in damage control trauma surgery for patients *in extremis* after injury. The term *damage control* was coined in 1993 by Dr. Rotondo and colleagues (1) at the University of Pennsylvania to describe a novel management strategy designed to abbreviate operative times for injured patients with nearly exsanguinating hemorrhage. While clearly saving many lives that would have otherwise been lost to prolonged operative interventions in the setting of hypothermia, acidosis, and coagulopathy, damage control techniques presaged an era of new management problems for the saved patients. These clinical challenges stemmed from increased intra-abdominal pressure, termed intra-abdominal hypertension (IAH). Excessive IAH leads to devastating abnormalities in diverse organ systems, many of which are readily discoverable with routine monitoring in the critical care unit, and all of which are related to decreased preload, increased afterload, and extrinsic compression, with decreased end-organ oxygen delivery and utilization. The resultant pressure-volume dysregulation syndrome is known as the abdominal compartment syndrome (ACS) (2). While originally described in trauma patients as the result of recurrent hemorrhage and visceral edema, the ACS is ubiquitous in nature and surfaces in both surgical and medical critical care units. This section will focus on the impact of IAH and the ACS on acute kidney injury and acute renal failure. To appreciate how IAH and the ACS influence renal function, it is essential to understand the pathophysiology that underpins the ACS. Unfortunately, the ACS and its consequences are not universally appreciated across different specialties, leading to disparate rates of recognition and therapy (3).

Intra-Abdominal Pressure and Intra-Abdominal Hypertension

In healthy individuals, a normal intra-abdominal pressure (IAP) is <5 to 7 mm Hg according to the consensus definition of the World Society of Abdominal Compartment Syndrome, and is generally interrogated as a patient's intravesical pressure (Fig. 1) (4). The upper limit of IAP is generally accepted to be 12 mm Hg by the World Society, reflecting the expected increase in normal pressure from clinical conditions that exert external pressure against the peritoneal envelope or diaphragm, including obesity and chronic obstructive pulmonary disease (5). In contrast, a constant increased pressure >12 mm Hg defines intra-abdominal hypertension. IAH may be conveniently divided into 4 grades (Table 1) that are further subdivided according to the rapidity of onset (6). The grades range from grade 1 (12–15 mm Hg) to grade II (16–20 mm Hg) to grade III (20–24 mm Hg) to grade IV (>25 mm Hg); the onset times range from chronic (which is rare) to acute to subacute to hyperacute. The vast majority of intra-abdominal hypertension that is associated with acute kidney injury or acute renal failure exceeds grade III and is associated with acute or hyperacute onset. Recognizing that host factors will interact with organ pressure–volume dynamics, an individualized approach may be entertained in determining the contribution of mean arterial pressure and IAP to organ blood flow. In a fashion similar to that of cerebral perfusion pressure, the interplay of inflow (mean arterial pressure) and egress pressure (IAP) is related via the abdominal perfusion pressure (7). The formula for abdominal perfusion pressure is as follows: abdominal perfusion pressure = mean arterial pressure − IAP (normal = 60 mm Hg). The abdominal perfusion pressure is useful in precisely defining the ACS.

Abdominal Compartment Syndrome

A sustained IAP >20 mm Hg and abdominal perfusion pressure <60 mm Hg occurring in association with a new and attributable organ dysfunction or failure describes the ACS (4). Table 1 depicts

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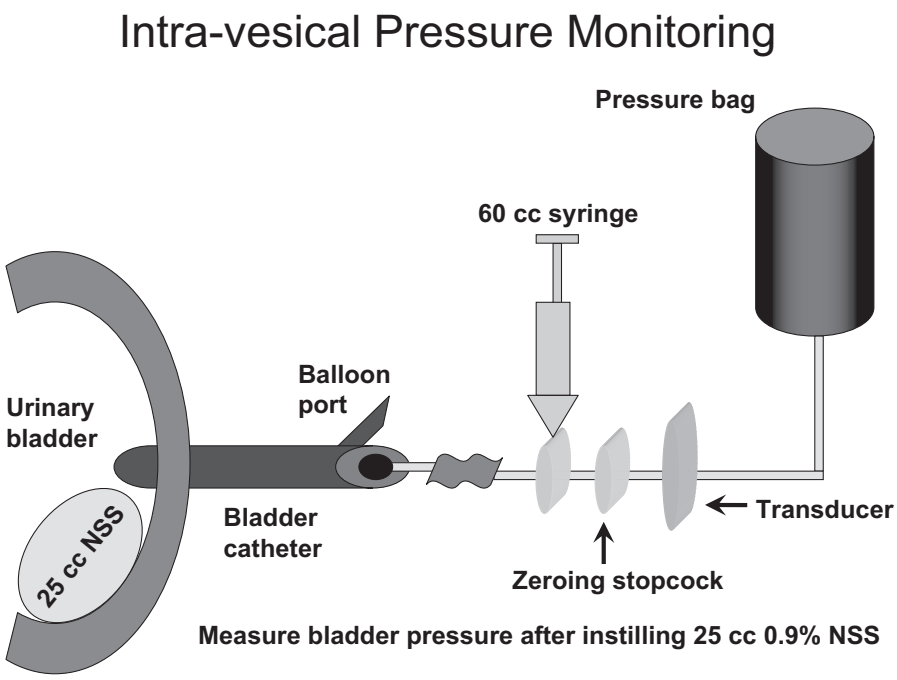

Figure 1. The intravesical method of monitoring intra-abdominal pressure. *NSS*, normal saline solution.

Table 1. Abdominal compartment syndrome–associated signs and organ failures

Hypovolemic shock
Systolic hypotension, narrow pulse pressure, lactic acidosis, tachycardia
Increased core to peripheral temperature gradient, weak pulses
Abnormal mentation
Acute kidney injury/acute renal failure
Oliguria, increased serum creatinine
Acute respiratory failure (new or worsened if pre-existing)
Hypoxia and hypercarbia
Increased peak airway pressures (volume cycled ventilation)
Decreased resultant tidal volumes (pressure-cycled ventilation)
Decreased release volumes (airway pressure release ventilation)
Acute hepatic failure
Increased liver function tests
Jaundice, coagulopathy

objective data in conjunction with organ dysfunction and failure that typify the ACS. Regardless of etiology, increased IAP compromises venous return, cardiac output, and systemic oxygen delivery. Visceral edema further limits diaphragmatic movement. Decreased diaphragm movement limits alveolar recruitment, and inappropriately elevates endobronchial, pleural, and intra-peritoneal pressures. The increased intra-thoracic pressure further reduces venous return, further limiting cardiac performance.

Due to the absence of valves in the right-sided venous return system, the increased intra-thoracic pressure is reflected down the inferior vena cava, leading to increased hepatic and renal vein pressure and diminished transhepatic and transrenal flux of oxygenated blood. The increased renal vein pressure is likewise increased by extrinsic retroperitoneal compression from increased intraperitoneal pressure. Furthermore, the decreased venous return leads to arterial hypotension (decreased mean arterial pressure), leading to decreased inflow to the end organs. The net effect of compromised inflow as well as increased afterload is further compounded by the untoward effects of organ edema, which contributes to extrinsic compression and intra-abdominal hypertension.

Organ Edema

Organ edema is a common accompaniment of all three forms of the ACS: primary, secondary, and recurrent (8). *Primary* ACS typically occurs in the setting of injury and stems from hemorrhage and visceral edema. *Secondary* ACS occurs in both surgical and medical patients and is associated with vigorous volume resuscitation with the acute formation of ascites as well as visceral edema, leading to increased intra-abdominal pressure and the ACS. The secondary compartment syndrome is increasingly common after the early goal directed therapy period for sepsis resuscitation management (9). There is some controversy whether the secondary compartment syndrome is iatrogenic or unavoidable in patients with peritonitis requiring emergency general surgery (10). *Recurrent* ACS was formerly called tertiary ACS and reflects an ACS that recurs after initial medical or surgical treatment of secondary compartment syndrome. A common theme with organ edema is tissue ischemia.

As end organs experience ischemia (venous or arterial), local release of vasodilatory substances including lactate and adenosine represents a local attempt to augment oxygenated flow. As ischemia progresses, loss of capillary integrity leads to extravasation of fluid, electrolytes, and proteins via hydrostatic pressure and loss of membrane integrity (11). The increased distance from capillary beds to metabolically active cells on the basis of tissue edema from extravascular organ water further cripples organ metabolic integrity. This viscous cycle compromises organ viability. In the case of the kidney, the clinician is unfortunately blind to this process until gross measures of organ injury are apparent.

Renal Injury

Clinically, one identifies the onset of renal injury in the setting of increased intra-abdominal pressure as oliguria with a rise in serum creatinine. This process was originally articulated by Dr. Richards and colleagues (12). According to the RIFLE criteria (a mnemonic for the progression of *r*isk of renal dysfunction, *i*njury to the kidney, *f*ailure of kidney function, *l*oss of kidney function, and *e*nd-stage kidney disease), oliguria with a rise in serum creatinine <0.3 mg constitutes acute kidney injury (13). Greater rises in creatinine coupled with oliguria constitute acute renal failure. Progression along the RIFLE continuum also correlates with mortality risk (14). Recognizing that the clinician's goal is to minimize risk and maximize outcome, recognition of IAH and an impending ACS is essential. A related syndrome in which excessive cavity pressure reduces venous return and cardiac performance is cardiac tamponade. However, unlike cardiac tamponade, in which supplemental fluid does reverse the sequelae of the tamponade (at least temporarily), once

IAH and the ACS have led to oliguria, no amount of fluid resuscitation reverses the renal injury (15). In one multicenter, prospective study of 265 patients admitted to a critical care unit, IAH was present in 32% (85 patients), of which 4% (11) were admitted with the ACS; 53% (140) had a normal IAP (16). IAH on admission was associated with multiple organ dysfunction and failure with nearly all of the patients with multiple organ dysfunction experiencing acute renal failure.

The well-documented renal response to impaired perfusion, regardless of cause, is activation of the rennin-angiotensin-aldosterone system in addition to up-regulation of antidiuretic hormone to preserve water (17). A profile consistent with acute tubular necrosis commonly follows relief of the ACS and is associated with both renal hypoperfusion and an oxygenated reperfusion injury. While the renal neurohormonal responses are adaptive in the face of hypoperfusion and reperfusion, they are bereft of the ability to reverse the extant renal cellular and subcellular injury (17). These derangements have been well characterized elsewhere in dedicated text (18). Accordingly, relief of the IAH provides the mainstay of therapy. Still, relieving the ACS after renal injury has occurred fails to achieve the ultimate goal—to detect the onset of an acute renal insult before establishing acute kidney injury or acute renal failure. To achieve that goal, one must await further investigation into the roles and utility of urinary biomarkers in detecting renal parenchymal ischemia and reversible injury (19). Currently, investigated biomarkers include serum cystatin C and neutrophil gelatinase–associated lipocalin, each of which has been associated with the onset of acute renal failure in specifically defined circumstances. To date, no single biomarker is universally applicable to both medical and surgical populations.

Therapeutic Interventions

The standard of care for intra-abdominal hypertension leading to the abdominal compartment syndrome is decompressive laparotomy with temporary abdominal wall closure to enlarge the peritoneal space and reduce the intra-abdominal pressure to normal level (Fig. 2). This management scheme parallels the standard of care for managing an extremity compartment syndrome or a thoracic compartment syndrome (20). Similar to the extremity compartment syndrome, once the cause of the intra-abdominal hypertension is controlled (hemorrhage, ascites) the abdomen may be closed primarily. If the ACS is accompanied by intestinal edema, primary closure less frequently occurs and is instead achieved by a variety of methods that expand the peritoneal envelope to prevent recurrent ACS. These techniques employ musculo-fascial separation techniques, prosthetic grafts, and skin grafts or flaps for abdominal wall reconstruction. While operative therapy is readily accepted in the surgical community, a variety of nonsurgical remedies have been explored as alternatives within both medical and surgical circles, including catheter drainage, renal replacement therapy, neuromuscular blockade, and prokinetic agents if intestinal gaseous distension is present. It is important to note than none of the alternatives has been subject to prospective, randomized controlled trial analysis to substantiate their efficacy compared with the gold-standard decompressive laparotomy. A detailed discussion of all of the therapeutic interventions is beyond the scope of this manuscript, and the reader is referred to other works more focused on intervention techniques for management of the ACS (21).

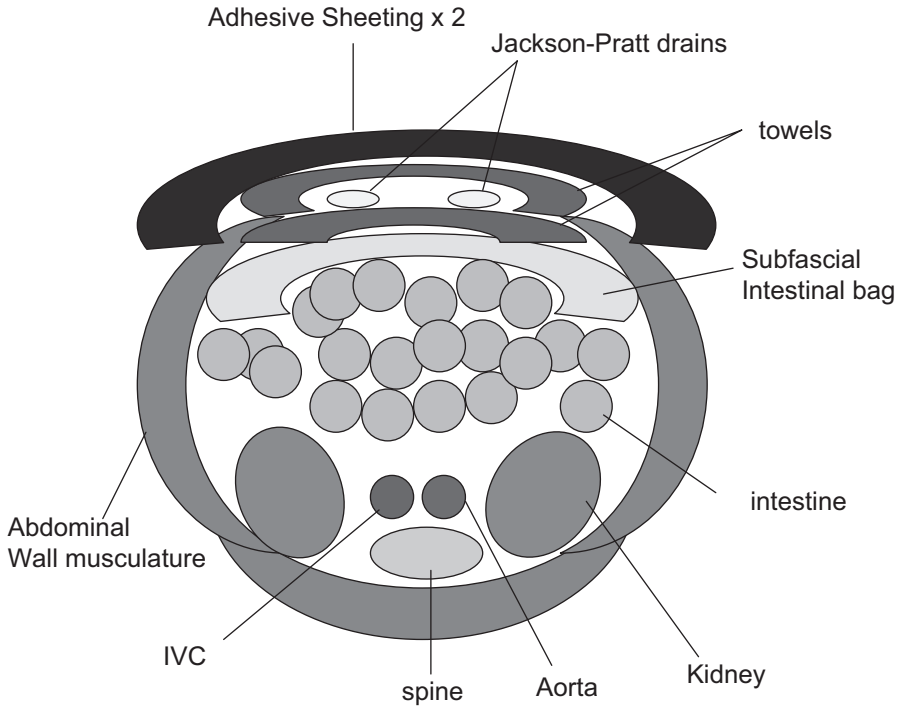

Figure 2. Diagram demonstrating one method of temporary abdominal wall closure using a sterile intestinal bag, sterile towels, and two no. 10 Jackson-Pratt drains covered by two thicknesses of impermeable adhesive plastic sheeting secured to the skin. IVC, inferior vena cava.

CONCLUSIONS

Abdominal hypertension commonly accompanies critical illness. Our current understanding of the onset of renal injury at the cellular level is lacking, and the clinician is left with only gross measures of renal injury, such as oliguria and rising serum creatinine. Thus, the current key to minimizing renal risk from intra-abdominal hypertension is to relieve the hypertension before the onset of the abdominal compartment syndrome. At present, no known strategies repair the renal injury once the compartment syndrome is present. The gold standard for the abdominal compartment syndrome is decompressive laparotomy and temporary abdominal wall closure until the source of the intra-abdominal hypertension is repaired and the abdomen may be closed either primarily or in some fashion that expands the peritoneal envelope to avoid compartment syndrome recurrence.

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